Endovascular repair of complicated chronic distal aortic dissections: Intermediate outcomes and complications

Woong Chol Kang, MD, PhD,^{a,d} Roy K. Greenberg, MD,^{a,b} Tara M. Mastracci, MD,^a Matthew J. Eagleton, MD,^a Adrian V. Hernandez, MD, PhD,^c Akshat C. Pujara, BA,^b and Eric E. Roselli, MD^b

Introduction: Patients with chronic distal aortic dissection (CDAD) remain at high risk for late aorta-related events and reinterventions, and the ideal management strategy remains undefined. Open surgical procedures carry morbidity, but scant data for thoracic endovascular aortic repair (TEVAR) of CDAD exist. This study reports our intermediate-term results with TEVAR for complicated CDAD.

Methods: All cases of TEVAR for complicated (aortic growth, malperfusion, intractable pain) CDAD at our institution between 2000 and 2007 were retrospectively reviewed. Demographic information, indications for repair, complications, and aortic morphologic changes were collected from medical records and imaging studies. Aortic morphology (aneurysm size, false lumen thrombosis) was assessed at multiple levels with 3-dimensional image analysis techniques. Kaplan-Meier analysis was used to estimate survival, freedom from reintervention, and likelihood of false lumen thrombosis, with log-rank tests used to discriminate between Kaplan-Meier curves.

Results: In total, 144 stent-grafts were implanted in 76 consecutive patients (49 male) with complicated CDAD. Early (<30 postoperative days) mortality was 5%. There was no paraplegia, and 1 patient died of stroke. At mean follow-up of 34 months, 12 patients had died (1 aorta-related death). Seventeen patients (22%) underwent 19 secondary aortic reinterventions, mainly for enlargement of the untreated aorta remote to stent-graft repair. Three secondary procedures treated retrograde proximal dissections. Estimated survivals were 86%, 82%, and 80% at 12, 24, and 36 months, respectively, and freedoms from both death and reintervention were 72%, 64%, and 59% at similar time points. Of 67 patients (88%) with complete imaging follow-up, TEVAR resulted in significantly decreased aortic diameter through the stent-grafted segment but not untreated segments. Complete thrombosis of the entire false lumen was uncommon in patients with extensive dissections (13% vs 78% $P < .001$).

Conclusions: Management of complicated CDAD remains challenging for clinicians. TEVAR is a reasonable treatment modality for dissections limited to the thoracic aorta and for prevention of focal aortic growth in extensive dissections. Late complications and the need for secondary interventions emphasize the complexity of this patient population and the need for long-term follow-up. (J Thorac Cardiovasc Surg 2011;142:1074-83)

Most patients that survive the acute phase of proximal or distal aortic dissection have a patent false lumen and thus have persistent disease of the aortic wall. Studies suggest that 75% to 97% of patients survive acute dissections now, making chronic distal aortic dissection (CDAD) a relatively common and concerning issue.^{1,2} Such patients remain at a high risk for secondary aorta-related events and thus

require subsequent reintervention.³⁻⁹ Late complications in patients with CDAD include aneurysmal growth, persistent pain, recurrent dissection, end-organ ischemia, and rupture.

Controversy surrounds the ideal management of patients with CDAD. Optimal medical therapy with anti-impulse treatment is used for most patients,^{8,10} and selected open surgical or endovascular repair^{11,12} is used in the setting of aneurysmal degeneration, malperfusion, persistent pain, or rupture.⁸⁻²² Once intervention is indicated, however, the best treatment strategies (open vs endovascular) and the required extent of repair have not been well defined.

Irrespective of the treatment strategy, the goal is to avoid late aorta-related death. Endovascular repair requires adequate decompression of false lumen perfusion to promote changes in aortic morphology by closure of the primary intimal entry tear.¹¹⁻²² The resulting passive decompression of the false lumen allows true lumen expansion. Although the results of the treatment of complicated acute aortic

From the Departments of Vascular and Endovascular Surgery,^a Thoracic and Cardiovascular Surgery,^b and Quantitative Health Sciences,^c Cleveland Clinic Foundation, Cleveland, Ohio; and the Department of Cardiology,^d Gil Hospital, Gachon University, Incheon, Korea.

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Address for reprints: Roy K. Greenberg, MD, Desk H-32, Department of Vascular Surgery, Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland, OH 44195 (E-mail: greenbr@ccf.org).

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Abbreviations and Acronyms

CDAD = chronic distal aortic dissection

TEVAR = thoracic endovascular repair

dissection appear to be favorable for thoracic endovascular repair (TEVAR), the success of TEVAR during the chronic phase of an aortic dissection remains controversial.^{18,20,23}

In contrast to acute aortic dissection, the chronically dissected aorta is characterized by a thick intimal flap with multiple mature fenestrations,^{16,18,24} which are thought to be less amenable to repair than when associated with acute aortic dissection. Reports on TEVAR for aortic dissection include mixed groups of patients in both the acute and chronic phases, with or without complications. The aim of this study was therefore to report our intermediate-term results with TEVAR in the specific population of patients with complicated CDAD.

MATERIALS AND METHODS**Patient Population**

All patients who underwent a thoracic stent-graft (SG) implantation for a complicated CDAD from 2000 to 2007 at our institution were retrospectively identified and assessed. CDAD was defined as a dissection involving the descending thoracic aorta that did not require any interventional or surgical procedure within the 14 days after the initial diagnosis.²⁵ Complicated CDAD included the following indications: (1) maximum aortic diameter of at least 55 mm, (2) rapid aortic growth (≥ 10 mm/y), (3) clinical or radiographic evidence of rupture, (4) intractable chest pain despite maximal medical therapy, and (5) visceral, renal, or lower extremity malperfusion. Patients with penetrating ulcers or intramural hematomas were excluded from this analysis.

Preoperative Management

Before intervention, a contrast-enhanced computed tomographic scan of the entire aorta was performed in all cases and supplemented with angiography, intravascular ultrasonographic analysis, or both as necessary. Patient selection for TEVAR required a satisfactory proximal landing zone at least 15 mm in length between the left common carotid artery. The diameter of the proximal landing zone had to be less than 38 mm and either not dissected or treated previously with an arch or elephant trunk graft repair. The distal landing zone, in contrast, was typically dissected; however, the total aortic diameter had to be smaller than 5.5 cm and the true lumen diameter had to be smaller than 38 mm above the celiac artery.

Endovascular Procedure

All procedures were performed in a hybrid operating room with fixed imaging, under conditions of general, epidural, or spinal anesthesia. Drainage of the cerebrospinal fluid was used in cases of extensive aortic coverage or previous infrarenal repair. Access was transfemoral except when conduits were required as a result of small or calcified iliac arteries. TEVAR was used to cover the primary intimal tear in the proximal thoracic aorta. Four types of stent-grafts were used: TX1 and TX2 (Cook Medical Inc, Bloomington, Ind), TAG (W. L. Gore & Associates, Inc, Flagstaff, Ariz), Talent (Medtronic, Inc, Minneapolis, Minn), and homemade devices. To achieve a satisfactory seal, devices were oversized by 10% to 15% with respect to the diameter of the aorta immediately proximal to the dissection.

Balloon dilatation was performed judiciously to avoid trauma to the dissected aorta. When no proximal landing zone was available, a hybrid elephant trunk technique was used, as previously described.²⁶

Postoperative Period and Follow-up

All patients were observed in the intensive care unit after the procedure for varying lengths of time. In the absence of specific contraindications (renal failure), contrast-enhanced computed tomographic scans of the entire aorta were performed before discharge, at 6 and 12 months, and yearly thereafter.

Data Collection

With the approval of our institutional review board, medical records and radiographic information were reviewed to determine the operative indications, the repair technique, the perioperative complications, and the later outcomes. Technical success was defined as a successful proximal entry tear coverage with TEVAR without conversion to open repair or diagnosis of type I or III endoleak within the 24 hours after the procedure.

CDADs were classified as limited (with dissection involving the aorta above the celiac artery level) or extensive (with dissection involving the aorta below the level of the celiac artery). The aortic morphology (diameter, false lumen patency, and relevant branch vessel status) was examined with arterial and delayed (5 minute) contrast phases at the following levels: the proximal thoracic aorta (2 cm below the left subclavian artery ostium), the midthoracic aorta (at the level of the left inferior pulmonary vein), the distal thoracic aorta (2 cm above the diaphragm), the celiac artery, the superior mesenteric artery, both renal arteries, and the infrarenal aorta.

Adverse clinical events (mortality, respiratory failure, limb ischemia, need for dialysis, stroke, and paraplegia) occurring during the postoperative period and throughout follow-up were recorded. Causes of death were determined from clinical reports, discussions with families, death certificates, and autopsy reports when available. Deaths were categorized as aorta related (within 30 days of any aortic procedure, suggestion of rupture or malperfusion), not aorta related, or unknown.

Statistical Analysis

Demographic and periprocedural characteristics are shown as number with percentage or mean $\pm$ SD. End points were survival, freedom from reintervention, freedom from both death and reintervention, and any persistent flow in the false lumen of the dissection. Kaplan-Meier analysis was used to estimate freedoms from the respective outcomes. The log-rank test was used to evaluate differences between Kaplan-Meier curves.

We graphically analyzed changes in aortic diameters with time at each of the 8 aortic measurement points as well as overall maximum aortic diameter. Linear mixed models were used to evaluate the changes in the aortic diameter with time for each of the 9 aortic measurements. These models provided 2 parameters to describe the behavior of the aortic diameter with time: an intercept (baseline aortic diameter) and a slope (aortic growth rate in millimeters per year). The presence of persistent flow in the false lumen of the dissection at each aortic measurement point was noted. Analysis was stratified by the extent of the dissection (limited vs extensive dissection). SAS 9.0 (SAS Institute, Inc, Cary, NC) and S-Plus 7.0 (Insightful Corporation, Seattle, Wash) statistical software packages were used for all statistical analyses.

RESULTS

A total of 76 consecutive patients (49 men, 65%) underwent TEVAR for the treatment of complicated CDAD during the study period. Mean age at intervention was 61.5 ± 12.5 years (range, 34–86 years), and the mean time between the index dissection and the intervention was 25.0 ± 31.0 months (range, 2.6–33.7 months). Baseline demographic

TABLE 1. Clinical data

Baseline clinical characteristics	
Total population (no.)	76 (100%)
Male (no.)	49 (64%)
Age at diagnosis (y, mean $\pm$ SD)	59.8 $\pm$ 12.4
Age at intervention (y, mean $\pm$ SD)	61.5 $\pm$ 12.5
Time between diagnosis and intervention (mo, mean $\pm$ SD)	25.0 $\pm$ 31.0
Comorbidities (no.)	
Hypertension	75 (99%)
Smoking	32 (42%)
Hyperlipidemia	26 (34%)
Diabetes mellitus	8 (11%)
Renal insufficiency (>1.5 mg/dL)	14 (18%)
Coronary artery disease	29 (38%)
Peripheral artery disease	7 (9%)
Chronic obstructive pulmonary disease	17 (22%)
Previous stroke	9 (12%)
Marfan syndrome	2 (3%)
Previous aortic surgery (no.)	
Ascending aortic repair for type A dissection	17 (22%)
Ascending aorta without arch	11
Ascending aorta and hemiarch	3
Elephant trunk repair	3
Infrarenal aneurysm repair	12 (16%)

data are detailed in Table 1. In 17 patients (22%), CDAD persisted after successful repair of a proximal aortic dissection. Thirty patients (39%) were considered to have a limited dissection, and the remainder (61%) had extensive dissections. Indications for repair were as follows: aneurysmal degeneration in 47 cases (62%), intractable pain in 15 (20%), rapid growth in 9 (12%), and other (malperfusion, rupture) in 5 (7%; Table 2)

Procedural Details of Initial Endovascular Repair

Technical success was achieved in 73 cases (96%). Technical failure occurred in 3 patients, all of whom had residual proximal type I endoleak (noted on postoperative angiography). A total of 144 stent-grafts were implanted. The left subclavian artery was covered in 29 patients (39%). Details pertaining to the implantation procedure are highlighted in Table 2. Clinical follow-up was available for all patients, and 67 patients (88%) had imaging follow-up available. Mean clinical and imaging follow-up periods were 33.5 ± 29.4 months (range, 6.1–51.9 months) and 24.2 ± 26.3 months (range, 3–37.8 months), respectively.

Mortality

There were 4 early (<30 postoperative days) deaths (5%), 2 of which occurred during the initial treatment hospitalization. One of the in-hospital deaths was that of a 59-year-old woman who presented with malperfusion (paraplegia and acute renal failure) at admission 2 months after the surgical repair of a proximal dissection. She died

TABLE 2. Procedural data

Indication	
Aneurysmal degeneration (>55 mm)	47 (62%)
Intractable pain	15 (20%)
Rapid growth (>10 mm/y)	9 (12%)
Lower limb claudication	2 (3%)
Visceral ischemia	2 (3%)
Rupture	1 (1%)
Stent-graft type	
Gore TAG*	32 (42%)
Cook Zenith†	27 (35%)
Homemade	10 (13%)
Medtronic Talent‡	3 (4%)
Multiple types	4 (5%)
Procedure details	
General anesthesia	43 (57%)
Cerebrospinal fluid drainage	46 (63%)
Coverage of the left subclavian artery	29 (39%)
Carotid-subclavian bypass	8 (10%)

All data represent numbers and percentages of patients. *W. L. Gore & Associates, Inc, Flagstaff, Ariz. †Cook Medical Inc, Bloomington, Ind. ‡Medtronic, Inc, Minneapolis, Minn.

of multiple organ failure on postoperative day 2. The second in-hospital death was that of a 65-year-old man who died of a subarachnoid hemorrhage 8 days after the treatment of a CDAD associated with a 7.5-cm aneurysm. Other deaths within the 30 days of treatment included those of a patient who died at 12 postoperative days of an unknown cause and of a second patient who died on postoperative day 17 of a ruptured retrograde proximal dissection (identified at autopsy; Figure 1). The latter patient had previously been treated for her dissection with a segmental open surgical descending thoracic graft. She had marked dilation develop proximal to that repair and underwent TEVAR, which required subclavian coverage and carotid stenting to achieve a seal. At the autopsy, the fenestration into the true lumen appeared to be related to the lack of apposition of the stent-graft along the lesser curvature of the arch.

Twelve additional patients died during follow-up. One patient died 6 years after the procedure of rupture of a chronic ascending aortic dissection. The ascending dissection in this patient had been observed for many years because the patient refused surgery and blood products for religious reasons. Eight deaths were classified as not aorta related (3 cardiac, 2 cancers, 3 pulmonary infections). In the 3 remaining cases, the cause of death could not be determined, and these deaths are considered unknown in etiology. According to Kaplan-Meier analysis, survivals were 86%, 82%, and 80% at 12, 24, and 36 months, respectively (Figure 2, A).

Morbidity, Endoleaks, and Secondary Aortic Interventions

Eight patients (11%) had severe but nonlethal postoperative complications, including respiratory failure (prolonged

FIGURE 1. Ruptured retrograde proximal dissection (arrows) identified at autopsy.

mechanical ventilation >48 hours or tracheostomy) in 7 cases and lower limb ischemia in 1 case. No paraplegia occurred, and the only stroke was that of the patient who died on postoperative day 8.

Nine patients (12%) had an endoleak diagnosed on the initial postoperative computed tomographic scan (7 with type I endoleak, 1 with type IV endoleak, and 1 with both type I and type III endoleaks), including 2 of the patients

FIGURE 2. Product-limit survival estimate curves. A, Overall survival. B, Freedom from reintervention. C, Survival free from intervention. D, Complete thrombosis of false lumen in patients with limited (blue) and extensive (pink) dissections.

TABLE 3. Details of the aortic-related reinterventions after thoracic endovascular aortic repair for complicated DCAD

Secondary reintervention		Initial repair	
Indication and type	Time from TEVAR (d)	Initial indication	Type of SG
Retrograde type A dissection			
Ascending and arch replacement	183	Rapid growth	Gore TAG*
Ascending and arch replacement	427	Aneurysm	Gore TAG*
Ascending and arch replacement	1098	Aneurysm†	Custom-made
Aortic enlargement remote to the SG			
Infrarenal SG	30	Aneurysm	Cook Zenith‡
Infrarenal replacement	31	Aneurysm	Gore TAG*
Thoracoabdominal replacement	244	Intractable pain	Gore TAG*
Infrarenal replacement	366	Aneurysm	Gore TAG*
Infrarenal SG	427	Aneurysm	Gore TAG*
Thoracoabdominal replacement	732	Aneurysm	Custom-made
Frozen elephant trunk	793	Aneurysm	Cook Zenith‡
Infrarenal SG	1549	Rapid growth§	Medtronic Talent
Thoracic SG	1555	Malperfusion	Gore TAG*
Endoleak			
Proximal SG (type Ia)	8	Rapid growth	Custom-made
Left subclavian artery embolization (type II)	61	Aneurysm	Gore TAG*
Frozen elephant trunk (type Ia)	365	Rapid growth§	Medtronic Talent
Stent-graft and true lumen collapse			
SG ballooning	6	Aneurysm†	Custom-made
Thoracic SG	92	Aneurysm	Custom-made
SG ballooning	123	Aneurysm	Gore TAG*

TEVAR, Thoracic endovascular aortic repair; SG, stent-graft. *W. L. Gore & Associates, Inc, Flagstaff, Ariz. †Same patient. ‡Cook Medical Inc, Bloomington, Ind. §Same patient. ||Medtronic, Inc, Minneapolis, Minn.

who died during the early postoperative period (subarachnoid hemorrhage on postoperative day 8 and unknown etiology on postoperative day 12). During follow-up, 4 endoleaks (3 type I and 1 type IV) resolved spontaneously. Two patients with a type I endoleak underwent secondary procedures. Two patients (the patient with both type I and III endoleak and a patient with a type I endoleak) did not undergo contrast-enhanced computed tomographic scans during follow-up because of renal failure; however, noncontrast scans demonstrated decreases in the proximal and mid-thoracic aortic diameters. One additional patient was seen with a large late type II endoleak originating from the left subclavian artery. This was treated with coil embolization 2 months after the initial TEVAR.

In total, 19 secondary aorta-related interventions were performed in 17 patients (22%). Indications for and types of these repairs are detailed in Table 3. Of note, 10 secondary repairs were indicated for enlargement of other aortic segments, 9 of which were distal to the stent-graft because of persistent growth of the untreated segment. Three patients, in addition to the death mentioned previously, had retrograde dissection develop and required ascending aortic replacement. Persistent true lumen compression resulted in 2 patients undergoing repeat ballooning in the setting of endoprosthesis collapse. According to Kaplan-Meier analysis, freedom from reintervention rates were 83%, 75%, and 73% at 12, 24, and 36 months, respectively (Figure 2, B), and freedom from both

death and reintervention rates were 72%, 64%, and 59% at 12, 24, and 36 months, respectively (Figure 2, C).

Aortic Diameter and False Lumen Thrombosis

A decrease in the maximal aortic diameter was noted with time (mean, -3.9 ± 6.0 mm, $P < .0001$). With respect to the fixed measurement points, this diminution was noted in the region of the stent-grafted aorta (proximal and middle thoracic aorta -1.6 ± 4.8 mm, $P = .01$, and -1.9 ± 7.2 mm, $P = .04$, respectively) for both limited and extensive dissections. The abdominal aortic diameters increased significantly in patients with extensive aortic dissections (Table 4). Diameter increases (≥ 5 mm) in the region of the stented aorta occurred in 10 cases (15%). This was attributable to type I endoleaks in 2 cases and to persistent retrograde flow in the false lumen through distal fenestrations in the 8 other cases. Five of these patients underwent an additional aortic reintervention (2 frozen elephant trunk procedures, 1 thoracoabdominal replacement, 1 infrarenal replacement, and 1 stent-graft ballooning, as described previously). Overall, complete thrombosis of the entire false lumen occurred in 26 of 67 patients (39%) but was more frequently noted in patients with limited dissections than in patients with extensive dissections (78% vs 13%, $P < .001$; Figure 2, D). False lumen thrombosis was more frequently observed in the stent-grafted aorta in the proximal (91%) and the midthoracic aorta (84%) than at other levels (Table 5).

TABLE 4. Arterial diameter changes

Location	All patients (n = 67)		Extensive dissection (n = 40)		Limited dissection (n = 27)	
	Baseline (mm)	Growth (mm/y)	Baseline (mm)	Growth (mm/y)	Baseline (mm)	Growth (mm/y)
MAD	52.3 ± 11.2	-3.87 ± 6.0*	53.6 ± 10.8	-5.24 ± 8.0*	50.3 ± 11.7	-2.42 ± 3.7*
PTA	41.2 ± 10.2	-1.56 ± 4.8*	42.7 ± 9.8	-2.74 ± 5.3*	38.9 ± 10.5	0.04 ± 3.1
MTA	45.0 ± 12.6	-1.90 ± 7.2†	45.2 ± 12.4	-2.29 ± 8.4	44.6 ± 13.0	-0.84 ± 2.8
DTA	36.5 ± 8.5	0.34 ± 3.2	39.3 ± 7.8	0.49 ± 3.4	32.5 ± 7.8	-0.30 ± 3.1
CA	33.1 ± 7.7	0.10 ± 3.2	36.5 ± 7.5	-0.10 ± 3.9	NA	NA
SMA	29.7 ± 6.6	0.40 ± 2.0	32.5 ± 6.4	0.58 ± 2.4	NA	NA
LRA	26.7 ± 6.4	0.40 ± 1.3†	29.2 ± 6.3	0.58 ± 2.1†	NA	NA
RRA	27.4 ± 6.8	0.59 ± 1.9†	30.1 ± 6.7	0.91 ± 2.3†	NA	NA
AA	25.4 ± 7.0	0.30 ± 1.1†	28.1 ± 7.1	0.40 ± 1.5†	NA	NA

MAD, Maximum aortic diameter; PTA, proximal thoracic aorta; MTA, midthoracic aorta; DTA, distal thoracic aorta; CA, celiac artery; NA, not available; SMA, superior mesenteric artery; LRA, left renal artery; RRA, right renal artery; AA, abdominal aorta. * $P < .01$. † $P < .05$.

DISCUSSION

The management of aortic dissection remains among the most challenging and controversial issues in vascular and cardiothoracic surgery. The refinement of treatment paradigms for acute aortic dissection during the past 50 years has increased the number of patients surviving to the chronic phase.^{1,2} Since the first report of TEVAR for acute aortic dissection and CDAD,^{27,28} the technique has been considered as an alternative to open surgery. Although early results of TEVAR for CDAD demonstrate its safety and have shown promise, they have been less favorable with regard to changing the morphology of the disease than have reports on the treatment of acute aortic dissection with TEVAR.¹³⁻²⁰ In this study, we demonstrated that the endovascular treatment of patients with complicated CDAD is a good option for patients when the dissection is limited to the thoracic aorta and in extensive dissections is frequently (85% of the time) effective at promoting thrombosis of the false lumen and positive remodeling in the treated segments. Yet, much as with open surgical repair of CDAD, patients often require subsequent procedures, and serious complications can occur.

The main treatment goal for CDAD is to minimize the risk of aorta-related death. This goal may be promoted by the false lumen exclusion. This occurred in our series in 85% of the patients within the proximal segments, and an

overall diameter reduction was noted in most patients. The safety of this technique was confirmed by demonstrating technical success in 96%, with a relatively low perioperative mortality of 5%. Of the 4 early deaths, 1 was that of a patient presenting in extremis, whereas the others were likely related to the TEVAR (including the death from unknown cause on postoperative day 12). These cases, and the 3 late retrograde type A dissections, attest to the fragility of the aorta in patients with this chronic disease. Careful assessment revealed that the cause of the 4 retrograde dissections always related to inadequate stent-graft apposition along the lesser curvature of the arch. This was not device specific (as it occurred with 2 TAG grafts, one TX2 graft, and a homemade device). Although a great deal of discussion has focused on the potential for barbs to create dissections, particularly in patients already prone to dissection, this was not observed in this series. We were not able to assess the impact of bare uncovered proximal stents because they have intentionally not been used in dissection patients. Inadequate apposition along the lesser curvature is a likely culprit in other complications (see Table 3) in this series, such as the patient who had a type I endoleak and eventually had graft collapse of a TAG device. These issues underscore the importance of disease-specific or anatomically specific device designs, coupled with an understanding of the aorta to stent-graft interface. At least 3 of the major stent-graft companies have attempted to address this issue with newer versions of arch-specific thoracic devices. The TX2 Proform (Cook Medical) modification allows deployment of the proximal stent after the second stent allowing it forcing it to align with the lesser curve. The cTAG (W. L. Gore) device has a bent shape that is more conformable to the lesser curvature. The Valiant device (Medtronic) uses a proximal uncovered stent intended to encourage alignment of the sealing stent along the lesser curve as well. Despite these issues, the overall survival in this group of patients with complicated CDAD (Figure 2, A) compares favorably with early and late survivals after open surgical repair for similar conditions.^{11,12} Thus it is logical to conclude that

TABLE 5. Complete false lumen thrombosis by region

Complete false lumen thrombosis	%
Proximal thoracic aorta	91
Midthoracic aorta	84
Distal thoracic aorta	55
Celiac artery	49
Superior mesenteric artery	35
Left renal artery	17
Right renal artery	21
Infrarenal aorta	19

endovascular grafting for complicated CDAD is justified for patients with disease that is limited to the descending aorta, for those with disease remote from the aortic arch, and for other patients that are considered to be at high risk when undergoing conventional surgical repair.

This study does not directly address the treatment of uncomplicated CDAD, but the poor event-free survival at 36 months after treatment (59%) in a population of patients requiring intervention at a mean of 2 years after the initial event raises the question of whether earlier intervention would be beneficial in a subset of patients to promote aortic remodeling. Medical therapy for uncomplicated CDAD was considered equivalent to endovascular treatment in the only randomized trial assessing subacute and chronic dissections.²⁹ That study, however, excluded all patients with complications resulting from their dissections, essentially the entire patient population in this study. The 2-year event-free survival of 74% for medical therapy versus a 72% event-free survival for patients undergoing TEVAR was far from statistically significant; $P = .91$). This event-free survival among patients with uncomplicated dissections actually compares favorably with the event-free survival among patients treated for complications resulting from their chronic distal dissections. The crossover between groups in the INSTEAD trial is indicative of our inability to predict who is in most need for an intervention and to determine the optimal extent or aggressiveness of an aortic repair.^{30,31} A more detailed understanding of patients with dissection is required so that we can tailor treatment paradigms to best suit the disease and its sequelae with medical, endovascular, or open surgical therapy.

This process begins with imaging the aorta, and the precise method of image acquisition and interpretation of CDAD merits discussion. Most reports of TEVAR for aortic dissection have focused on false lumen thrombosis and relied on an analysis of only the arterial contrast phase of the computed tomographic scan.¹⁸⁻²⁰ It is important to consider that false lumen flow may persist in a delayed fashion, much as is seen with endoleaks. It is therefore likely that most reports overestimate the degree of false lumen thrombosis unless the analysis includes delayed phase contrast-enhanced imaging in the study. In this study, both the arterial and the 5-minute contrast delayed phases were analyzed. The delayed phase images altered the patency status of the false lumen in 15% to 20% of the cases. One must remember that contrast-enhanced computed tomographic studies rely on the bolus tracking technique to trigger the arterial imaging acquisition. This is usually set within the true lumen in the mid descending thoracic aorta. Thus several factors may affect the amount of contrast within the false lumen, such as the size and location of fenestrations, the cardiac output, and the dose and concentration of contrast administered. Consequently, a multi-phase study is required for proper assessment of flow

within the false lumen. Even the absence of contrast in the delayed phase study does not imply thrombosis, although it does denote a low-flow state. These issues likely explain the variability observed in the literature regarding false lumen thrombosis rates and our observation of a low likelihood of complete false lumen thrombosis (13%) in the setting of extensive dissection.¹³⁻²² Similarly, previously published studies on morphologic aortic changes reporting aortic diameters have not routinely used imaging techniques providing images orthogonal to the centerline of flow.³² Without sophisticated image analysis methods, fair comparisons between studies at different follow-up time points may be fraught with error. Traditional centerline of flow assessment techniques are difficult in patients with CDAD, however, because most semiautomated programs cannot differentiate between lumens and contrast levels. We found that a standardized method of film evaluation with manually assisted centerline of flow generation and multiplanar views ensured the accuracy of image comparisons during follow-up.

An important goal of TEVAR for CDAD has been to promote aortic remodeling by achieving false lumen thrombosis because persistent false lumen patency has been associated with poor long-term outcomes.³³⁻³⁵ Chronic dissections, typically characterized by a thickened, less mobile septum and multiple mature fenestrations between the true and false lumens, potentially makes the aorta less susceptible to remodeling than in patients with acute aortic dissection (Figures 3 and 4).¹⁸ In this study, false lumen thrombosis and aortic diameter diminution occurred concurrently but were generally limited to the treated aortic segments, not the other levels (Table 5). We are unable, however, to assess whether the untreated segments would have grown faster, slower, or not at all in the absence of proximal TEVAR. Complete false lumen thrombosis was uncommon in this series (13%) when dissections were extensive. These observations have triggered other device-specific studies, such as the PETTICOAT Trial being conducted by Cook Medical. In that trial, patients with dissection are being treated proximally with a stent-graft, akin to the methods described here. Distally, uncovered stents are placed within the true lumen with the hope of inducing thrombosis or positive remodeling of the noncovered stented aorta. Irrespective of the state of the distal false lumen, the most common site of aneurysmal degeneration during the chronic phase of an aortic dissection is in the proximal and midthoracic aorta, regardless of extent. These segments are also the most amenable to TEVAR. The failure of TEVAR to treat this region occurred in only 15% of our patients and was partially explained by the lack of proper sealing (type I or III endoleaks), coupled with persistent false lumen flow in the stented segment. Again, the ability to predict which patients will have persistent flow or inadequate sealing on the basis of preoperative imaging will help

FIGURE 3. A 54-year-old woman presented with a true lumen compression 4 months after the initial dissection. The preoperative CT scan showed an entry tear in the mid-descending aorta (arrow, A) with a thick intimal flap. The true lumen was extremely reduced in size (arrows, B) with subsequent renal ischemia, as confirmed by preoperative angiography. Of note, the right kidney was perfused by the false lumen (arrows, C). The true lumen enlarged significantly immediately after endograft deployment (arrows, D). At 1 year follow-up, the CT scan shows a thrombosis of the false lumen and a satisfactory expansion of the true lumen (E). The bypass to the kidney remains patent on 3D reconstructions (F).

to focus therapies for such patients in the future. In our series, 17 patients (22%) underwent secondary aortic procedures, more than half of which (9/17) entailed aortic repairs distal to the TEVAR site. It is likely that similar outcomes occur after limited open repairs, and thus all patients with residual vulnerable aorta must undergo serial imaging for their lifetime. The decision to limit extent of the aortic repair must be based on a balance of paraplegia risk, invasiveness of such a repair, and patient comorbidities.²⁹ Without better predictors of long-term outcomes, no definitive

recommendation can be universally applied to all patients with CDAD.

In this single-center report on TEVAR for CDAD, we were limited by the retrospective nature of the analysis, the small number of patients (relative to other aortic populations), and incomplete follow-up in some cases. The mortality was relatively low for this patient population, technical success was reasonable, and late aorta-related death was uncommon, making TEVAR a viable alternative to open surgery for complicated CDAD. Furthermore, the

FIGURE 4. Chronic dissection (A) is characterized by a thickened, less mobile septum and multiple mature fenestrations between the true and false lumens, making the aorta less susceptible to remodeling than is seen with acute aortic dissection (B).

ability to halt or reverse aortic growth in the stented segment in 85% of patients is evidence that TEVAR can protect the aortic wall from adverse morphologic changes. The lack of complete distal false lumen thrombosis and persistent growth of untreated segments implies that an optimal means of treatment still eludes us. Will the addition of uncovered stents alter the morphologic changes distal to the grafted aortic segment, or should repairs be carried to a more distal level, such as the celiac? Can modified devices that improve conformation along the lesser curve of the aortic arch lessen the risk of retrograde dissection? When should fenestrated or branched devices be used to repair the arch or entire thoracoabdominal segment? These questions, in addition to the persistent desire for understanding the truly long-term durability of stent-grafts in the setting of aortic dissection, remain, and they are the subject of ongoing studies.

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