Patient outcomes and thoracic aortic volume and morphologic changes following thoracic endovascular aortic repair in patients with complicated chronic type B aortic dissection

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Objective: **True and false lumen changes and patient outcomes following thoracic endovascular aortic repair (TEVAR) for patients with stable type B dissection have been described by the The Investigation of Stent Grafts in Aortic Dissection (INSTEAD) trial. However, these changes have not been described in TEVAR patients treated for complications of chronic dissection.**

Methods: **A single-institution study was conducted of 73 prospectively evaluated patients treated for complications of chronic type B dissection from 2002 to 2010. Spiral computed tomography reconstructions using M2S (Medical Media Systems, West Lebanon, NH) were analyzed for sequential changes in aortic volume and diameter during patient follow-up. Changes in aortic volume and diameter were tabulated as a percent change from preoperative values. Patient outcomes were determined by sequential evaluations postprocedure.**

Results: **TEVAR was successfully performed in 72 out of the 73 patients (99%). Indications for intervention were aortic enlargement (n = 62), failure of medical management (n = 7), and perforation (n = 4). The 30-day all-cause mortality rate was 14%; events were due to retrograde dissection (n = 4), cardiac-related (n = 4), and rupture (n = 2). Eleven out of the 72 patients (15%) required a secondary procedure for endoleak (n = 7) and persistent distal perfusion of the false lumen (n = 4). Mean percentage expansion of the thoracic true lumen was noted during the follow-up period: 38%, 46%, 71%, and 114% at 1-, 3-, 6-, and 12-month follow-up, respectively. Concomitant regression of the thoracic false lumen of −65%, −68%, −84%, and −84% was observed at the same intervals, respectively. Patients with an initial extension of the thoracic dissection into the infrarenal aorta (n = 46) had an increase in mean percentage change of aortic diameter and volume to 21% and 17% at 1 year, respectively. By contrast, in the patient group without infrarenal dissection (n = 14), the infrarenal aortic diameter and volume remained relatively unchanged at 3% and −0.9%, respectively, at 1-year postintervention.**

Conclusions: **TEVAR is a potential treatment option for patients experiencing complications of chronic type B dissection. During follow-up, there is a predictable expansion of the thoracic true lumen and regression of the thoracic false lumen. These findings correlate with those of the INSTEAD trial, which demonstrated false lumen regression and true lumen expansion in a cohort of patients with stable type B dissection. However, many patients with extension of thoracic dissection into the infrarenal aorta demonstrate continued aortic dilation and, on occasion, the need for secondary intervention for persistent distal perfusion. Further analysis is needed in this subgroup of patients so as to better determine potential predictors and the clinical significance of post-TEVAR infrarenal expansion. Moreover, further investigations may support a role for secondary endovascular intervention in remedying persistent infrarenal aortic expansion after TEVAR for chronic dissection. (J Vasc Surg 2012;56:644-50.)**

Thoracic endovascular aortic repair (TEVAR) has emerged as a treatment option in the management of aortic dissection.[1] TEVAR is being considered as the front-line therapy for patients with acute descending thoracic dissection with malperfusion, provided that timely endovascular intervention is available. Recent series suggest that TEVAR is a promising treatment in the setting of acute type B dissection with malperfusion, rupture, chest pain, and acute aortic enlargement.[2,3] In this group of patients, there appears to be favorable and predictable aortic remodeling during follow-up after intervention.[2] Outcome studies suggest that mortality rates at 30 days and 1 year following TEVAR for acute complicated type B dissection are acceptable, compared with surgical and medical treatment options.[3]

In the setting of chronic type B dissection, defined by the presence of aortic dissection for greater than 2 weeks, endovascular stenting has been debated.[4] Of those patients with uncomplicated chronic type B dissection, series have shown that medical management is equal, if not superior, to TEVAR. The Investigation of Stent Grafts in Aortic Dissection (INSTEAD) trial suggests that endovascular stenting results in a predictable diametric regression of the false lumen and expansion of true lumen in patients with uncomplicated type B dissection. Despite these changes,

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the 2-year data from the INSTEAD trial concluded that TEVAR in the setting of stable chronic dissection does not improve survival nor adverse events, in comparison to medical therapy.[5] However, recently presented, yet unpublished 5-year data from the INSTEAD trial show significant crossover of the medically treated patients to the intervention group.[6] The role of TEVAR in patients with complicated chronic aortic dissection, as defined by aortic enlargement, persistence of symptoms despite medical therapy, and perforation, is largely unknown.

The purpose of our study is to analyze a cohort of patients who underwent TEVAR for complications of chronic type B dissection in a Food and Drug Administration (FDA)-approved, single-center study. Our analysis includes patient outcomes, reintervention rates, and aortic remodeling with follow-up.

METHODS

A total of 73 patients with complicated chronic aortic dissection were treated as part of an FDA-approved single-center investigational device exemption (IDE) from 2002 to 2010 at Harbor-UCLA Medical Center in Torrance, California. Indications for intervention included aneurysmal enlargement (n = 62), failure of medical management (n = 7), and perforation (n = 4). Aneurysmal enlargement was defined as (1) thoracic aneurysm size of 6 cm or greater or (2) increase in maximal thoracic aortic diameter of >0.5 cm per year on surveillance imaging. Patients categorized as failing medical management were those who had intractable chest pain, deteriorating renal function, or refractory hypertension despite adequate control of blood pressure. Patients fell into the perforation group if they had a known type B dissection and evidence of aortic perforation on computed tomography (CT) imaging.

Endovascular devices used at the time of procedure were endografts manufactured by Medtronic, Inc (Santa Rosa, Calif) and included Talent from 2002 to 2008 and Valiant Captivia from 2008 to 2010. Following TEVAR, patients were prospectively followed at 1, 3, 6, and 12 months, followed by yearly thereafter unless follow-up findings warranted more frequent observation. Devices were deployed via an open femoral approach. Both intravascular ultrasound and angiography were used to assist in device deployment. Spiral CT imaging was conducted at each interval with aortic diameter, volume and device configuration using M2S (Medical Media Systems, West Lebanon, NH). A sequential chart review was conducted to assess patient characteristics, outcomes, and reintervention rates.

Measures of aortic remodeling included maximal thoracic aortic diameter, thoracic aorta true lumen volume, thoracic aorta false lumen volume, maximal infrarenal aortic diameter, and infrarenal aorta total volume. In addition, values for maximal thoracic true lumen diameter and maximal thoracic false lumen diameter were analyzed. Values were tabulated as a percent change from preoperative aortic diameter and volume measurements.

Table I. Patient information

Average age	58 years
Number of male patients	52 (71%)
Number of female patients	21 (29%)
Average ASA score	3
Average follow-up	>18 months
Intervention for aortic enlargement	62 (85%)
Intervention for failure of medical management	9 (12%)
Intervention for aortic rupture	2 (3%)

ASA, American Association of Anesthetists.

Table II. Patient outcomes

Procedure success	72/73 (99%)
30-day procedure-related mortality	10/73 (14%)
Aortic rupture	2/10 (20%)
Retrograde dissection	4/10 (40%)
Cardiac events	4/10 (40%)
1-year all-cause mortality	14/73 (19%)
Stroke rate	1/72 (1%)
Paraplegia rate	1/72 (1%)
Reintervention rate	11/72 (15%)
Endoleak	7/11
Persistent distal perfusion	4/11

Statistical analysis was performed via MedCalc software; a χ^2 normal distribution was performed on collected data and comparison of means was performed via *t*-test. Graphical analysis was conducted using Microsoft Excel.

RESULTS

TEVAR was successfully performed in 72 out of the 73 patients (99%) with complicated chronic type B dissection. The procedure was aborted in one patient as a result of excessively tortuous anatomy. Average patient age was 58 years and average preoperative patient American Association of Anesthetists (ASA) score was 3.2. At 30 days, the procedure-related mortality rate was 14%. Causes included retrograde dissection (n = 4), cardiac-related (n = 4), and rupture (n = 2). Two patients had significant procedure-related morbidity: one patient had a postoperative stroke and another patient had paraplegia following TEVAR (Table I).

Mean patient follow-up was 18 months. During this follow-up period, a total of 11 out of the 72 patients (15%) required a secondary intervention. The two determinants for reintervention were endoleak (n = 7) and persistent distal perfusion (n = 4). All seven patients with endoleak were treated with a second endoluminal procedure. Two of the patients with persistent distal perfusion were managed via endoluminal stent while the other two required a debranching procedure and abdominal aortic exclusion. All-cause mortality rate at 1 year was 19%; 1-year procedure-related mortality was 14%, unchanged from the 30-day procedure-related mortality. There were no procedure-related deaths following reintervention (Table II).

Expansion of the true lumen, compared with preoperative measurements, was noted in the follow-up period following TEVAR. Thoracic true lumen volume expanded

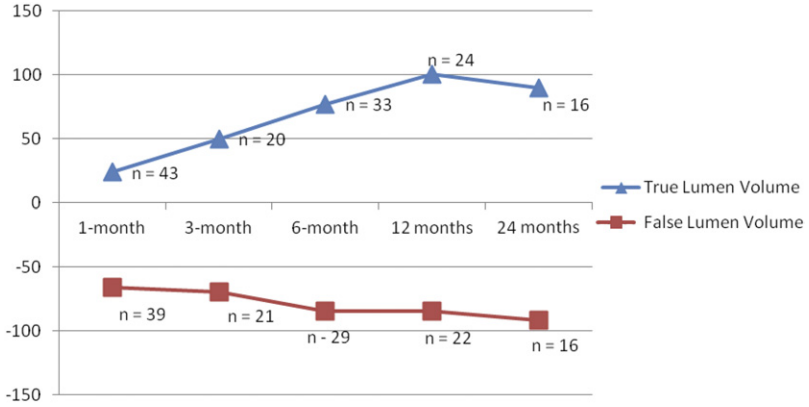

Fig 1. Percent change of thoracic true lumen and thoracic false lumen volume over time.

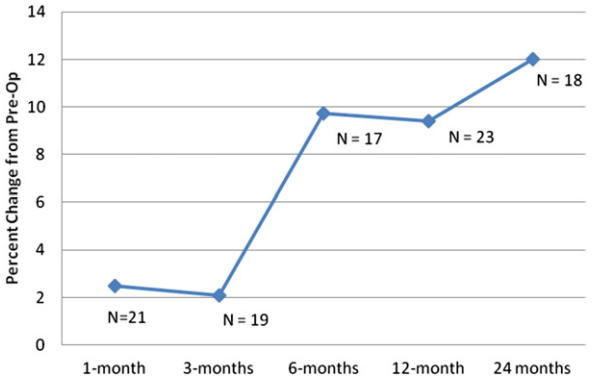

Fig 2. Percent change in thoracic true lumen diameter over time.

38% (relative standard deviation [RSTD] = 3.1), 46% (RSTD = 2.1), 71% (RSTD = 1.3), and 114% (RSTD = 1.2) at 1-, 3-, 6-, and 12-months postprocedure, respectively (Fig 1). True lumen diameter also increased during follow-up: 2.5% (RSTD = 8.8), 2% (RSTD = 14.4), 10% (RSTD = 3.0), and 9.5% (RSTD = 1.9), respectively (Fig 2). Concomitant regression of the thoracic false lumen was observed during patient follow-up; thoracic false lumen volume regressed −65% (RSTD = 0.59), −68% (RSTD = 0.66), −84% (RSTD = 0.27), and −84% (RSTD = 0.13) at 1-, 3-, 6-, and 12-month follow-ups. Measures of maximal thoracic false lumen diameter also showed regression over the follow-up period: −24% (RSTD = 1.3), −16% (RSTD = 2.0), −27% (RSTD = 1.0), and −30% (RSTD = 1.0) at these intervals, respectively (Fig 3). Overall maximal thoracic aortic diameter was observed to regress during the follow-up period: 5% (RSTD = 2.1), 4% (RSTD = 2.6), 2% (RSTD = 5.5), and 1% (RSTD = 19.6) at 1, 3, 6, and 12 months.

Across all patients, infrarenal maximal true lumen diameter increased in the postprocedure period. At 1, 3, 6, and 12 months, infrarenal true lumen maximal diameter expanded by 9%, 2.5%, 8.5%, and 3% from the preoperative measurements, respectively (Fig 4). Maximal infrarenal false lumen diameter regressed postintervention: 3%, 3%, 4%, and −3% at 1, 3, 6, and 12 months (Fig 5).

Infrarenal volumetric and diameter changes varied between those patients who had intervention for type B dissection with infrarenal involvement vs those who did not. Of 46 patient with infrarenal extension of the type B dissection, 23 (50%) had increasing infrarenal aortic volume following TEVAR. By comparison, four out of the 14 patients (27%) without infrarenal dissection had infrarenal aortic volume expansion during patient follow-up. In patients with infrarenal type B dissection, infrarenal maximal diameter was seen to increase by an average of 4%, 9%, 6%, and 17% at 1, 3, 6, and 12 months. Conversely, in patients without infrarenal dissection, the infrarenal maximal diameter was 1%, −2%, 4%, and −1% at these intervals, respectively (Fig 6; Table III).

Volumetric analysis also indicates infrarenal expansion following TEVAR in patients with type B dissection with infrarenal involvement. In this group, total infrarenal volume was noted to increase by 8%, 11%, 18%, and 22% at 1-, 3-, 6-, and 12-months postprocedure. By comparison, in patients without preoperative infrarenal dissection, volumes were −6%, 5%, 1%, and 3% at these follow-ups, respectively (Fig 7; Table IV).

DISCUSSION

Patient outcomes in our analysis appear to be comparable to the current literature. Recent series suggest a perioperative mortality rate of 5% to 11% in the setting of TEVAR for complicated type B aortic dissection.[7] Although our 30-day procedure-related mortality rate of 14% may appear high in relation to current studies, one patient death occurred due to aortic rupture following TEVAR in a patient with Marfan's syndrome. The other rupture patient was a 78-year-old male who had intervention for aortic enlargement. In addition, one patient with cause of death categorized as retrograde dissection was found to have an ascending dissection on autopsy

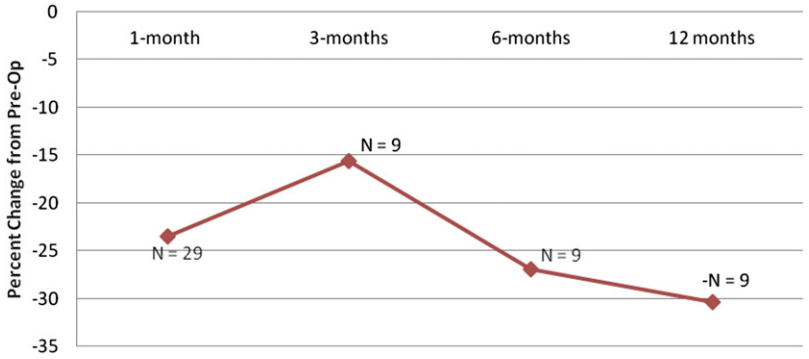

Fig 3. Percent change in thoracic false lumen maximal diameter over time.

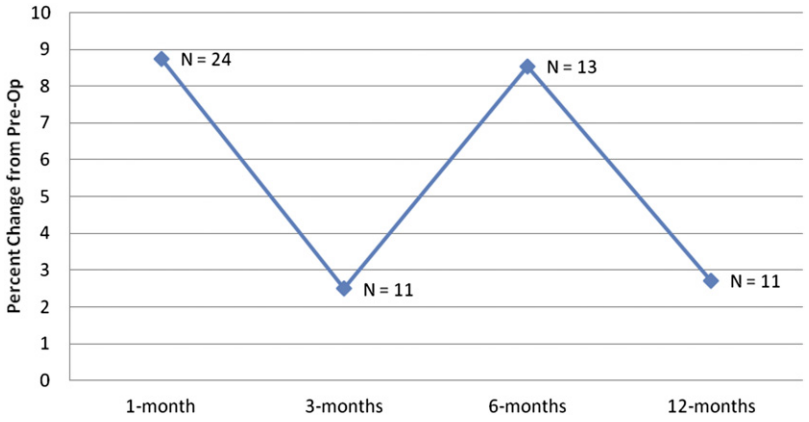

Fig 4. Percent change in infrarenal true lumen maximal diameter over time.

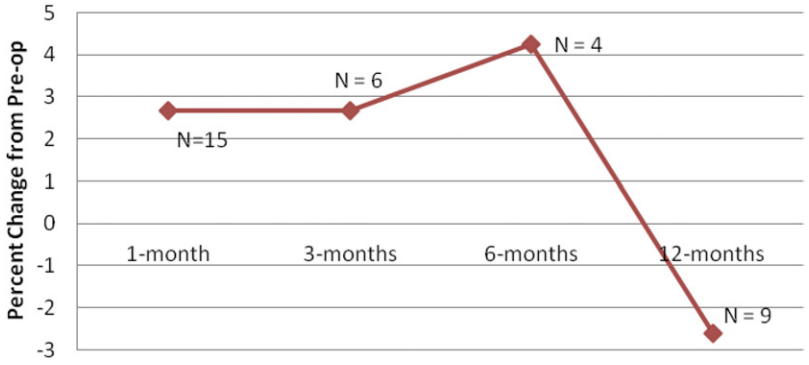

Fig 5. Percent change in infrarenal false lumen maximal diameter over time.

that originated from the anastomosis of a previous aortic graft, done as part of a debranching procedure, and thus not endograft-related. Therefore, three out of the 72 treated patients, or 4%, had a device-related retrograde dissection postoperatively. This is comparable with the retrograde dissection rate seen in prior series.[8] Of the remaining three deaths due to retrograde dissection, two were individuals who had TALENT grafts placed. Only one out of the 39 patients who had Valiant-Captivia tip-capture devices died secondary to retrograde dissection. These findings may suggest that the newer tip-capture devices may reduce the risk of retrograde dissection-related complications.

The average preoperative patient ASA score of 3.2 suggests that our study population had severe underlying systemic illness at baseline. The stroke and paraplegia rate of 1% noted in our study is also comparable to current literature. It is worth noting that the patient who

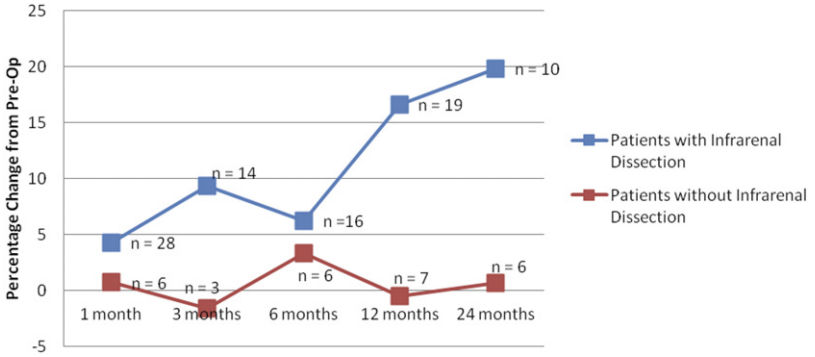

Fig 6. Percent change in maximal infrarenal diameter: Patients with preoperative evidence of infrarenal dissection vs patients without preoperative evidence of infrarenal dissection.

Table III. Comparison of average percent change in infrarenal aortic diameter

Follow-up	*Infrarenal dissection group*	*No infrarenal dissection group*	P *value*
1 month	4.25%	0.72%	.46
3 months	9.34%	−1.64%	.43
6 months	5.93%	4.10%	.72
12 months	16.61%	−0.57%	.24
24 months	9.64%	0.64%	.18

had paraplegia was an individual who had prior open repair of ascending and descending dissection, but had subsequently developed descending dissection distal to the previous graft anastamosis.

The observed reintervention rate of 15% is acceptable and similar to other published data. Other investigations have reported reintervention rates as high as 22% following TEVAR for complicated type B aortic dissection.[8,9] There were no procedure-related deaths following reintervention in the study cohort.

As a referral institution, there were limitations to certain aspects of patient data. The exact duration of chronic aortic dissection was known in less than half of all patients. As a result, we were unable to reliably quantify the average age of chronic dissection in our patient population. Therefore, we were unable to perform analysis on duration of disease and aortic remodeling following TEVAR.

Our analysis suggests that aortic remodeling following TEVAR for complicated chronic type B dissection occurs in a predictable fashion. Diametric and volumetric true lumen expansion and false lumen regression is observed after intervention in this group of patients. The INSTEAD trial, which looked at patients with uncomplicated type B dissection, had similar findings in their study cohort. Other series have similarly shown favorable aortic remodeling following thoracic intervention for type B dissection.[10] While the INSTEAD trial looked at maximal thoracic false and true lumen diameter changes, their analysis did not include volumetric changes. Moreover, INSTEAD and prior studies did not look at changes in the infrarenal aorta following endoluminal intervention. While the thoracic aorta appears to remodel in a predictable manner following TEVAR, remodeling of the infrarenal aorta does not. Our volumetric and diameter measurements suggest that postprocedure infrarenal remodeling may be influenced by the extent of type B dissection.

In patients with thoracic dissection extending below the renal arteries, there is infrarenal aortic expansion following TEVAR. Both the infrarenal aortic diameter and volume increased following intervention in this patient subgroup. Comparatively, individuals without infrarenal dissection involvement had relatively no changes in infrarenal diameter and volume following thoracic endovascular intervention. Proposed mechanisms for persistent distal perfusion include (1) distal re-entry tears, (2) retrograde filling of the false lumen, and (3) kinetic motion of the dissection flap.[4] The first of these mechanisms suggests that re-entry tears in the intimal flap may be present that are not visualized on CT imaging. As a result, these re-entry segments may provide perfusion of the false lumen. The second mechanism proposes retrograde perfusion of the false lumen through visceral branch vessels. The role of intimal flap motion has been analyzed in recent series.[11] It is conceivable that unimpeded septal motion may prevent stagnation of flow in the aortic false lumen, thereby preventing thrombosis.

The finding of persistent infrarenal aortic expansion post-TEVAR is not a new observation. Prior literature has identified that as many as 50% to 70% of all patients experience abdominal aortic expansion following endoluminal exclusion for thoracic aortic dissection.[12] However, previous studies did not describe the patient subgroup in which this remodeling occurs. Our study suggests that the infrarenal aortic enlargement occurs to a greater degree in those patients who have preintervention evidence of type B dissection extending below the renal arteries.

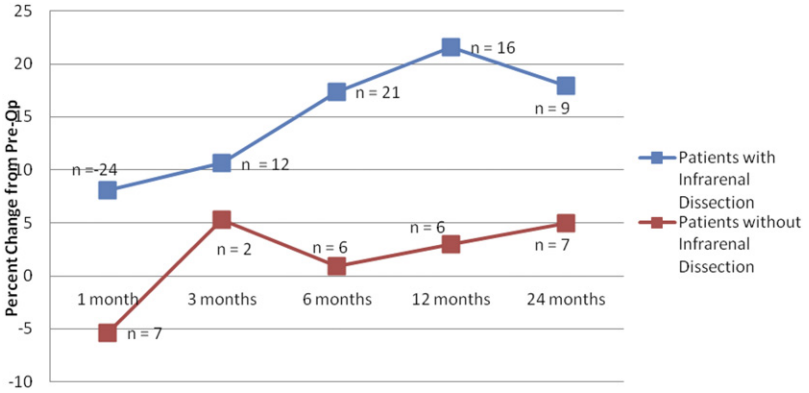

Fig 7. Percentage change in total infrarenal volume: Patients with preoperative evidence of infrarenal dissection vs patients without preoperative evidence of infrarenal dissection.

Table IV. Comparison of average percent change in infrarenal aortic volume

Follow-up	*Infrarenal dissection group*	*No infrarenal dissection group*	P *value*
1 month	8.04%	−6.30%	.06
3 months	10.59%	5.31%	.70
6 months	17.34%	0.90%	.17
12 months	21.61%	5.02%	.20
24 months	17.90%	12.14%	.81

A potential remedy for persistent infrarenal aortic perfusion following TEVAR has been proposed in the literature. The PETTICOAT technique involves the placement of bare metal scaffolding stents as an adjunct to or staged procedure following TEVAR. Nienaber and colleagues performed this technique in 12 patients previously treated by TEVAR for complications of chronic dissection. Each patient had progressive infrarenal aortic enlargement following thoracic intervention. Following deployment of the bare metal scaffolding stents, all patients demonstrated improved infrarenal aortic remodeling after 1 year.[13]

CONCLUSIONS

TEVAR is an appropriate treatment option for individuals with complications of chronic aortic dissection. However, infrarenal involvement of type B dissection appears to be a risk factor for progressive infrarenal aortic enlargement following thoracic intervention. Further analysis, longer follow-up, and a larger study cohort are necessary to adequately power statistical analyses. At the minimum, the findings of this analysis should prompt clinicians to include the abdominal aorta on follow-up CT imaging following TEVAR. Analysis of current tip-capture devices should include rates of retrograde dissection and associated mortality; the newer tip-capture devices may decrease the risk of this dreaded complication in relation to older devices. Moreover, continued investigation of newer endovascular devices and the use of large open stents should be pursued to address the issue of infrarenal aortic expansion following thoracic intervention.

AUTHOR CONTRIBUTIONS

Conception and design: IA, CD, IW, GK, RW
Analysis and interpretation: IA, CD, FO, GK, RW
Data collection: IA, FO, GK
Writing the article: IA, CD, FO, RW
Critical revision of the article: CD, IW, RW
Final approval of the article: CD, IW, RW
Statistical analysis: IA
Obtained funding: Not applicable
Overall responsibility: IA

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DISCUSSION

Dr Ben Starnes (*Seattle, Wash*). I have the following questions for the authors:

The goal of this therapy is to prevent death due to aneurysm rupture and NOT to treat the underlying dissection. Did any patient in this series continue to expand over the follow-up period, and more importantly, did any patient go on to rupture and die?

Dr Iden David Andacheh. Given the fact that our study population was part of an FDA-approved investigational device exemption (IDE), patient mortality has been closely followed. As previously mentioned, there were two cases of rupture within the 30-day postoperative period. Since the publication of these data, we have had two patients who required debranching procedures as a result infrarenal aortic expansion during long-term follow-up. No patient has died due to rupture during long-term follow-up.

Dr Starnes. Described in this series are four cases of retrograde aortic dissection leading to death. The Medtronic Talent device has an uncovered proximal stent to aid in fixation and the company recently added a tip-capture mechanism (Valiant-Captivia) to avoid a commonly observed deployment deficiency described as "retroflexion" of this uncovered stent. Do the authors believe that this device, with its uncovered proximal stent, is the best device for managing dissections, and furthermore, have any lethal retrograde dissections been observed since using the new graft?

Dr Andacheh. Our institution used the Medtronic Talent device from 2002 to 2007 and the Valiant-Captivia device from 2008 to 2010. Of the 34 patients treated with Talent grafts, two patients were complicated with a device-related retrograde dissection. By comparison, one out of the 39 patients treated with Valiant-Captivia grafts had a device-related retrograde dissection. This may suggest a benefit to using the Valiant-Captivia device, but clearly, further analysis is needed.

Dr Starnes. As we all know, most type B dissections begin at the left subclavian artery and thus an adequate proximal seal may require a debranching procedure such as a carotid-subclavian bypass to achieve success. How often was a debranching procedure done in this series? Along those lines, what was the rate of type I endoleak?

Dr Andacheh. A debranching procedure was required in four out of the 72 treated patients. Of the seven patients treated for endoleak, four were for a type I endoleak.

Dr Starnes. What was the role of neural protection in your series to prevent the dreaded complication of paraplegia? Was spinal drainage used, and if so, what were the indications to do so?

Dr Andacheh. Spinal drain was not routinely used. Intraoperative maintenance of MAP pressures was used to reduce the risk of paraplegia. Placement of a spinal drain was considered when subclavian artery coverage was required.

Dr Starnes. Finally, can the authors please expand on any techniques or strategies for obliterating false lumen flow with coils or glue or some sort of obliterative therapy to increase rates of favorable aortic remodeling?

Dr Andacheh. We have not used coils, glue, or other obliterative therapies. We rely on adequate proximal exclusion and remodeling of the aorta. We have only intervened by adding additional devices when the source of aortic expansion was identified.