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Aortic remodeling after endovascular repair of acute complicated type B aortic dissection

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Objective: **The role of thoracic endovascular aortic repair (TEVAR) in the management of acute type B aortic dissection remains undefined. Entry tear coverage during the acute phase is an appealing method to treat acute complications, and by inducing false lumen thrombosis, might also prevent late aneurysm formation. This study evaluated structural changes by serial computed tomography (CT) in the thoracic aorta after TEVAR performed for acute complicated aortic dissection.**

Methods: **Between August 2005 and October 2007, 33 patients with complicated acute type B aortic dissection were treated with TEVAR (19 from a prospective industry sponsored trial, 14 from our institution). CT images obtained preprocedurally (PP), at 1 month (1M), and 1 year (1Y) were evaluated for each patient. Four patients with no postprocedural imaging were excluded. The largest diameters of the thoracic aorta, dissection true lumen, and false lumen were recorded at each time point. Canges in total aortic and true and false lumen diameters were evaluated using a mixed effect analysis of variance model of repeated measures.**

Results: **The average age was 58 years (range, 38-87 years); 26 (81%) were male. Indications for TEVAR included malperfusion syndrome in 17 (53%), refractory hypertension in 14 (44%), impending rupture in 12 (28%), and refractory pain in 14 (44%); 19 (59%) had more than one indication. The average length of aorta covered was 19.5 cm (range, 10-29.3 cm). The maximum aortic diameter decreased over time ($P = .04$) and averaged 39.9 (PP), 41.3 (1M), and 34.8 mm (1Y). The true lumen diameter increased over time ($P = .02$) and averaged 23.7 (PP), 29.0 (1M), and 31.1 mm (1Y). The false lumen diameter decreased ($P = .046$) and averaged 19.5 (PP), 12.1 (1M), and 9.6 mm (1Y). Partial or complete thrombosis of the false lumen along the stented segment of aorta was recorded in 87% (PP), 93% (1M), and 88% (1Y).**

Conclusions: **TEVAR of acute complicated aortic dissection appears to promote early aortic remodeling. Nearly 90% of patients maintained at least partial false lumen thrombosis at 1 year. Because continued false lumen patency correlates strongly with late aneurysm formation, such favorable remodeling is considered a surrogate for prevention of late aneurysm, but longer follow-up is required. (J Vasc Surg 2009;50:510-7.)**

Acute aortic dissection (AD) is the most common catastrophic event affecting the aorta, with an incidence exceeding that of ruptured abdominal aortic aneurysm. Indeed, Khan et al[1] predicted that the mortality rate of acute AD left untreated will exceed 22.7% ≤6 hours, 50% ≤24 hours, and 68% ≤ 1 week, with death from dissections of the descending thoracic aortic caused by aortic rupture or, more commonly, end organ compromise due to obstruction of visceral or extremity vessels (malperfusion syndrome).[2,3] The morbidity and mortality for complicated Stanford type B AD (dissection distal to the origin of the left subclavian artery) greatly exceeds that of type B AD without such compromise (ie, those treated medically).[4,5] Indeed, 85% to 90% of patients who present with uncomplicated type B AD are discharged with medical therapy alone, whereas >50% of patients with complicated AD are at risk of dying and require an emergency intervention of some kind.[6]

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Traditionally, patients who present with type B AD complicated by impending rupture or a malperfusion syndrome were treated with open graft replacement of the proximal descending thoracic aorta, resulting in a mortality rate of 6% to 69% in several large series.[7-10] Because of the high morbidity and mortality associated with open surgery in the acute phase, medical management centered around blood pressure control is the accepted standard of care for patients with uncomplicated acute type B AD. Unfortunately, aneurysmal dilation of the false lumen will develop in 25% to 40% of medically managed patients ≤5 years.[11-13]

In 1999 the endovascular treatment of complicated acute type B dissections (TEVAR) with stent graft coverage of the proximal entry tear was described in two sentinel reports.[14,15] This approach has successfully decreased the perioperative mortality rate in such patients, but few data exist regarding the long-term aortic structural changes after TEVAR.[16,17] This study evaluated structural changes (aortic remodeling) in the thoracic aorta after TEVAR performed for acute complicated type B dissection.

METHODS

The study population included 33 patients who underwent TEVAR for complicated acute Stanford type B AD between August 2005 and October 2007. Of these, 19 patients were treated as part of a prospective, multicenter industry-sponsored trial (TAG-0401), and the remaining

14 patients underwent TEVAR at our institution. Demographic and clinical factors were collected for each patient, as were 30-day and 1-year mortality data. Hypertension, chronic obstructive pulmonary disease, and history of stroke were considered present if they were part of the patient's history regardless of current medications or past interventions. Renal insufficiency was defined as a baseline creatinine ≥1.5 mg/dL, and patients were considered to have coronary disease even if they had undergone revascularization in the past. Clinical end points for the study include mortality and anatomic follow-up based on serial computed tomography (CT) scanning.

Massachusetts General Hospital cohort. During the study interval, 16 patients underwent TEVAR for acute complicated type B AD at Massachusetts General Hospital (MGH). Two of these patients were enrolled in the TAG-0401 study and the remaining 14 comprise the MGH cohort. All patients included in the MGH cohort met inclusion criteria for the TAG-0401 study. Data for these patients were collected prospectively but reviewed in a retrospective fashion for this study. Patients were included if they underwent TEVAR for type B AD complicated by malperfusion with end-organ ischemia, hypertension refractory to medical therapy, aortic rupture or impending rupture, or persistent pain despite adequate blood pressure control. These conditions were chosen because they were the inclusion criteria for the TAG-0401 trial.

TEVAR was performed in all patients ≤14 days of onset of symptoms so that all AD were truly acute at the time of therapy. All patients underwent preoperative CT scanning in preparation of TEVAR, and our follow-up protocol included CT scanning at 4 to 6 weeks and then at 6 months and 1 year after the procedure.

Digital diameter measurements of the descending thoracic aorta between the left subclavian artery to the celiac access were obtained at each scanning time point using our in-house image viewing software (Amicus Inc, Austin, Tex). These included the largest total descending thoracic aortic diameter, the largest true lumen diameter, and the largest false lumen diameter. Every attempt was made to obtain a true diameter measurement, and aortic angulation was taken into account when determining the location for each measurement; however, software that allows automatic centerline measurements was not used. Each individual measurement was not necessarily taken at the same level in the aorta so that addition of the maximum true and false lumen diameters could result in a number that exceeds the largest total aortic diameter measured.

In addition to diameter evaluation, the false lumen along the treated segment of aorta was evaluated for the presence of thrombosis and was characterized as none and partial/complete. This method of anatomic evaluation was used to conform with the TAG-0401 protocol. The MGH Institutional Review Board (IRB) approved this protocol and individual consent was waived.

TAG-0401. The Gore TAG-0401 study is a prospective, nonrandomized, literature controlled, multicenter trial of TEVAR of complex aortic pathologies that enrolled patients between August 2005 and February 2007. The study has three arms, and the 19 patients enrolled in the acute complicated type B AD arm are included in this patient cohort. As part of the inclusion criteria, all patients patients were considered candidates for open repair at the time that they underwent TEVAR. The 19 patients in this arm of the study were treated at six institutions, including two patients from MGH. All patients presented with acute complicated type B AD, as detailed in the MGH cohort.

Patients underwent imaging with noncontrast and contrast-enhanced, thin-sliced (range, 1-2.5 mm) spiral CT with multi-planar reconstruction of the chest, abdomen, and pelvis to confirm study eligibility and for preprocedural planning. Patients were then monitored with serial CT scans at 30 days, 6 months, 1 year, and yearly afterward, with planned completion at 5 years. Additional imaging studies were performed at the discretion of the treating physician.

Clinical data were reported by individual centers that were monitored by sponsor representatives. Image analysis, including serial diameter measurement, was performed by the imaging services department of the sponsor. The protocol and procedures of the TAG-0401 trial were approved by the United States Food and Drug Administration (FDA) and the individual IRBs of each participating center. Patients, or their surrogates, signed a research informed consent before participation in this study.

MGH operative details. TEVAR at MGH is performed in an endovascular operating suite equipped with digital subtraction angiography by participating vascular surgeons. General anesthesia was used for all procedures, and spinal drains were placed at the discretion of the operating surgeon. Stent grafts were introduced into the aorta through a cutdown on the common femoral artery. Iliac conduits were used in patients with unsuitable iliac vessels for stent graft delivery (2 patients in the MGH cohort).

Several technical components of the procedure should be detailed here. All patients underwent seal of the proximal entry tear at the level of the left subclavian artery with the Gore TAG covered stent graft (W.L. Gore and Associates, Flagstaff, Ariz). True lumen access was obtained from a brachial or femoral approach. Because the tear in the type B dissection is distal to the left subclavian artery, rapid true lumen access is easily obtained through a right transbrachial approach. In the MGH cohort, intravascular ultrasound (IVUS) imaging was performed from the aortic arch to the iliac vessels to define the relationship of the dissection flap to the visceral and renal arteries and confirm true lumen position at the level of the left subclavian artery (Fig 1).

Angiography was performed sparingly and limited to hand injections of selected branch arteries to prove that the catheter/wire was beyond the dissection and in the true lumen of the healthy artery. This practice limits the contrast load during the procedure, thus avoiding further insult to the kidneys. In addition, power injection in the true lumen of the dissected aorta may give the false impression of adequate perfusion to branch vessels compromised by

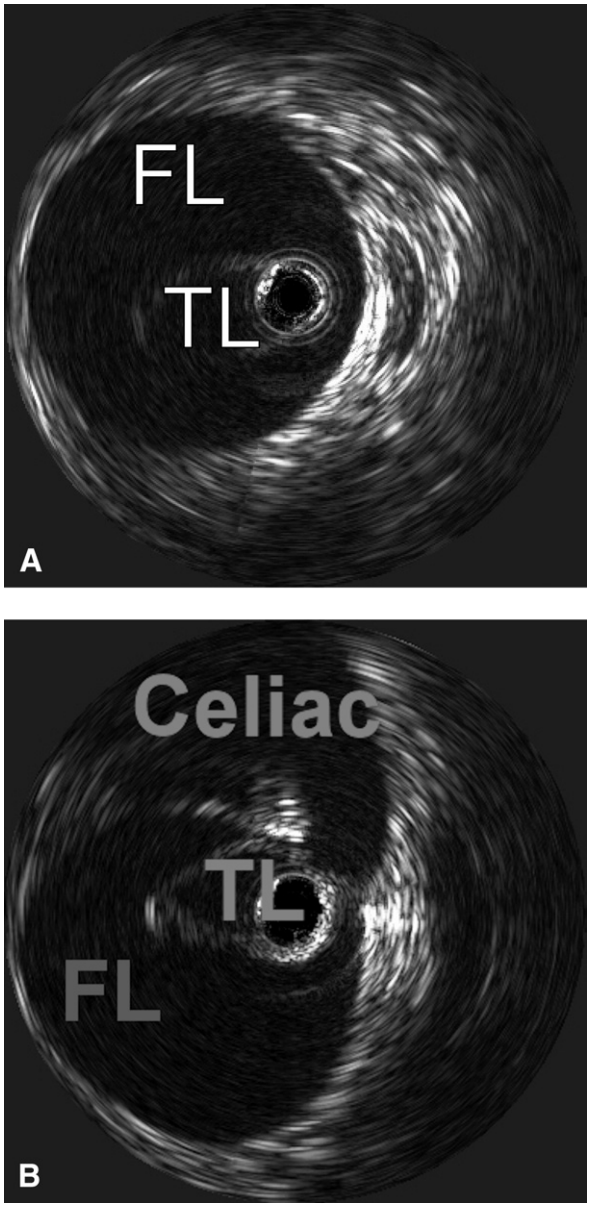

Fig 1. Intravascular ultrasound (IVUS) evaluation of the thoracic aorta. **A,** The true lumen *(TL)* is compressed and free floating in the thoracic aorta, and the false lumen *(FL)* is larger than the TL. IVUS has been used to confirm that the wire is in the true lumen. **B,** The relationship of the dissection flap/true lumen (TL) to the celiac artery is shown.

dynamic obstruction. Objective evidence of malperfusion was confirmed by the presence of a gradient between the aortic root and branch (not aorta) end-hole catheter pressures, as described by Barnes et al.[18]

Endografts were deployed proximally with or without coverage of the left subclavian artery to obtain an adequate seal of the entry tear. Most patients were treated with one endograft prosthesis; additional pieces were placed only when the initial graft did not produce the desired result of coverage of the entry tear and expansion of the true lumen as determined by intraoperative IVUS imaging and angiography (Fig 2). Bare metal stents were placed at the origin of the visceral and renal vessels when necessary to ensure adequate end-organ blood flow.

Statistical analysis. Descriptive statistics were performed on the available variables for the entire study cohort of 33 patients. Differences in aortic diameter measurements over time were determined using a mixed-effect analysis of variance model of repeated measures. A value of $P = .05$ was considered significant.

RESULTS

The study cohort comprised 33 patients. Available demographic and clinical factors are detailed in Table I. The two groups were similar except for prevelance coronary disease, which was present only in the MGH cohort. All patients underwent TEVAR for complications related to their acute dissection, as summarized in Table II. The average length of aorta covered was 19.5 cm (range, 10-29.3 cm).

Four patients died (3 from TAG-0401 and 1 from MGH) for a 30-day mortality of 12%. Two patients died of complications related to periprocedural strokes, and one died of a postprocedural aortic rupture. The third patient's recovery was complicated by respiratory failure and flaccid lower extremity paralysis and the family withdrew care. Two patients (6%) presented with lower extremity weakness after TEVAR. The first was the patient who died, and the second experienced transient weakness that resolved with spinal drainage and hypertensive therapy. In-hospital complications are summarized in Table III. During the 1-year follow-up, an additional four patients died for a total 1-year mortality of eight (24%).

Complete radiographic follow-up was available on 21 of the 25 patients who remained alive at 1 year. The average maximum aortic diameter showed a slight increase from the preprocedural scan to the 1 month follow-up, but then decreased by 1 year leading to a significant decrease over time, respectively, of 39.9, 41.3, and 34.8 mm ($P = .04$; Fig 3). The average maximum diameter in mm of the true lumen significantly increased over time from 23.7 postproceduraly to 29 at 1 month to 31.1 at 1 year ($P = .004$; Fig 4). The average maximum false lumen diameter in mm significantly decreased, respectively, from 19.5 postproceduraly to 12.1 at 1 month to 9.6 at 1 year ($P = .0001$; Fig 5).

The initial diameter of the false lumen was not predictive of change in aortic diameter at 1 year. Partial or complete thrombosis of the false lumen along the treated aortic segment was recorded in 87% of patients who had an immediate postprocedural scan, in 93% at 1 month, and in 88% at 1 year. False lumen expansion occurred in four patients during follow-up, and two of these had no thrombosis of the false lumen.

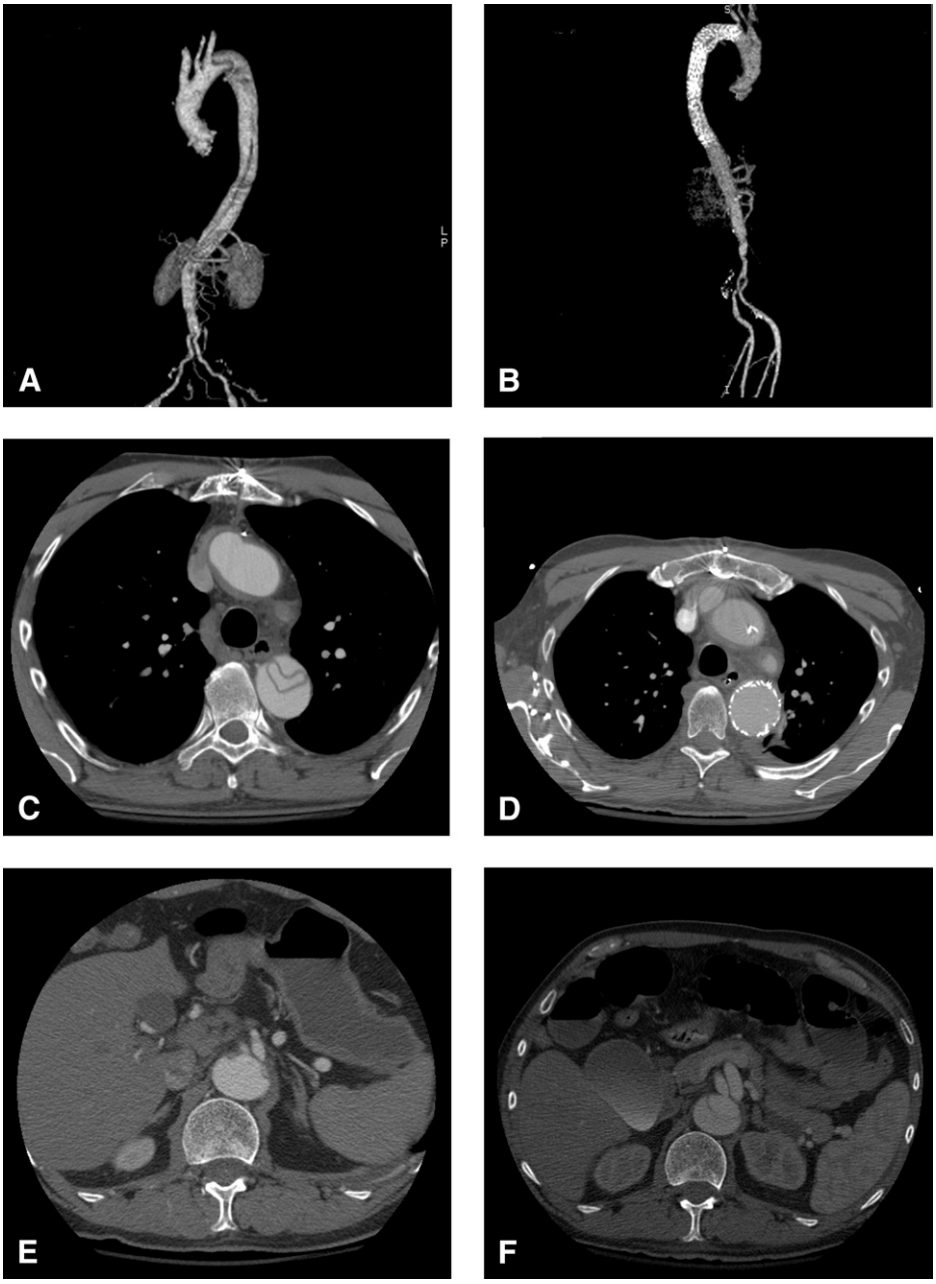

Fig 2. Computed tomography angiogram of the aorta. **A,** Three-dimensional reconstruction of acute aortic dissection shows compressed true lumen. **B,** Three-dimensional reconstruction shows dissection after placement of endograft. **C,** Axial image of dissection shows compressed true lumen that is completely detached from the surrounding adventitia. **D,** Axial image of thoracic aorta in same patient after endografting shows expansion of the true lumen with no flow in the false lumen. **E,** Axial image shows the celiac axis with compressed true lumen. **F,** Axial image shows the celiac axis in same patient after placement of proximal endograft. The true lumen has now expanded.

DISCUSSION

The optimal management of acute type B AD remains a topic of ongoing investigation. The ability of TEVAR to accomplish the anatomic goal of surgical graft replacement of the proximal entry tear has engendered a new era in the treatment of acute type B AD. Treatment options have evolved first from the precept that medical therapy produced equivalent outcomes to surgical resection of the entry tear.[19] Next, it has been amply demonstrated that uncomplicated patients who can be medically treated have favorable early outcomes.[4,5] Finally, and as intuitively expected, patients with secondary complications

Table I. Demographic and clinical data for 33 patients undergoing thoracic endovascular aneurysm repair for acute, complicated type B aortic dissection

Variable	*Total, No. (%)*	*TAG-0401, No. (%)*	*MGH, No. (%)*	P
Male gender	26 (79)	16 (84)	10 (71)	.37
White race	25 (76)	15 (79)	10 (71)	.62
Hypertension	31 (94)	18 (95)	13 (93)	.82
COPD	6 (18)	3 (16)	3 (21)	.68
CAD	4 (12)	0 (0)	4 (29)	.01[a]
History of stroke	5 (15)	3 (16)	2 (14)	.91
Renal insufficiency	6 (18)	3 (16)	3 (21)	.68
Age, y	58	58.9	58.1	.88

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *MGH,* Massachusetts General Hospital.
[a]Significance.

Table II. Indications for thoracic endovascular aneurysm repair in 33 patients

Indication	*No. (%)*
Malperfusion syndrome	17 (53)
Refractory hypertension	14 (44)
Impending rupture	12 (28)
Uncontrolled pain	14 (44)
>1 indication	19 (59)

Table III. Complications after thoracic endovascular aneurysm repair for acute type B dissection

Complication	*No. (%)*
≥1 major event	25 (76)
Ruptured aorta	2 (6)
Endoleak	3 (9)
Bleeding	2 (6)
Additional dissection	3 (9)
Cardiac	5 (15)
Pulmonary	8 (24)
Renal insufficiency	4 (12)
Cerebrovascular	4 (12)
Deep vein thrombosis/pulmonary embolus	2 (6)
Spinal cord ischemia	1 (3%)
Groin hematoma	2 (6%)

of their dissection fare significantly worse than uncomplicated cases.[3,20,21]

Despite advances in anesthesia and operative techniques, death after open repair of the descending aorta for patients with branch vessel compromise remains high.[7-10,22] A recent review of 476 patients with acute type B AD from the International Registry of Acute Aortic Dissection (IRAD) database showed that 82 (17.2%) were treated surgically with descending aortic replacement (70%), partial arch replacement (21%), or total arch replacement (8%).[10] The perioperative mortality rate of this cohort was 29.3%, with the highest death rate occurring in patients who required operation ≤24 hours of presentation. The

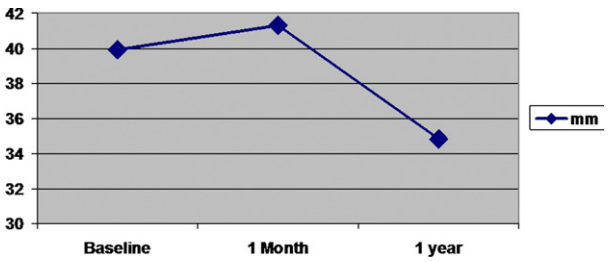

Fig 3. Changes in maximal aortic diameter were significantly different over time. ($P = .04$).

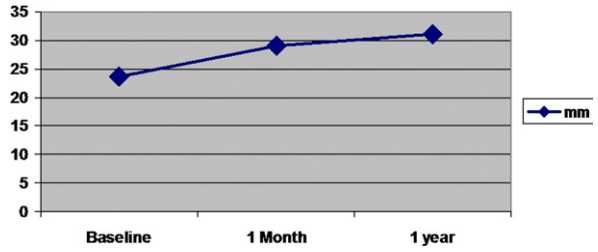

Fig 4. Changes in maximal true lumen diameter were significantly different over time ($P = .004$).

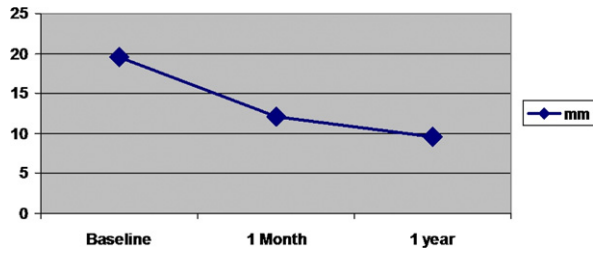

Fig 5. Changes in maximal false lumen diameter were significantly different over time ($P = .0001$).

30-day mortality rate of 12% in the current study compares favorably with this contemporary series of open thoracic aortic replacement. An additional IRAD study showed that the in-hospital mortality was 30.8% in patients who underwent surgical therapy compared with 9.1% for endovascular repair ($P < .001$), making TEVAR an attractive option when possible.[23]

Early reports of TEVAR have been favorable for complicated acute type B AD.[14-16] From a practical perspective, however, stent graft repair of AD in the United States has only been available since April 2005, coincident with initial FDA approval of a commercially available thoracic stent graft. To date, the application of available TEVAR devices in AD outside of clinical trials constitutes off-label usage because such devices are currently only FDA-approved in the United States for treatment of degenerative thoracic aneurysms. Although the low mortality rate seen with initial reports has made TEVAR the first line of therapy at MGH for patients with complicated acute type B ADs, the

role of TEVAR in patients with uncomplicated AD remains unclear.

To potentially clarify (although it has not) the role of stent graft treatment of type B AD, the Investigation of Stent-grafts in Aortic Dissection (INSTEAD) trial was designed as a prospective, randomized, multicenter trial performed in Europe that compared stent grafting with medical therapy for the treatment of chronic uncomplicated type B AD. All patients were randomized 2 weeks after symptom onset; thus, patients with early complications—those who would most likely benefit from endovascular therapy—were essentially subtracted from the study cohort. Not surprisingly, the investigators found no difference in 30-day mortality between the two groups. In fact, the 1-year survival probability was 91% in TEVAR patients vs 97% with medical treatment ($P = .16$), reflecting a periprocedural mortality rate of 3%.

The encouraging data reported in this trial were with respect to aortic remodeling, which may be considered a surrogate for prevention of late aneurysm formation. To wit, >90% of patients in the stent graft cohort experienced false lumen thrombosis at 1 year, which was significantly higher than the medically treated group.[24] This number was similar to our results of 88% of patients with partial or complete false lumen thrombosis at 1 year.

Although most late deaths are related to comorbid conditions, subsequent complications such as aneurysmal development and late rupture have been estimated to occur in 20% to 50% of patients.[25-27] The identification of a higher false lumen thrombosis rate in the TEVAR cohort of the INSTEAD trial is promising, because persistent false lumen patency is an independent risk factor for aneurysmal degeneration of medically treated dissections.[14,28-32] Indeed, Sueyoshi et al[33] showed that a patent false lumen will enlarge 3.3 mm/year even in stable, uncomplicated cases.

In a review of the IRAD data, Tsai et al[32] showed that partial thrombosis of the false lumen in medically treated patients is an independent predictor of death after discharge, with partial thrombosis having the worst results at 31.6% compared with 13.7% for patent and 22.6% for complete thrombosis.[32] The proposed mechanism of this is that formation of thrombus may occlude the distal outflow fenestrations, leading to a significant increase in the mean arterial and diastolic pressure in the false lumen compared with that of a patent lumen with adequate outflow or one that is totally thrombosed.[32]

This model was based on the assumption that partial false lumen thrombosis occurs in the distal portion of the dissection with continued pressurized perfusion proximally. In theory, TEVAR should address this problem, because coverage of the proximal entry tear promotes proximal thrombosis so that persistent flow through the patent distal false lumen should be associated with lower pressures. Thus, the concept of inducing false lumen thrombosis by sealing the aortic tear with an aortic endograft has the potential to reduce early and late complications of type B AD, and in our study, the false lumen was at least partially thrombosed in 88% of patients at 1 year.

The goals of TEVAR for acute type B AD include coverage of the proximal entry tear, expansion of the true lumen with restoration of flow to the visceral vessels, and obliteration of false lumen flow with subsequent complete thrombosis. This concept of false lumen obliteration and true lumen expansion has been termed aortic remodeling and stabilization of the aorta. When these components of therapy are successful, aortic remodeling should occur, with subsequent prevention of future aneurysmal degeneration of the outer wall of the false lumen. Indeed, the false lumen measurements in our study decreased over time and the true lumen increased, resulting in early remodeling as evidenced by the decrease in the thoracic aortic diameter. Thus, although there has been appropriate emphasis on TEVAR to treat acute complications, our data and that of others indicate that it also has great promise to prevent the principle late complication of aneurysm formation.[16,34]

Several recent studies have looked at early aortic remodeling after TEVAR for acute aortic dissection. Schoder et al[17] detailed 28 patients with acute type B AD treated during a 5-year period in Europe. They divided the aorta into three segments related to the area of stent graft coverage and found a significant decrease in the false lumen diameter in the stented area without a change in the maximum aortic diameter.[17] Several other series reported diameter measurements at specific anatomic locations along the aorta after TEVAR for acute type B AD. They showed a significant reduction in false lumen diameter over time and an increase in true lumen, with the greatest changes noted along the proximal one-third of the aorta.[16,34]

Division of the aorta into specific sections was not possible with the current cohort of patients because of the constraints of the prospective trial; however, our findings of continued false lumen shrinkage with true lumen expansion support the notion of early aortic remodeling after TEVAR.

There is currently no consensus regarding the best way to measure or report aortic changes after TEVAR. Although many studies, including this one, have used maximal aortic diameters because they are the most relevant to clinical decision making, others have argued that volumetric measurements are a more accurate way to characterize aortic remodeling. Czermak et al[35] have published several reports of TEVAR for acute type B AD that describe changes in true and false lumen volumes over time at specific locations along the aorta. They found that the true lumen volume significantly increased (nearly 59%) ≤12 months, whereas the false lumen volume significantly decreased over time. They also noted that length of stent graft, percentage of stented dissection length, and origin of visceral vessels did not predict changes in these volumes.[35,36] Whether diameter or volumetric measurements are the most accurate way to describe aortic remodeling remains to be seen and will likely be the topic of future study.

This study has several limitations. The addition of our single-center experience to the prospective, controlled trial data could inject bias into the results because 50% of the patients are now from a single institution. In addition,

because the trial data will be described in detail in a separate report, we were unable to relate demographic and treatment factors to follow-up measurements, so that no predictors of remodeling could be evaluated. Finally, the follow-up period is short for the evaluation of aneurysmal degeneration, and longer studies will confirm durability of our early results.

CONCLUSIONS

The use of TEVAR in the treatment of aortic dissections holds promise to reduce both early complications and the progression over time to aneurysmal degeneration by early aortic remodeling. The instantaneous and dramatic occurrence of false lumen thrombosis by coverage of the aortic entry tear appears to be a key determinant in the process; the significance of small distal fenestrations remains unknown. These results support the notion that comparative clinical trials are needed to clarify the role of TEVAR in both complicated and uncomplicated acute distal dissections.

AUTHOR CONTRIBUTIONS

Conception and design: MC, CK, DB, TB, RPC
Analysis and interpretation: MC, RSC, TB, RPC
Data collection: MC, CK
Writing the article: MC, RSC, CK, DB, TB, RPC
Critical revision of the article: MC, RSC, CK, DB, TB, RPC
Final approval of the article: MC, RSC, CK, DB, TB, RPC
Statistical analysis: MC, RSC
Obtained funding: Not applicable
Overall responsibility: MC

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