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Endovascular Repair of Ascending Aortic Dissection

A Novel Treatment Option for Patients Judged Unfit for Direct Surgical Repair

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Objectives This paper sought to report the outcomes of patients who are considered unfit for urgent surgical repair of ascending aortic dissections (AADs) who were treated using a novel endovascular repair strategy.

Background AAD is best treated by direct surgical repair. Patients who are unable to undergo this form of treatment have poor prognoses. Previously, clinical case reports related to endovascular repair of AAD have been controversial.

Methods Between May 2009 and January 2011, 41 consecutive patients with AAD were treated in our institution. Fifteen patients were considered poor candidates for direct surgical repair and subsequently underwent the endovascular repair.

Results The nature of the referral process to our tertiary care facility made the median time from aortic dissection onset to treatment 25.5 days (range: 6 to 353 days). Dissections in 5 patients (33.3%) were considered acute, and those in 10 patients (66.7%) were considered chronic. The rate of successful stent-graft deployment was 100%, and there were no major morbidities or deaths in the perioperative period. Median follow-up was 26 months (range: 16 to 35 months). One new dissection occurred in the aortic arch at 3 months and was treated with a branched endograft. Significant enlargements of true lumens and decreases of false lumens and overall thoracic aorta were noted after the procedures.

Conclusions Endovascular repair of AAD was an appropriate treatment option in patients who were considered poor candidates for traditional direct surgical repair by the clinical criteria used in our institution. A larger series of cases with longer follow-up is needed to substantiate these results. (J Am Coll Cardiol 2013;61:1917–24)

Stanford type A aortic dissection is the most common form of thoracic aortic dissection, accounting for approximately 60% of all cases. Untreated, this condition has a high mortality, which emphasizes the need for emergency repair. Classic therapy is direct surgical replacement of the ascending aorta with a prosthetic graft and maybe combined aortic valve replacement, reconstruction of the aortic arch and its branches, or both, as clinically indicated. Morbidity and mortality for direct surgical repair both have improved substantially in recent years (1,2). Unfortunately, the International Registry of Acute Aortic Dissection showed that approximately 28% of patients with acute type A aortic dissections were considered unfit for open surgery and received only medical management (3). Survival of these medically managed patients is compromised significantly in both acute and chronic circumstances compared with those who receive direct surgical repair (4).

Recent improvements in endograft technology raised the consideration that these new shorter, larger-diameter endografts may be useful as a novel endovascular treatment for patients with acute ascending aortic dissection (AAD) who were considered poor candidates for direct surgical repair (5). Zimpfer et al. (6) conducted an ex vivo study demonstrating that stent-graft placement for AAD was feasible and could achieve complete exclusion of the false lumen. Isolated case reports describing endovascular repair of AAD have been published, but patient follow-up was extremely limited (7–9). Some of these reports identified major complications, including procedural-related cerebral ischemia and progressive aortic valvular insufficiency, and therefore raised serious questions

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Abbreviations and Acronyms

AAD = ascending aortic dissection

CTA = computed tomography angiography

LV = left ventricle/ventricular

regarding the value of endovascular repair of AAD (10). On the basis of our previous experience with endovascular repair of aortic dissections, we combined newly designed stent grafts with our endovascular techniques for managing aortic dissection to offer treatment to a select group of patients with AAD (11–15).

Methods

Pre-operative evaluation. Between May 2009 and January 2011, 41 consecutive patients with AADs verified by computed tomography angiography (CTA) were treated at our institution, Changhai Hospital, Second Military Medical University, Shanghai, China. All patients initially were treated at other hospitals using aggressive medical management and were referred to our hospital for further treatment. The indications for referral usually were refractory hypertension; persistent or recurrent chest pain, back pain, or both; or progressive aneurysmal dilatation of the aorta (increased more than 5 mm per 6 months or more than 10 mm per year). A consultation board, consisting of vascular surgeons, cardiac surgeons, cardiologists, and anesthetists, reviewed all cases. Most patients were offered direct surgical repair for their AAD. Those patients who were judged poor candidates for direct surgical repair were offered an endovascular repair following the technique described in this paper. It should be noted that most patients with acute aortic dissection in China, as in other parts of the world, usually receive emergency direct surgical repair for the dissection with ascending aortic resection, prosthetic graft reconstruction, and management of aortic valvular issues and aortic arch pathological features, as indicated. The patients described in this paper represent those who were treated medically for their acute AAD for a variety of reasons specific to the hospital where they initially sought treatment. These patients were referred because of persistent symptoms or anatomic findings that were thought to require therapeutic intervention. These are patients who survived the initial phase of acute aortic dissection with medical therapy alone. The exclusion criteria for open surgery used by our consultation board were established as follows: advanced age (≥70 years), American Society of Anesthesiologists classification of IV or more, New York Heart Association classification (1994) of cardiac function of III or C or more, or dysfunction of other important organ systems (i.e., severe chronic obstructive pulmonary disease or renal or hepatic insufficiency) (16,17).

Patients unfit for direct surgical repair were offered an endovascular treatment using our novel technique if they had suitable anatomic features. Those patients considered unfit for endovascular repair were defined as follows: length of proximal or distal aortic landing zone <20 mm, involvement of coronary artery orifice, evidence of acute myocardial infarction, severe aortic regurgitation (grade 3 or 4) documented by echocardiography, history of potentially life-threatening ventricular arrhythmia, severe tortuosity or narrowing of the descending aorta, or connective tissue disorders (Marfan's or Ehlers Danlos syndrome). Patients with intramural hematoma or penetrating aortic ulcers were excluded from this study. In all, 15 patients with AADs were treated using our endovascular technique. The other 26 patients were treated either by direct surgical repair (n = 21) or continued best medical therapy (n = 5). The endovascular procedure used in this study was approved by our internal review board, and appropriate informed consent was obtained for each patient.

CTA was used to evaluate the entire aorta and iliac arteries, and 3-dimensional reconstruction was obtained using Aquarius (TeraRecon, Foster City San Mateo, California). Status of patency or thrombosis of the false lumen was assessed using delayed phase imaging. Echocardiography was used to evaluate cardiac myocardial and valvular function.

Selection of endovascular prosthesis. The size and length of the stent grafts used in each case were determined by information derived from the CTA and intraoperative aortography with calibrated catheters. Zenith TX2 Pro-Form endografts (Cook, Bloomington, Indiana) were used in the ascending aorta.

Endovascular procedure. General anesthesia was used in all cases. Routine aortography of the ascending aorta from the sinus of Valsalva was used to localize the proximal aortic tear, the orifice of the coronary arteries, and the branches of the aortic arch. A temporary bipolar pacemaker device (Medtronic, Minneapolis, Minnesota) was placed in the right ventricle from the right femoral vein. The cardiac pacing threshold was fewer than 60 beats/min. A defibrillator was available immediately for use as required. The Cook endograft was positioned in the ascending aorta over an extra-stiff guidewire (Cook). A pigtail catheter (OptiMed, Ettlingen, Germany) was positioned in the ascending aorta close to the aortic valve. In all cases, it was necessary for the endograft to be placed in the very proximal ascending aorta just distal to the orifices of the coronary arteries. The design of the Cook TX2 system uses a long tapered nose cone as part of the delivery system. This nose cone had to be passed through the aortic valve and into the left ventricle (LV) to position the endograft at the appropriate deployment location. Initially, we planned to use rapid cardiac pacing to diminish cardiac output and blood pressure to facilitate accurate endograft deployment. However, when the nose cone of the deployment system contacted the LV wall, ventricular tachycardia was induced in every patient, resulting in significant hypotension. The endograft was deployed rapidly and, after the delivery system was withdrawn from the ventricle, ventricular tachycardia or fibrillation resolved in most cases within 30 seconds. If it did not, chemical and/or electrical cardio-

version using the defibrillator was accomplished. Arteriographic visualization of the aorta from the root to the distal aortic arch was obtained to verify patency of the coronary and brachiocephalic arteries and to show successful entry tear exclusion. Complete details of the remaining portion of the procedure, including removal of the delivery system and repair of femoral access, have been described previously (18).

Follow-up. Overall aortic and true lumen diameters were measured at the level of the sinotubular junction, the ostium of the innominate artery, the point of maximum ascending aortic diameter, and the ostium of the left subclavian artery. Three-dimensional TeraRecon reconstructions were used for these measurements and were obtained at 6 months after the procedure and annually thereafter. Additional CTA examinations were obtained if patients demonstrated new symptoms or signs of adverse events. All measurements were made by the first author and were verified independently by the coauthor.

Statistical analysis. Continuous variables were reported as mean ± SD. Skewed variables were summarized as median and range. SPSS software version 13.0 (SPSS, Inc., Chicago, Illinois) was used for all analyses. The comparisons of diameters were drawn by 2-sided paired sample *t* tests. A value of $p < 0.05$ was considered significant.

Results

The demographics of patients who underwent endovascular procedures are presented in Table 1. The mean age of patients was 65.0 ± 12.1 years (range: 45 to 78 years). The median time from onset of acute dissection to repair was 25.5 days (range: 6 to 353 days). We consider the 14-day period after onset of aortic dissection to be the acute phase. After that period, the dissection was considered chronic. Endovascular treatment was accomplished in 5 patients (33.3%) in the acute phase and in 10 patients (66.7%) in the chronic phase. Treatment delay was intrinsic to the hospital referral system except in 1 patient treated at 353 days after occurrence of the dissection because of progressive false lumen enlargement. The false lumen in the ascending aorta expanded by 12 mm, resulting in tracheal compression, recurrent laryngeal nerve dysfunction, dyspnea, and hoarseness. The endovascular repair was undertaken because of these issues.

Table 1 Patient Characteristics and Clinical Outcomes

Sex (male)	12	80% (12/15)
Age (yrs)	65.0 ± 12.1	
Risk of open surgery		
Advanced age	7	38.9% (7/18)
Severe COPD	3	16.7% (3/18)
Cardiac dysfunction	2	11.1% (2/18)
Renal insufficiency	2	11.1% (2/18)
Others*	4	22.2% (4/18)
Dissection range		
Confined to ascending aorta	1	6.7% (1/15)
Involving descending aorta	1	6.7% (1/15)
Involving abdominal aorta	13	86.7% (13/15)
ICU stay (days)	3.3 ± 1.0	
Hospitalization time (days)	9.4 ± 2.5	
In-hospital complication	2 (arrhythmia)	13.3% (2/15)
In-hospital mortality	0	0%
Follow-up time (median, range), months	22, 12–31	—
Follow-up complications	1 (new dissection)	6.7% (1/15)
Reintervention	1	6.7% (1/15)
Follow-up mortality	0	0%

Values are n, % (n/N), or mean ± SD. *Others: 1 case with hepatic inadequacy (post-hepatitic cirrhosis, Grade B of Child-Pugh Classification) and airway obstruction, 1 with prior thoracotomy, and 1 stroke.

COPD = chronic obstructive pulmonary disease; ICU = intensive care unit.

One patient required angioplasty and stenting at a referral hospital 16 days after onset of the dissection because of false lumen–induced compromised flow to the left renal artery (Fig. 1). Two patients showed the proximal entry tears in the ascending aorta to be excluded, but flow persisted in the false lumen. This false lumen flow resulted from secondary tears in the descending aorta with retrograde flow, and these secondary tears were repaired using stent-graft coverage in the descending thoracic aorta (Hercules 36-60 stent graft, Microport, Shanghai, China, and Zenith TX2 36-197 stent graft, Cook) (Fig. 2).

Mean endovascular procedure time was 128.6 ± 26.2 min. The results of intraoperative measurement are recorded in Table 2. The mean ratio of the diameter of the stent graft to that of the aorta (proximal to the tear) was 137.3 ± 11.6%. Average stent-graft coverage of the ascending aorta was 84.1 ± 9.3%.

One episode of ventricular tachycardia occurred during guidewire placement in the LV, and this resolved with guidewire repositioning. One patient experienced a supraventricular arrhythmia on the first post-operative day and required chemical cardioversion for resolution. Length of stay was 3.3 ± 1.0 days, and the total hospital length of stay was 9.4 ± 2.5 days (Table 1).

Median follow-up was 26 months, ranging from 16 to 35 months. A new dissection, secondary to an entry tear in the arch, was noted by CTA 3 months after primary procedure. This was treated successfully using a customized branched stent graft. No other complications or deaths were recorded in the follow-up period. Complete thrombosis of the false lumen of the ascending aorta was noted in all 15 patients. Complete thrombosis of the false lumen of the descending aorta was noted in 10 patients (10 of 14, 71.4%), and partial thrombosis was noted in 4 patients (4 of 14, 28.6%). No developed aortic insufficiency was observed in the follow-up period. The pre-operative LV end-systolic diameter was 37.9 ± 10.3 mm, and the follow-up LV end-systolic diameter was 41.3 ± 4.2 mm ($p = 0.168$). The LV ejection fraction was 54.6 ± 2.8% before the procedure and 53.0 ± 2.5% during the follow up ($p = 0.058$).

The diameter of the sinotubular junction was not enlarged after the procedure. The pre-operative diameter of the sinotubular junction was 26.6 ± 3.4 mm, and the

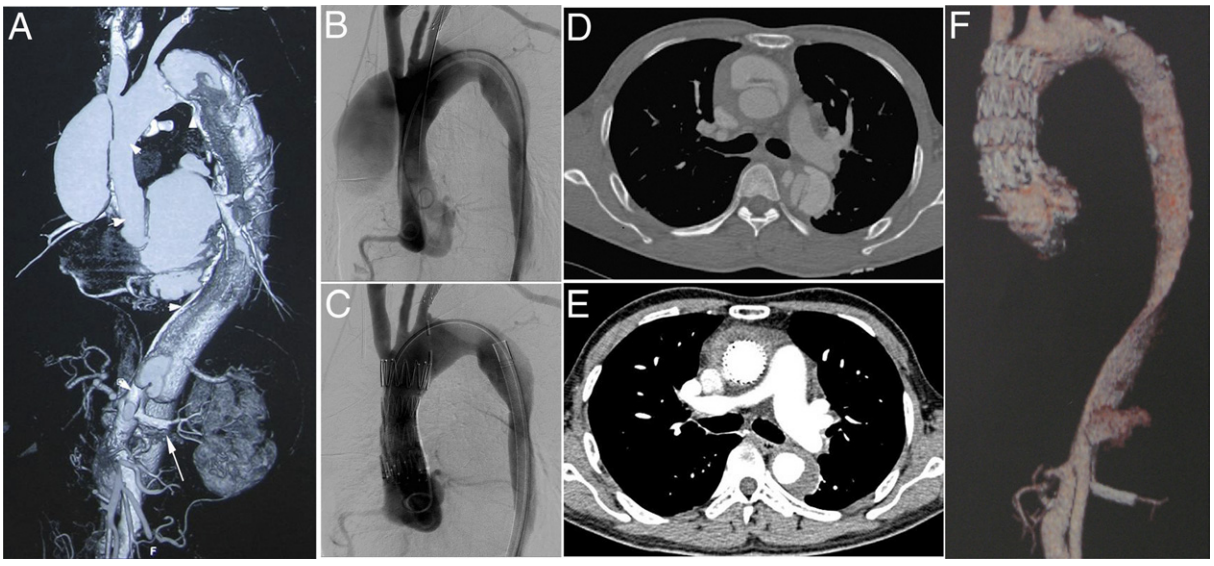

Figure 1 **Ascending Aortic Dissection with Compromised Branch Artery**

(A) Computed tomography angiography (CTA) image obtained before stent grafting showing that the primary stent reopened the left renal artery, which had been compromised by the dissection **(arrows)**. **(B)** Pre-operative aortography showing the aneurysmal expansion of the ascending aortic dissection. **(C)** Completion aortography showing that the entry tear was completely excluded. Comparison between **(D)** pre-operative CTA image and **(E)** CTA image obtained at the 24-month follow-up at the same level of maximal ascending aorta showing that the diameter of ascending aorta shrank and the true lumen expanded. **(F)** Follow-up CTA image confirming complete thrombosis of the false lumen along the ascending aorta and the patency of the coronary arteries and the supra-arch branch arteries.

follow-up diameter was 27.4 ± 3.8 mm (p = 0.068). Mean maximal diameter of the ascending aorta significantly decreased from 56.88 ± 6.7 mm before the stent grafting to 45.51 ± 4.6 mm during follow-up (p = 0.001). Further, the diameter of distal ascending aorta significantly decreased from 46.5 ± 11.3 mm to 41 ± 11.7 mm (p = 0.04). The mean diameter of the proximal ascending aorta (50.8 ± 5.9 mm vs. 46.0 ± 5.1 mm, p = 0.214) and mean diameter of the proximal descending aorta (39.2 ± 3.0 mm vs. 36.5 ± 4.4 mm, p = 0.185) were not significantly different between pre-operative and post-operative follow-up measurements (Fig. 3A). True lumen diameter significantly increased during follow-up. Mean diameter increased from 22.21 ± 5.8 mm to 36.37 ± 6.1 mm at the level of maximal

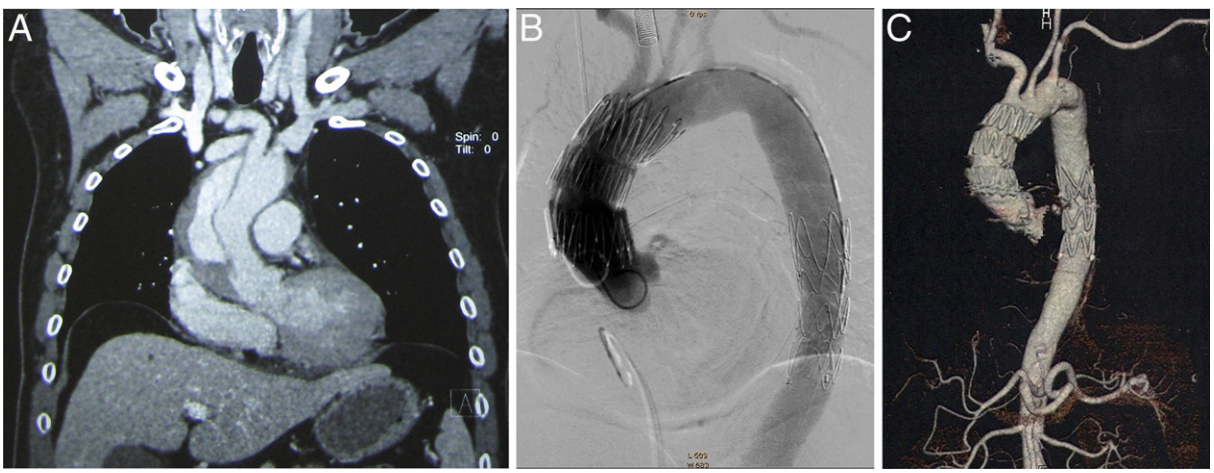

Figure 2 **Exclusion of the Tears in Ascending and Descending Aorta in 1 Procedure**

(A) After 36 days of medical treatment, the patient reported recurrent chest pain and was referred to our institution. Immediate CTA revealed the entry tear located in the middle part of the ascending aorta and that the false lumen was patent. **(B)** Completion aortography confirming that the important branch arteries were patent and **(C)** 24-month CTA image showing that the false lumen was completely thrombosed. Abbreviation as in Figure 1.

Table 2 Intraoperative Measurements and Stent-Graft Choice

Intraoperative measurements (mm)	
Aortic diameter (proximal to the tear)	28.4 ± 7.1
Aortic diameter (distal to the tear)	33.0 ± 4.0
Length from STJ to IA	92.9 ± 12.5
Length from STJ to tear	50.4 ± 22.7
Length from tear to IA	92.9 ± 12.5
Longitudinal length of tear	8.7 ± 7.5
Stent-graft choice	
Stent-graft diameter (mm)	39.0 ± 3.5
Stent-graft length (mm)	78.9 ± 2.0
Stent-graft diameter/proximal landing zone diameter (%)	137.3 ± 11.6
Stent-graft diameter/distal landing zone diameter (%)	116.8 ± 14.4
Stent-graft length/ascending aorta length (greater curvature, %)	84.1 ± 9.3

Values are mean ± SD.
IA = innominate artery; STJ = sinotubular junction.

ascending aortic diameter (p = 0.001), from 25.4 ± 7.1 mm to 33.3 ± 5.5 mm at the level of the proximal ascending aorta (p = 0.003), from 32.7 ± 4.4 mm to 36.3 ± 3.4 mm at the level of the distal ascending aorta (p = 0.022), and from 28.3 ± 2.9 mm to 31.0 ± 3.3 mm at the level of the proximal descending aorta (p = 0.011) (Fig. 3B). The diameter of the false lumen at different levels significantly decreased in the follow-up phase: from 34.1 ± 8.5 mm to 9.6 ± 6.2 mm at the level of the maximal ascending aorta (p < 0.001), from 25.8 ± 8.1 mm to 12.1 ± 6.1 mm at the level of proximal ascending aorta (p < 0.001), from 13.6 ± 6.4 mm to 4.9 ± 3.1 mm at the level of the distal ascending aorta (p < 0.001), and from 11.8 ± 4.2 mm to 5.1 ± 3.2 mm at the level of the proximal descending aorta (p < 0.001) (Fig. 3C). The results of patients treated by open surgery or best medical therapy are presented in Table 3.

Discussion

Several risk models previously were reported that predict surgical mortality for repair of type A aortic dissection. Using these parameters, 10% to 30% of patients with AAD were considered high risk for direct surgical repair and received medical therapy (16,17). Mortality for medically treated patients has been substantially higher than those treated by direct surgical repair (4). One study showed that thrombosed false lumen documented by transesophageal echocardiography in type A aortic dissections is an indicator for survival (19). This implies that closure of the dissection entry tear and subsequent false lumen thrombosis should improve long-term survival in these patients. Our results show that when an endovascular repair induced false lumen thrombosis, the maximum diameter of the ascending aorta decreased and survival during the follow-up period occurred. Sobocinski et al. (5) retrospectively reviewed the CTA images of 102 cases of acute Stanford type A aortic dissections who had undergone open repairs and concluded that approximately half of the cases were potential candidates for endovascular repair.

Endovascular repair of AAD was reported first in 2003 (8). At 1 year of follow-up, the patient evidenced cardiomegaly, severe aortic valvular insufficiency, and impaired left heart contractility documented by echocardiography. This patient was converted to open repair, a Bentall operation, 21 months after the endovascular procedure (10). There have been sporadic reports of other endovascular AAD repairs, some of which describe significant risks for procedure-related cerebral infarction, fatal arrhythmias, myocardial

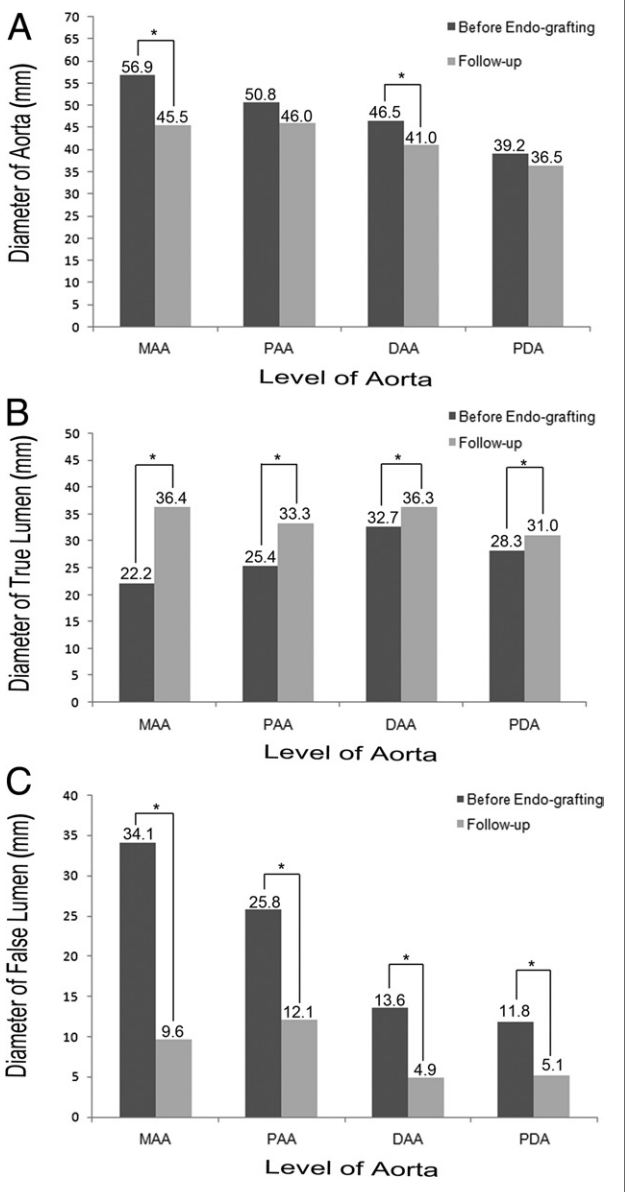

Figure 3 Mean Diameters Before and 12 Months After Endovascular Procedure

Bar graphs showing the mean diameters of **(A)** the total aorta, **(B)** the true lumen, and **(C)** the false lumen compared between pre-operative and 12-month follow-up measurements. The asterisk indicates that the change was significant (p < 0.05). DAA = distal ascending aorta; MAA = maximal ascending aorta; PAA = proximal ascending aorta; PDA = proximal descending aorta.

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Table 3 Outcomes of Open Surgery and Medically Treated Patients

	Age (yrs)	Phase*	Outcome
Open surgery	59.3 ± 3.2	16 acute	1 early death, 1 lung infection and prolonged ICU stay
		5 chronic	1 significant stroke
Medicine	67.5 ± 7.2	1 acute	Died
		4 chronic	1 sudden death

*The acute or chronic phase refers to the time when the patients were referred to our hospital.
ICU = intensive care unit.

infarctions, and cardiac perforation (7,9,10,20,21). In this study, we found that aortic regurgitation could be stable after the endografting for AAD. This may be related to the stable diameter of the sinotubular junction and to the positive hemodynamic change in the ascending aorta when the true lumen was expanded in the follow-up period.

Endovascular repair for AAD certainly is not possible for every patient. In an attempt to define which patients are best suited for endovascular treatment, we divided the ascending aorta into 3 sections. If the entry tear is located in the distal third of the ascending aorta, a branched endograft or hybrid or fenestration procedure is necessary to maintain perfusion to the brachial cephalic vessels (22). Entry tears located in the middle third of the ascending aorta are ideal cases for tube stent-graft repair (7,23). Entry tears in the proximal third of the ascending aorta do not offer a suitable proximal landing zone for the endograft in most cases (21).

A variety of stent grafts have been reported as useful to repair entry tears in the ascending thoracic aorta (e.g., the 46 × 100-mm Talent or Valor graft [Medtronic] and the 40 × 100-mm CTAG graft [W. L. Gore & Associates, Inc., Flagstaff, Arizona]) and a custom-made covered stent of 46 × 85 mm (Jotec, Hechingen, Germany) (7,9,21). Endografts for the ascending aorta must be of relatively short length (100 mm or less) and larger diameter (≥46 mm diameter) (24). Metcalfe et al. (25) presented 1 case of endovascular repair of an acute AAD using an endovascular stent graft manufactured specifically for the ascending aorta. The endograft used has proximal and distal bare stent fixation. We do not recommend proximal bare stent configuration because these exposed metal fixation points could injure the aortic valve and may compromise the ability to achieve an appropriate proximal seal zone (18). Zenith TX2 extension stent grafts (Cook), with a diameter varying from 32 to 42 mm and length ranging from 77 to 81 mm, were used in this study.

Several access sites for the delivery system used for endovascular ascending aortic repair have been suggested (26,27). Chuter (28) reported that the retrograde carotid approach often produced carotid obstruction from the obstructor effect of the delivery system, and therefore increased the risk of cerebral ischemia or infarction. He preferred a conduit attached to the distal innominate artery. We believe that the flexibility of the current stent-graft delivery system allows the device to be passed across the aortic arch in most cases without difficulty. The retrograde femoral route for device delivery is preferred for most cases. When the external iliac or femoral arteries are small, tortuous, or severely stenosed, common iliac or abdominal aortic conduits may be required to introduce the delivery system. Endograft deployment into the ascending aorta retrograde from the apex of the LV also has been reported to be similar to deployment of the Stentor version of the prosthetic aortic valve (18).

The current design of the delivery systems for thoracic aortic endografts offered by most manufacturers is less than optimal for use in the ascending aorta. The long tapered nose cone associated with these devices that facilitates passage of the device through the iliac vasculature and around the aortic arch often requires that this nose cone traverse the aortic valve and enter the LV (8). In our series of 15 patients, everyone had ventricular tachycardia when the delivery system nose cone contacted the wall of the LV. When the delivery system was withdrawn, either conversion to normal rhythm occurred or the patient required chemical or electrical cardioversion. We recommend that temporary cardiac pacing and a defibrillator be readily available for use if necessary during ascending thoracic aortic endografting.

At no other site in the aorta is precise deployment of an endograft more important than when it is used in the ascending thoracic aorta. Preservation of coronary artery flow, aortic valve function, and perfusion of the branch vessels of the aortic arch are critical to successful outcome of these procedures. Elective adenosine-induced cardiac arrest was introduced to decrease cardiac output and to maximize accuracy of device deployment by avoiding systolic blowback of the device during deployment (29). We did not use temporary adenosine-induced arrest in our patients. We planned to use temporary rapid ventricular pacing (heart rate to 180 beats/min) to decrease blood pressure and cardiac output. This technique has been described and used by many authors as a means of insuring accurate deployment of the endograft (7). In our patients, delivery system nose cone contact with the LV wall-induced ventricular tachycardia or fibrillation with resultant hypotension and decreased cardiac output so that we could deploy our endograft accurately. Intraoperative use of intraoperative transesophageal echography has been recommended to assist with precise endograft deployment, especially when the proximal deployment location is very close to the coronary sinus (7,9). There also have been successful cases reported without using transesophageal echocardiography (8,21). The value of this imaging method is related to the experience of the operator and the quality of the imaging system.

In endovascular repair of an abdominal aortic aneurysm, an endostapling system has been developed for treatment of proximal graft migration and type I endoleak (30). To our knowledge, this endostapling system has not been used in the ascending aorta in humans.

Transcatheter aortic valve implantation has become the standard of care under certain circumstances in many

countries. It is conceivable that combining this technology with an endograft may make the possibility of treating AADs associated with compromised aortic valve function a reality (31). It should be noted that multidisciplinary heart and aorta teams, including vascular surgeons, cardiac surgeons, and cardiologists, making the decisions of treatment strategy and cooperating in the procedure are critical to give the patients fittest treatment and to enhance the safety of the endovascular procedure.

Study limitations. This study demonstrates experience with the endovascular repair of AADs in a small group of patients. It also documents the aortic remolding after the endovascular repair. The limitations of this study are the small sample size (n = 15) and the median follow-up of only 26 months. Further study with a larger sample size and longer follow-up are necessary before any significant conclusions can be reached regarding the effectiveness of this form of treatment of AAD. Future studies should begin by comparing the effectiveness of endovascular treatment of AAD with that of best medical therapy. Only if the results of endovascular repair prove durable will a trial comparing this treatment method with direct surgical repair, the current gold standard, be appropriate.

Conclusions

Our results of endovascular repair of AAD have given us an optimistic view of this procedure. In patients who are considered high risk for a potentially poor outcome related to direct surgical repair of their AAD, an endovascular approach may prove to be a very acceptable alternative therapy. Design improvements for the delivery system, especially with respect to the nose cone, will be a necessary consideration for endograft devices planned for use in the ascending aorta.

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