Clinical and Morphologic Outcomes of Endovascular Repair for Subacute and Chronic Type B Aortic Dissection

Tina Hellgren,[1] Marek Kuzniar,[1] Anders Wanhainen,[1] Johnny Steuer,[1,2] and Kevin Mani,[1] Uppsala and Stockholm, Sweden

Background: The objective of the study was to assess long-term remodeling, survival, and reintervention outcomes after thoracic endovascular aortic repair (TEVAR) for subacute and chronic type B aortic dissection (TBAD).

Methods: All patients who underwent TEVAR for subacute or chronic TBAD at a tertiary referral center between 1999 and 2015 were included in this cohort study. The primary outcome was aortic remodeling, and secondary outcomes included survival, rate of major complications, and reinterventions.

Results: Fifty patients were included, with mean age of 62.4 years, 10 (20%) DeBakey type IIIA and 40 (80%) DeBakey type IIIB dissection; 45 standard TEVAR, 2 branched TEVAR, 3 TEVAR combined with fenestrated or branched EVAR. Indication for TEVAR was intact ($n = 40$) or ruptured ($n = 1$) postdissection aneurysm, hypoperfusion ($n = 4$), treatment-refractory pain ($n = 2$), or a combination ($n = 3$). Mean clinical follow-up was 76 months, and median radiological follow-up was 46 months. Thirty-day survival was 96%, stroke 4%, renal failure 0%, paraplegia 0%. Three- and five-year survival was 92% (95% confidence interval (CI) [79; 97]) and 77% (95% CI [61; 87]), respectively. Of 19 late deaths, 6 were confirmed aorta related. Five-year freedom from reintervention was 69% (95% CI [53–80]). Distal stent graft extension due to aortic dilatation composed most reinterventions. Mean maximal aortic diameter was 58.7 mm preoperatively and 51.9 mm on last follow-up ($P = 0.003$). On thoracic level, true lumen expanded (+10.0 mm, 95% CI [6.4; 13.6]) ($P < 0.001$) and false lumen decreased (−11.9 mm, 95% CI [−15.2; −8.5]) ($P < 0.001$) from baseline to the last computed tomography. In the abdominal aorta, true lumen diameter change was +3.1 mm (95% CI [1.4; 4.8]) ($P = 0.001$); false lumen diameter change was +1.0 mm (95% CI [−1.8; 3.8]) ($P = 0.464$).

Presentation information: Preliminary results of this study were presented orally at the 31st ESVS annual meeting in Lyon, France, 19–22 September 2017.

Type of Research: This is a single-center observational cohort study based on retrospective analysis of prospectively collected registry data.

Key Findings: Thoracic endovascular aortic repair (TEVAR) for subacute or chronic type B aortic dissection (TBAD) in 50 patients resulted in significant expansion of true and shrinkage of false lumen in the thoracic but not abdominal aorta. Postoperative mortality was 4%, stroke 4%, renal failure 0%, paraplegia 0%. Five-year survival was 77%. Eighteen (36%) patients needed reinterventions.

Take-home Message: TEVAR for subacute and chronic TBAD has favorable remodeling outcomes in the thoracic but not abdominal aorta, and reinterventions are often necessary.

Table of Contents Summary: TEVAR resulted in favorable remodeling of the thoracic but not abdominal aorta, and need for reintervention was common, in this retrospective study of 50 patients treated for subacute and chronic type B aortic dissections. Continued surveillance of these patients is important.

Competing interests: The authors have no competing interests to declare.

Funding was provided by the Swedish Heart and Lung Foundation (grants 20170647 and 20170647) and Uppsala University Hospital research funds.

Authors' contributions: T.H., M.K., A.W., J.S., and K.M. contributed to the study design. T.H. and M.K. contributed to data collection. T.H., M.K., A.W., J.S., and K.M. contributed to analysis and interpretation. T.H. contributed to writing. M.K., A.W., J.S., and K.M. contributed to critical revision.

[1]Department of Surgical Sciences, Section of Vascular Surgery, Uppsala University, Uppsala, Sweden.

[2]Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet, Stockholm, Sweden.

Correspondence to: Tina Hellgren, Department of Surgical Sciences, Section of Vascular Surgery, Uppsala University, 75185 Uppsala, Sweden; E-mail: tina.hellgren@surgsci.uu.se

Ann Vasc Surg 2021; 72: 390–399
https://doi.org/10.1016/j.avsg.2020.08.107

Manuscript received: May 2, 2020; manuscript accepted: August 12, 2020; published online: 2 September 2020

Conclusions: TEVAR for subacute and chronic TBAD results in favorable remodeling of the thoracic but not the abdominal aorta. Five-year survival is almost 80%, but late aortic deaths still occur. Aortic dilatation distal to the treated segment requiring reintervention is common, emphasizing the importance of follow-up.

INTRODUCTION

Based on favorable survival outcomes compared with open repair, thoracic endovascular aortic repair (TEVAR) has become the treatment of choice in acute complicated type B aortic dissection (TBAD).[1] Medical treatment alone remains the gold standard in uncomplicated acute TBAD, although preemptive TEVAR is advocated by some to reduce the risk for late aortic complications.[2] A study on the natural history of medically treated acute TBAD reported the intervention-free 6-year survival to be less than 50%.[3] The most common indication for intervention is aneurysmal degeneration of the false lumen. TEVAR is increasingly used in treatment of chronic TBAD,[4,5] but the scientific basis for this is scarce.[1] The aim of TEVAR is to cover the dissection entries, achieve false lumen thrombosis and thereby aortic remodeling with false lumen regression and decreased risk of rupture. Aortic remodeling has been associated with improved survival, whereas it has been suggested that lack of aortic remodeling may be associated with worse outcomes.[6] There are reports of a lesser probability to achieve aortic remodeling when treating a chronic dissection.[7]

The aim of this study was to assess the clinical and morphologic long-term outcomes of TEVAR for subacute and chronic TBAD.

METHODS

All patients (n = 328) who underwent TEVAR at Uppsala University Hospital during the period December 1999 through December 2015 were identified using prospective local hospital registries and the Swedish National Vascular Registry (Swedvasc). All patients who had an underlying subacute or chronic aortic dissection and Stanford type B or residual type B after type A repair were included. Patients undergoing thoracoabdominal hybrid procedures (combining endovascular false lumen exclusion and open surgical visceral artery debranching) were excluded.

Selection of Patients and Procedure Details

An aortic dissection was considered to be in the subacute phase 15 to 90 days after the onset of dissection and the chronic phase when >90 days had passed. The indications for surgical treatment of subacute and chronic TBAD were aneurysmal degeneration with aneurysm diameter ≥55 mm,[1,8] aortic rupture, treatment-refractory pain, and/or hypoperfusion. The diameter threshold was lowered in cases of rapid expansion (>1 cm/year) and elevated to 60 mm for complex aortic aneurysms. Before treatment, all cases were discussed at a multidisciplinary team conference including vascular surgeons, cardiothoracic surgeons, and interventional radiologists. TEVAR has been performed since 1999 at the study center, and during the first 5 years of the millennium, it gradually replaced open surgical repair as the first-choice strategy for treatment of subacute and chronic TBAD.

Spinal drainage was inserted in elective cases where >20 cm aortic coverage was planned or in case of previous abdominal aortic repair. Supra-aortic vessel revascularization was performed routinely before TEVAR when the proximal landing zone was planned to be proximal to the left common carotid artery. Left subclavian artery revascularization was always performed in cases of expected >20 cm aortic coverage, in presence of a left internal mammary artery graft, a dominant left vertebral artery or a left-sided brachiocephalic fistula for hemodialysis, and otherwise selectively based on patient-specific anatomy and characteristics.

Data Collection and Analysis

Patient characteristics and outcome data were collected from medical records. Preoperative comorbidities were registered, including medically treated hypertension, history of myocardial infarction, chronic obstructive pulmonary disease, renal failure (creatinine concentration >150 μmol/L or renal replacement therapy), medically treated diabetes mellitus, cerebrovascular disease (ischemic or hemorrhagic stroke), and preceding aortic surgery. Operative details and adjunct procedures were registered. The primary outcome was aortic remodeling, and secondary outcomes were survival, rate of major complications (stroke, paraplegia, renal failure requiring renal replacement therapy), and reinterventions. Morphological data were collected from preoperative and postoperative computer tomography (CT) imaging studies. All patients treated electively had a preoperative CT within 6 months before TEVAR. In accordance with the local follow-up protocol, the first follow-up CT is at 1 month and the second, 1 year after the procedure, then annually thereafter. When available, we assessed one preoperative CT, one from 0–90 days (first postoperative), one from 6–18 months (second postoperative), one from 19–30 months (third postoperative), one from 31–42 months (fourth postoperative), and one from 43–54 months (fifth postoperative) after TEVAR, for every patient.

The short axis diameter of the true, false, and whole aortic lumen as well as false lumen thrombosis was assessed at 5 fixed levels along the aorta (2 cm distal to the left subclavian artery, bifurcation of the pulmonary trunk, celiac trunk, right renal artery, and infrarenal aorta). The measurements were performed on CT images with intravenous contrast in the arterial phase. Absence of contrast enhancement in the false lumen was interpreted as false lumen thrombosis. The final analyses on true and false lumen remodeling were performed on data regarding the descending thoracic aorta at the level of the bifurcation of the pulmonary trunk and the infrarenal aorta. Dissections were separated in accordance with the DeBakey classification; extending from distal to the left subclavian artery, type IIIA only involves the descending thoracic aorta, whereas type IIIB continues beyond the diaphragm. The maximal aortic diameter was assessed by measuring the diameter, perpendicular to the aortic lumen, where it was the largest, anywhere along the aorta. The length of aortic coverage was assessed by measuring the stent-grafted aorta along the center of its lumen, from the proximal to the distal end of the stent graft, on the first postoperative CT, using multiplanar reconstruction. Stent graft landing zones were categorized in accordance with the classification system proposed by Fillinger et al.[9]

To determine survival, the unique personal identification number of each patient was used for cross-matching with the Swedish national population registry to ensure completeness. Survival follow-up was performed from 7 to 10 February 2017. The cause of death registry was consulted for deceased patients. Reintervention was defined as any unplanned open or endovascular aortic procedure after the index TEVAR procedure.

The study was approved by the regional Ethical Review Board in Uppsala. Informed consent was obtained from all patients who were alive when the study was performed.

Statistics

Continuous variables were assessed for normality with histograms and presented with mean (normally distributed data) or median (nonnormally distributed data). Comparative analysis was performed with Student's *t*-test for continuous and the chi-squared test for categorical variables. The paired samples *t*-test was used to analyze the change in true and false lumen diameter over time. The diameter data from each postoperative CT were compared with the preoperative CT. A *P*-value of <0.05 was considered statistically significant. Thirty-day survival was calculated as ratio, whereas long-term survival and freedom from reintervention were estimated with Kaplan-Meier. Statistical analysis of the data was performed with SPSS, version 21 (IBM Corp, Armonk, NY).

RESULTS

Preoperative Parameters

In total, 50 patients were included; 39 (78%) with a Stanford type B and 11 (22%) with a residual type B dissection after type A repair; 10 (20%) patients were with a DeBakey type IIIA (DBIIIA) and 40 (80%) with a DeBakey type IIIB (DBIIIB) aortic dissection. Patient characteristics are presented in Table I. A total of 11 (22%) patients were treated in the subacute and 39 (78%) in the chronic phase. Nine out of 11 (82%) subacute dissections were DBIIIB dissections. Median time from onset to TEVAR was 27.6 months (interquartile range (IQR) 65.7). Forty (80%) were treated electively and 10 (20%) urgently (i.e., within 24 hours from presentation). Seven urgent cases were complicated dissections in the subacute phase, and three were chronic dissections with acute complications. Seventeen patients (34%) had undergone previous aortic repair; of these, 12 had open surgical thoracic aortic repair (10 for acute type A aortic dissection), 3 had abdominal aortic aneurysm repair, 1 had abdominal aortic aneurysm repair and thoracic aortic repair for type A aortic dissection, and 1 had suprarenal aortic bare stents for chronic type B dissection.

In forty cases (80%), the indication for TEVAR was to prevent rupture of a postdissection aneurysm (mean diameter 62.2 mm, standard deviation (SD) 11.3). There was one case (2%) of aortic rupture, 2 (4%) of intestinal malperfusion, 1 (2%) of renal malperfusion, 1 (2%) of suspected renal malperfusion but mainly treatment-refractory hypertension, and 2 (4%) of treatment-refractory pain. All cases of

Table I. Baseline characteristics of the 50 patients

Age, years, mean (SD)	62.4 (11.2)
Maximal aortic diameter, mm, mean (SD)	58.7 (13.7)
Male sex, *n* (%)	36 (72)
Hypertension, *n* (%)	46 (92)
COPD, *n* (%)	10 (20)
Cerebrovascular disease, *n* (%)	6 (12)
Renal insufficiency, *n* (%)	6 (12)
Diabetes mellitus, *n* (%)	5 (10)
Myocardial infarction, *n* (%)	3 (6)
Previous aortic surgery, *n* (%)	17 (34)

Values are reported as number (%) unless otherwise indicated. COPD, chronic obstructive pulmonary disease.

hypoperfusion and treatment-refractory pain occurred in the subacute phase. In 3 cases (6%), a combination of the mentioned indications led to TEVAR.

Procedure Details

The median number of stent grafts used was 1.0 (IQR 1.0). The most commonly used stent-graft types were Gore TAG (WL Gore & Associates Inc., Flagstaff, AZ) ($n = 27$) and Cook TX2 (Cook Medical Inc., Bloomington, IN) ($n = 15$). The other types used were Bolton Relay (Bolton Medical Inc., Sunrise, FL) ($n = 3$), Medtronic Valiant (Medtronic, Minneapolis, MN) ($n = 2$), or combinations of different stent graft types ($n = 3$). The mean length of the aorta covered was 22.1 cm (SD 7.0).

The most frequent adjunct procedures were revascularization of supra-aortic arteries and cerebrospinal fluid drainage, both efforts to avoid spinal ischemia (Table II). Five patients received fenestrated or branched stent grafts. Two patients with aortic arch aneurysms received branched arch stent grafts, and 2 patients with thoracoabdominal aneurysms received both thoracic stent grafts and stent grafts with branches for the visceral arteries. One patient with a distal descending aortic aneurysm received a stent graft with a fenestration for the celiac trunk and a scallop for the superior mesenteric artery.

Aortic Remodeling

Median radiological follow-up was 46 months (IQR 67). Ninety percentage of DBIIIA and 30.8% of DBIIIB dissections had complete false lumen thrombosis on the last follow-up CT ($P = 0.001$). The mean maximal aortic diameter was 58.7 mm preoperatively and 51.9 mm on the last CT ($P = 0.003$) (Fig. 1); the reduction remained significant when

Table II. Procedure details

Variables	*N* (Total number of patients = 50)
Proximal landing zone[a]	
0	2
1	6
2	22
3	15
4	2
5	1
Distal landing zone[a]	
4	10
5	34
6	2
7	1
8	0
9	1
10	1
Covered arteries with revascularization	
Left subclavian	16
Left common carotid	6
Left vertebral	2
Aberrant right subclavian artery	1
Covered arteries without revascularization	
Left subclavian	12
Celiac trunk	2
Visceral artery stenting	
Right renal artery	2
Superior mesenteric artery	1
Celiac trunk	1
Prophylactic cerebrospinal fluid drainage	23

[a]Zones of attachment as classified by Fillinger et al.,[9] Journal of Vascular Surgery, 2010.

subacute cases were excluded. Aortic true and false lumen remodeling on thoracic and infrarenal level is illustrated in Figure 2. The proportion of patients with available follow-up CT for each time point is illustrated in Figure 3. On thoracic level, the mean diameter change from baseline to the last CT was +10.0 mm (95% confidence interval (CI) [6.4; 13.6]) ($P < 0.001$) for the true and −11.9 mm (95% CI [−15.2; −8.5]) ($P < 0.001$) for the false lumen. After subacute and DBIIIA dissections were excluded, aortic remodeling remained significant on thoracic level. Only DBIIIB dissections were included in the analysis on infrarenal level because only these involve the abdominal aorta. The mean diameter change from baseline to the last CT at this level was +3.1 mm (95% CI [1.4; 4.8]) ($P = 0.001$) for the true and +1.0 mm (95% CI [−1.8; 3.8]) ($P = 0.464$) for the false lumen. An

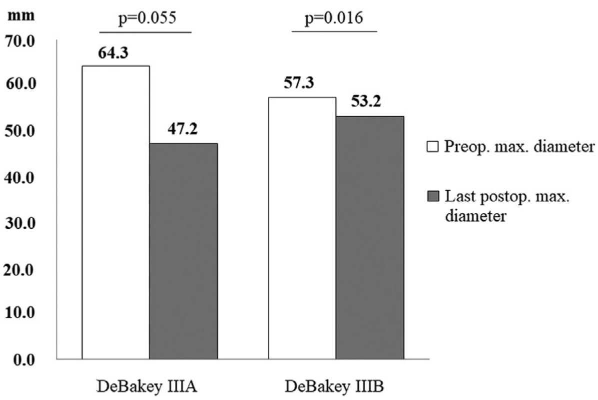

Fig. 1. Mean maximal aortic diameter change from baseline to the last CT obtained after a median 46 months (IQR 67) after TEVAR.

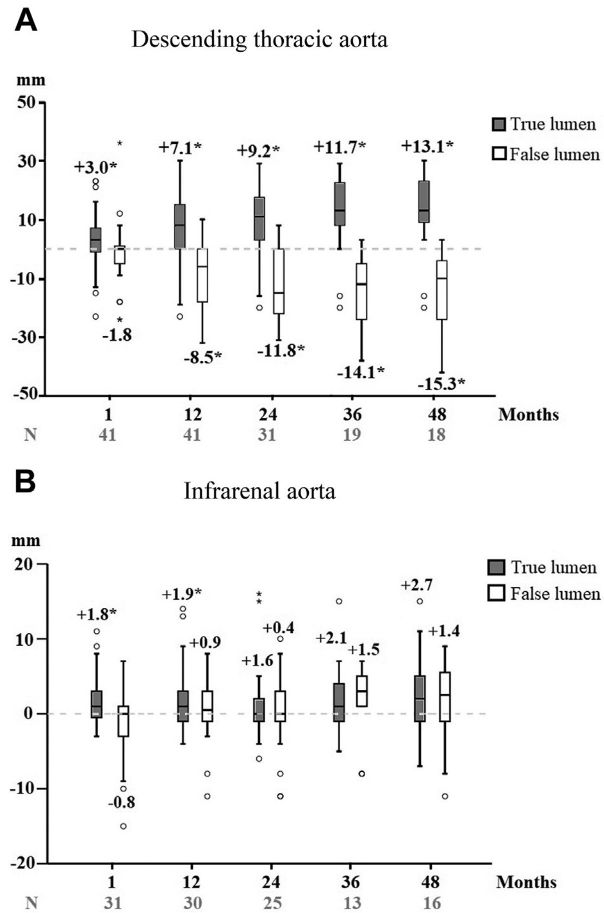

Fig. 2. Change of true and false lumen diameter after TEVAR for chronic type B aortic dissection. Mean change in millimeter is displayed above or below the respective box. * = $P \leq 0.05$. **(A)**. Descending thoracic aorta. **(B)**. Infrarenal aorta.

example of thoracic aortic remodeling with continued perfusion of the false lumen distal to the stent graft is illustrated in Figure 4.

Major Complications

There were 2 deaths within 30 days; 30-day survival was 96.0% (95% CI [86; 100]). Two patients (4%) suffered a perioperative stroke. One had an aberrant right subclavian artery and underwent bilateral carotid-subclavian bypass procedures before TEVAR, then suffered a stroke 5 days later. The other presented urgently with pain and rapid false lumen expansion, underwent an acute TEVAR with coverage of the left subclavian artery, and had an intraoperative minor stroke. No patient developed renal failure or permanent paraplegia. One perioperative death occurred after 4 days because of heart failure in a 74-year-old patient who had undergone a standard TEVAR and who had previous history of atrial fibrillation. The other was a sudden unexpected death after 28 days, cause unknown, in a 67-year-old patient who had received a thoracic stent graft and a stent graft with branches for the visceral arteries and who had a history of previous open descending aortic repair due to rupture.

Long-term Survival

Mean clinical follow-up was 76 months (SD 46). The Kaplan-Meier estimated 1-, 3-, and 5-year survival was 94.0% (95% CI [83; 98]), 91.7% (95% CI [79; 97]), and 76.5% (95% CI [61; 87]), respectively (Fig. 5A). There were no late deaths among the patients who received fenestrated or branched stent grafts. In patients treated with standard TEVAR, 19 late deaths occurred. The cause of death was documented in 13 cases, and 6 deaths were aorta related. In detail, 1 patient who had TEVAR with distal landing zone 5 and where CT follow-up revealed false lumen perfusion died after 2 months because of a new aortic dissection with abdominal aortic rupture. A second death occurred 3.7 years after TEVAR because of aortic rupture 2 cm distal to the stent graft at site of a stent-graft–induced new entry (SINE). This was known and the patient was scheduled for reintervention, but succumbed before this. A third patient, who received a 12-cm stent graft and had continued false lumen perfusion on follow-up, died 4.3 years after TEVAR in a thoracic aortic rupture. A fourth patient died 4.9 years after TEVAR because of multiorgan failure after a thoracoabdominal hybrid procedure with open surgical debranching of the visceral arteries and stent graft exclusion of a paravisceral aortic aneurysm. A fifth patient, who underwent TEVAR with distal landing

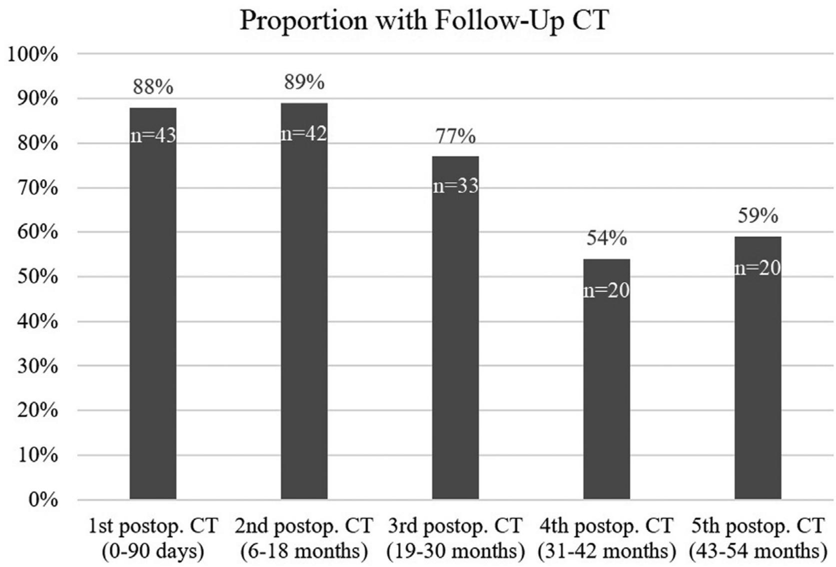

Fig. 3. Proportion of patients with available follow-up CT for each time point.

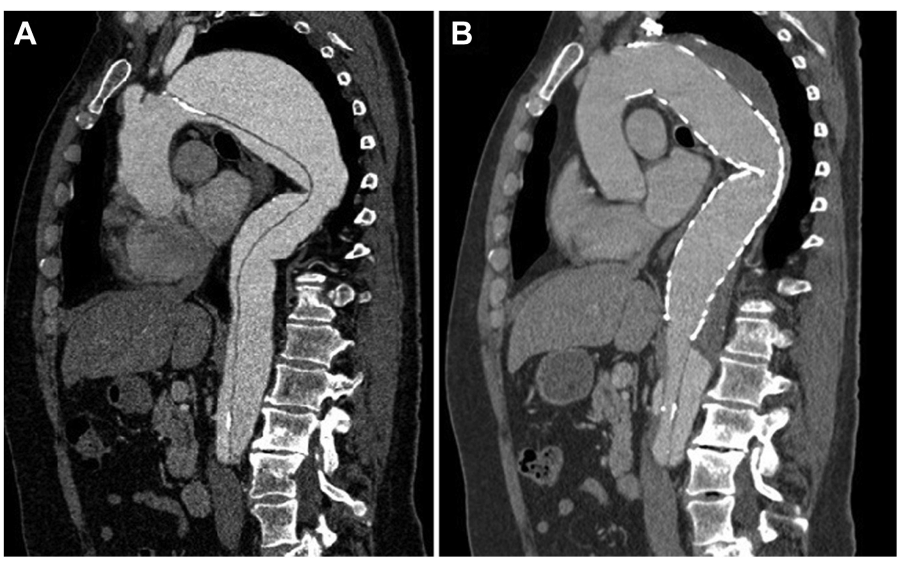

Fig. 4. A 65-year-old male treated for a chronic DeBakey type IIIB dissection with aneurysmatic dilatation, 16 years after initial dissection. **(A)**. Preoperative image. The true lumen is compressed, whereas the false lumen is perfused and expanded. The primary entry tear is at the level of the left subclavian artery. **(B)**. One year after TEVAR, with carotid-subclavian bypass and insertion of a plug in the proximal left subclavian artery. False lumen thrombosis and shrinkage on thoracic level. The false lumen is still perfused distal to the stent graft.

zone 5, where there seemed to be total false lumen thrombosis on the second postoperative CT, died in a thoracic aortic rupture after 6.7 years. In the last case, a patient dying 6 years after TEVAR, the cause was reported to be aorta related but not further specified. Among the 6 late aorta-related deaths, there were 3 cases of false lumen shrinkage on thoracic level. Because of lag in the registration process, there was no documented cause of death available for the 6 deaths that occurred after 2014.

Reinterventions

Five-year reintervention-free survival was 68.6% (95% CI [53; 80]) (DBIIIA 60.0%, DBIIIB 71.2%)

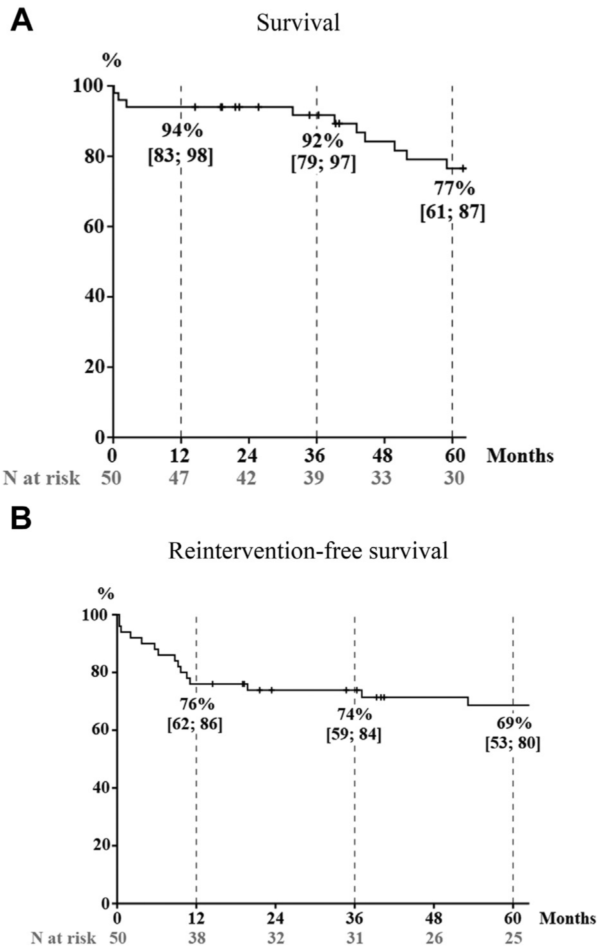

Fig. 5. Long-term **(A)** survival and **(B)** reintervention-free survival, after TEVAR for chronic type B aortic dissection. Ninety-five percentage CI presented within brackets. Standard error <10% for all survival estimates.

(Fig. 5B). For the 20 patients with ≤20 cm stent graft coverage of the aorta, the 5-year reintervention-free survival was 74.7% (95% CI [49; 89]). Median time to first reintervention was 9.4 months (IQR 37.8). In total, 25 reinterventions were performed on 18 patients. The reinterventions are summarized in Table III. A total of 16 out of 17 distal stent graft extensions were due to false lumen expansion distally to the first stent graft and 1 due to partial collapse of the first stent graft with a SINE. One proximal extension was performed to exclude a saccular aneurysm just proximal to the stent graft and one to exclude a new dissection with expanding false lumen compressing the primary stent graft.

Subgroup Analyses

Residual dissections after type A repair were treated with a mean of 2.0 (SD 0.8) stent grafts, and the median length of aortic coverage was 24.0 cm (IQR 7.0). The mean change of false lumen diameter in the thoracic aorta between the preoperative and the last follow-up CT was −7.4 mm (95% CI [−13.2; −1.6]) ($P = 0.018$). Thirty-day survival was 100%. There were no cases of stroke, permanent paraplegia, or renal failure. Two of the late aorta-related deaths previously described, occurred in patients with residual dissections after type A repair.

The subacute dissections were treated with a mean number of 1.1 (SD 0.3) stent grafts, and the median length of aortic coverage was 16.0 cm (IQR 4.0). There was no perioperative death, stroke, paraplegia, or renal failure, and 5-year survival was 100%. The estimated 5-year reintervention-free survival was 72.7% (95% CI [37; 90]), all 3 reinterventions within the first year after TEVAR. Ten (91%) subacute dissections displayed shrinkage of the false lumen on thoracic level; the mean false lumen diameter change was −16.6 mm (95% CI [−22.1; −11.1]) ($P < 0.001$). Six dissections treated in the subacute phase (55%) displayed complete false lumen thrombosis on the last CT.

DISCUSSION

This long-term assessment of aortic remodeling and survival after endovascular repair of subacute and chronic TBAD suggests that TEVAR results in reduction of false lumen aneurysm diameter in the thoracic aorta over time, whereas distal remodeling does not occur. Patients with DBIIIA dissection have a greater chance of complete false lumen thrombosis after TEVAR, whereas the false lumen remains partly perfused in the more extensive DBIIIB dissections.

Aortic Remodeling

There are a limited number of prior reports analyzing change of true and false lumen dimensions after TEVAR for chronic dissection. A small study including 10 patients with aneurysmal expansion secondary to chronic TBAD reported a significant reduction in false lumen diameter at the level of the stented aorta.[10] In 2014, the VIRTUE registry investigators reported an increase in true but no reduction in the false lumen area for chronic dissections, whereas subacute dissections had both an increase in true and a reduction in the false lumen area.[5] The mean maximal aortic diameter decreased during follow-up in the present study, in line with a recent report on promising results regarding sac shrinkage.[11]

Table III. Reinterventions

Type of reintervention	Standard TEVAR ($n = 45$)	Fenestrated or branched TEVAR ($n = 5$)
Stent graft extension		
Distal	17	0
Proximal	1	1
Stent graft in the celiac trunk to cover reentry	1	0
Visceral bypass with distal stent-graft extension	1	0
Plug bronchial arteries[a]	1	0
Embolization lumbar artery[a]	2	0
Balloon-dilatation of the aorta and deployment of a suprarenal stent[b]	1	0

[a]Performed due to persistent flow in the false lumen parallel to the stent graft.
[b]Performed due to the compressed true lumen distal to the stent graft and visceral hypoperfusion.

It is well established that TEVAR promotes aortic remodeling in acute TBAD,[12] whereas the tendency for remodeling is not as obvious in chronic dissection.[7,13] The most acknowledged explanation is a thicker, less mobile dissection membrane with multiple reentries.[14,15] Our findings indicate that, on the contrary, when many years have passed since onset of dissection, TEVAR can still induce remodeling of the thoracic aorta with expansion of the true and shrinkage of the false lumen, although this occurs slowly over time. Use of false lumen embolization techniques may increase the rate of total false lumen thrombosis and aortic remodeling.[16] Further studies are necessary to evaluate the risk of clinically significant aneurysm formation in the abdominal aorta. Fenestrated and branched stent grafts may offer a good treatment solution in cases of complex thoracoabdominal chronic dissection aneurysm.[17,18]

Survival and Reinterventions

The perioperative survival of 96% is comparable with that presented by others ranging between 92.5% and 100%.[6,13,19–21] Five-year survival after TEVAR for chronic dissection varies widely in the literature, at least from 65% to 89%.[19,21,22] In recent reports on outcomes of open surgical repair, perioperative survival is 91–96%, and 5-year survival ranges from 72% to 83%.[23–26] Early- and long-term survival in the present study is thus comparable with or better than previously reported endovascular and open repair outcomes. A substantial number of late deaths in the present study were aorta related. One of these deaths was clearly avoidable and occurred in a patient awaiting reintervention, and another was due to postoperative complications after thoracoabdominal repair. However, one of the ruptures occurred long time after TEVAR and despite previous imaging suggesting total false lumen thrombosis, underlining the progressive nature of and the long-term risks associated with aortic dissection. Experimental studies have shown that pressurization of the false lumen may be present even after achieving thrombosis,[27] resulting in a potential risk of rupture despite false lumen thrombosis. Selected patients may benefit from distal treatment to achieve total false lumen exclusion.

Reinterventions were necessary in one-third of cases, emphasizing the importance of radiological follow-up. Other studies with similar sample size report reintervention-free survival ranging from 61 to 88% at 3-year follow-up.[5,6,25,26] Most reinterventions were stent-graft extensions to deal with aortic expansion at the distal end of the primary stent graft. In the early period of this study, the main strategy of TEVAR for chronic dissection was to cover the primary entry tear. The high rate of distal extensions, however, underlines the importance of extensive thoracic aortic coverage when treating chronic dissections, which was adopted over time.[14] Our current strategy is to cover the entire descending thoracic aorta, from left subclavian artery proximally to the celiac trunk distally. Most of the patients in the present study had an established chronic aortic dissection with aneurysmal dilatation. Results from the INSTEAD trial suggest that there may be a benefit of TEVAR in the early chronic phase to promote aortic remodeling and avoid aneurysm formation.[2] However, TEVAR in the early chronic phase is not without risk, and far from all patients require an intervention in the long term. Therefore, the role of preemptive TEVAR in patients with uncomplicated aortic dissection to avoid future aortic complications is still not fully established.[1]

Limitations

There are several limitations of the present study. It is a single-center report, and therefore, the study population is small. The retrospective design poses a risk of missing data and unmeasured confounders. Reports on TEVAR for chronic dissection with long-term follow-up and detailed analysis of aortic morphology are however scarce, and this report thus contributes to the overall knowledge regarding this complex pathology. The high clinical and radiological follow-up rate, in combination with complete survival follow-up, increases the validity of the results. False lumen thrombosis is difficult to assess because the evaluation differs depending on the examination method. We used contrast enhancement on arterial phase CT for evaluation of false lumen thrombosis because the venous phase was not always available. Arterial phase CT has been reported to overestimate the volume of false lumen thrombosis; these assessments are usually more accurate when performed on a venous phase CT. It is also suggested that magnetic resonance imaging with intravenous contrast can show contrast enhancement in the false lumen where CT does not.[28] However, the clinical significance of a remaining low flow in the false lumen which is not visible on the CT angiogram is unclear.

CONCLUSIONS

This long-term assessment of outcomes after TEVAR for subacute and chronic aortic dissection suggests that stent graft treatment results in favorable remodeling of the true and false lumen in the thoracic aorta. However, the abdominal aorta continues to expand slowly after TEVAR in patients with DBIIIB dissection. Five-year survival after TEVAR is comparable with previous reports on open repair, but late aortic deaths still occur. Reinterventions were necessary in one-third of cases, emphasizing the importance of continued surveillance.

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