Aortic remodeling following hybrid arch repair with zone 0 to 5 thoracic endovascular aortic repairs for complex arch and descending thoracic aortic pathologies

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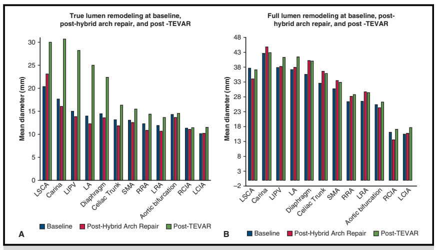
ABSTRACT

Objective: For high-risk patients with aortic arch pathology, hybrid aortic arch repair with simultaneous or staged thoracic endovascular repair of the descending aorta may be a viable alternative to open repair. However, data on postintervention aortic remodeling remain limited. We report the short-term outcomes of remodeling of the thoracoabdominal aorta after hybrid arch repair + thoracic endovascular repair.

Methods: All patients undergoing hybrid arch repair with planned zones 0 to 5 thoracic endovascular repair from January 2020 to March 2022 were retrospectively reviewed. Computed tomography angiography scans preoperatively, after hybrid aortic arch repair, and on long-term follow-up were analyzed for thoracoabdominal aorta remodeling. Mean change in aortic true luminal diameter and full luminal diameter was calculated at every level, and paired-samples *t* test was used to compare means.

Results: Of 39 patients, 38 had follow-up data at a mean duration of 14.9 months. There were a total of 3 (7.7%) deaths, 0 (0.0%) strokes, and 0 (0.0%) paralysis. For the 35 patients undergoing thoracic endovascular repair for aortic dissection, at follow-up, there was a significant increase in the mean true luminal diameter at each level ($P < .05$), except at the aortic bifurcation and common iliac arteries. The largest increase in mean true luminal diameter ($P < .01$) was observed at the level of the left inferior pulmonary vein (mean difference +13.22 mm, 95% CI, 10.38-16.07), tracheal carina (mean difference +13.06 mm, 95% CI, 10.05-16.07), and inferior left atrium (mean difference +11.19 mm, 95% CI, 7.84-14.53).

Conclusions: Hybrid arch repair with zones 0 to 5 leads to improved true lumen augmentation in zones 0 to 8 with complete false lumen thrombosis down to zone 5 at short-term follow-up. Zones 9 to 11, if involved, may require adjunctive treatment strategies for total aortic remodeling and complete false lumen obliteration. (JTCVS Open 2024;17:23-36)



Graphical representation of (A) true lumen and (B) full lumen remodeling.

CENTRAL MESSAGE

HAR with zone 0 to 5 TEVAR leads to improved true lumen augmentation with high rates of false lumen thrombosis down to zone 8 at short-term follow-up.

PERSPECTIVE

We evaluate the clinical outcomes and remodeling data of 39 patients undergoing complex HAR with zone 0 to 5 TEVAR. There were 3 deaths during the study period with no stroke or paralysis. At follow-up, there was a significant increase in the mean TLD at each aorta level, except at the aortic bifurcation and common iliac arteries.

The surgical management of complex aortic arch disease in comorbid and frail patients poses a unique clinical challenge. Traditionally, complex arch pathologies have been treated using open elephant trunk techniques to repair arch-limited disease or followed by staged open or endovascular repair of the descending thoracoabdominal aorta (DTA).[1] Despite increasing institutional experience, these interventions remain associated with high operative mortality, reinterventions, and neurological complications.[2] Given the invasiveness of staged open repair, they remain a relative contraindication in critically ill and frail patients limiting them to conservative management with an even higher mortality risk.[3] For this population, hybrid arch repair (HAR) with thoracic endovascular aortic repair (TEVAR) of the descending aorta may be a viable alternative to open repair.[4]

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Abbreviations and Acronyms

FLD	= full luminal diameter
HAR	= hybrid arch repair
LSCA	= left subclavian artery
MD	= mean difference
TEVAR	= thoracic endovascular aortic repair
TLD	= true luminal diameter

HAR refers to the surgical debranching of supra-aortic branches to create a proximal landing zone for simultaneous or staged TEVAR to exclude the diseased aortic segment (Figure E1). Although previous reports have shown satisfactory outcomes using HAR + TEVAR, data on aortic remodeling after HAR remain relatively scarce.[5,6]

Positive remodeling after intervention for aortic pathologies refers to expansion of the true aortic lumen coupled with shrinkage and full thrombosis of the false aortic lumen in cases with arch and DTA aneurysmal dissection and stabilization/regression. Prior reports have suggested that positive aortic remodeling with false lumen thrombosis is associated with improved survival after TEVAR in patients with type B aortic dissections.[7] Partial false lumen thrombosis has been shown to predispose patients to aneurysm formations and need for reintervention for aortic dissection in cases of aortic arch/DTA aneurysms.[8] However, data on postintervention aortic remodeling remain limited. We report the short-term outcomes of remodeling of the aortic arch and thoracoabdominal aorta after HAR + TEVAR.

MATERIAL AND METHODS

Study Population

The study was approved by the hospital Institutional Review Board in July 2020 (#2000020356). All patients 18 years and older undergoing HAR with planned zones 0 to 5 TEVAR from January 2020 to March 2022 were included and retrospectively reviewed. Patient demographics, operative outcomes, and aortic arch and thoracoabdominal aorta remodeling data were extracted from patient electronic health records. We recorded the following data: patient characteristics (age, gender, body mass index, smoking status, presenting etiology [aortic dissection/aneurysm]), baseline comorbidities (hypertension, dyslipidemia, diabetes, chronic obstructive pulmonary disease, previous cardiac surgery, dialysis, peripheral vascular disease), postoperative outcomes (mortality, stroke, paralysis, acute kidney injury, myocardial infarction, arrythmia), and remodeling of the thoracoabdominal aorta.

Operative Technique

All HAR operations were performed in a hybrid operating room with transesophageal echocardiography guidance and fluoroscopy during endograft deployment. Our operative approach to HAR with simultaneous or staged TEVAR has been reported.[5,9] Briefly, type I HAR is performed in patients with isolated arch aneurysm or dissection with good native landing zone 0 and 3/4. Typically, more than 2 cm of the native aorta or more than 4 cm of Dacron graft in zone 0 is a favorable landing zone for type I HAR. It involves reimplantation of the great aortic vessels using a 4-branched Dacron graft sutured to the ascending aorta superior to the sinotubular junction. This is followed by deployment of stent grafts antegrade via a limb of the 4-branch graft into the aortic arch or via a retrograde iliofemoral route.

Type II HAR involves hemiarch replacement with Dacron graft or distal anastomosis in zones 1/2 and is performed in patients with proximal extension of disease into the ascending aorta precluding a potential zone 0 stent graft landing. This approach also is used in patients with an ascending aortic diameter more than 4 cm due to the risk of retrograde type A aortic dissection. Notably, type II HAR requires cardiopulmonary bypass with or without circulatory arrest during proximal aortic reconstruction. After great vessel debranching zone 0 reconstruction, an endograft can be deployed through the arch with a new Dacron zone 0 landing zone or preferably in a staged retrograde approach.

Type III HAR builds on type II HAR by deploying a frozen elephant trunk in the descending thoracic aorta to create a longer proximal landing zone for staged TEVAR. This approach is often reserved for extensive aortopathies or aneurysmal dilation more than 6 cm due to its increased invasiveness and high risk for operative mortality and paraplegia. Adjunct spinal protection strategies are used for these patients to minimize risk of paraplegia.

For the TEVAR component of the procedure, depending on the anatomy and extent of distal aortic disease, grafts are deployed in a retrograde fashion from the femoral vessels or antegrade through one of the side arms of the aortic Dacron graft. As much as possible, we aim to perform staged TEVAR to minimize the time on table during the index procedure as well as lower risk of spinal injury. Simultaneous TEVAR is typically performed in patients with acute aortic dissection with signs of malperfusion and organ injury requiring immediate restoration of distal perfusion. In our practice, we use spinal drains in all patients undergoing HAR plus simultaneous or staged TEVAR because of its benefits in reducing spinal cord injury. Adjunct spinal protection strategies such as maintaining high mean arterial pressure and cerebrospinal fluid pressure monitoring are used in the postoperative management of these patients.

Assessment of Aortic Arch and Thoracoabdominal Aorta Remodeling

Computed tomography angiography scans performed preoperatively, after hybrid aortic arch repair, and at 6-month to 1-year follow-up were analyzed for thoracoabdominal aorta remodeling for each of the patients with aortic dissection. Aortic true lumen diameter (TLD) and full luminal diameter (FLD) were recorded at 12 anatomic levels including the left subclavian artery (LSCA), tracheal carina, left inferior pulmonary vein, inferior left atrium, diaphragm, celiac trunk, superior mesenteric artery, renal arteries, aortic bifurcation, and common iliac arteries. Additionally, visceral vessel origin from the true/false lumen and the extent of false lumen thrombosis were assessed.

Statistical Analysis

Categorical variables were reported as counts with percentages, and continuous variables were logged as medians with their interquartile range or means with their SDs after assessment for normality using the Shapiro–Wilk test. Categorical variables were assessed using Mann–Whitney *U* test. Mean change in aortic TLD was calculated at every level, and paired-samples *t* test was used to compare means. All analyses were performed using R (version 3.5.1 R Project for Statistical Computing) within R Studio.

RESULTS

Patient Characteristics

A total of 39 patients were included in our clinical analysis, of whom 38 (97.4%) had follow-up data at a mean duration of 14.9 months (566.2 patient months). The mean age of patients undergoing HAR + TEVAR was 58.1 ± 10.1 years, and 31 patients (79.5%) were male.

Thirty-five patients (89.9%) had hypertension, 19 patients (48.8%) had dyslipidemia, and 11 patients (28.2%) had diabetes. Twenty-two patients (56.4%) had a history of smoking. The mean Society of Thoracic Surgeon risk score for this cohort was 23.8% (SD, 24.9). Four patients (10.5%) presented with aneurysms (zone 0-3), 28 patients (71.8%) presented with type A aortic dissection (zone 0 to 3), 2 patients (5.1%) had type B aortic dissection with retrograde arch extension (zone 1 to 10), and the remaining patients had mixed etiology. Within the type A aortic dissections, 14 patients (50.0%) had acute presentation. Of these patients, all, except 1, with acute type A dissection underwent repair under circulatory arrest with 1 repaired with crossclamp. The detailed baseline characteristics of included patients are presented in Table 1. Details of intraoperative variables are reported in Table 2 and Figure E2.

TABLE 1. Patient baseline demographics and characteristics of included patients

Variable	No. of patients (%), N = 39
Age, mean (SD), y	58.1 (10.1)
Male (%)	31 (79.5)
Body mass index, mean (SD)	30.0 (5.9)
Diabetes mellitus	11 (28.2)
Hypertension	35 (89.9)
Dyslipidemia	19 (48.8)
Coronary artery disease	5 (12.8)
Dialysis	1 (2.6)
Chronic obstructive pulmonary disease	7 (20.6)
Smoking history	22 (56.4)
Prior coronary artery bypass grafting surgery	3 (7.7)
Prior aortic surgery	19 (48.8)
Society of Thoracic Surgery Risk Score, Mean (SD)	23.8 (24.9)
Presenting etiology	
Type A aortic dissection	28 (71.8)
Acute	14 (50.0)
Subacute	0 (0.0)
Chronic	14 (50.0)
Type B aortic dissection	2 (5.1)
Acute	0 (0.0)
Subacute	1 (50.0)
Chronic	1 (50.0)
Aneurysm	4 (10.3)
Acute	2 (50.0)
Subacute	0 (0.0)
Chronic	2 (50.0)
Type A/type B aortic dissection*	4 (10.3)
Acute	3 (75.0)
Subacute	0 (0.0)
Chronic	1 (25.0)
Type B aortic dissection/ aneurysm	1 (2.6)
Acute	1 (100.0)
Subacute	0 (0.0)
Chronic	0 (0.0)

SD, Standard deviation. *Two acute type A on type B dissections, 1 acute type B dissection in patient with prior type A repair, and 1 chronic type B aortic dissection with propagation of known type A dissection.

TABLE 2. Summary of intraoperative management, cerebral protection strategies, cannulation strategies, and graft parameters

Variable	No. of patients (%), N = 39
Hybrid arch approach	
Type 1	1 (2.6)
Type 2	31 (79.5)
Type 3	7 (17.9)
Cardiopulmonary bypass time (min), mean (SD)	287.7 (84.2)
Aortic crossclamp time (min), mean (SD)	148.6 (84.5)
TEVAR intervention	
Simultaneous	10 (25.6)
Staged	29 (74.4)
Days to TEVAR, Median, (IQR)	38.5 (9.25-120)
Concomitant intervention	
Coronary artery bypass grafting	3 (7.7)
Aortic valve surgery	22 (56.4)
Aortic root surgery	22 (56.4)
Cannulation strategy	
Axillary	24 (61.5)
Central	10 (25.6)
Femoral	5 (12.8)
Cerebral protection strategy	
Anterograde	23 (59.0)
Retrograde	8 (20.5)
Anterograde + retrograde	8 (20.5)
Lupiae graft diameter, mean (SD)	26.3 (4.1)
Proximal TEVAR graft diameter [mm], mean (SD)	35.3 (3.2)
Proximal TEVAR graft length [mm], mean (SD)	175.7 (36.8)
Distal TEVAR graft diameter [mm], mean (SD)	34.8 (3.8)
Distal TEVAR graft length [mm], mean (SD)	164.0 (26.9)

SD, Standard deviation; *TEVAR*, thoracic endovascular aortic repair; *IQR*, interquartile range.

Postoperative Outcomes

In patients with aortic aneurysm, there was 1 postoperative death (2.6%) due to cardiac arrest before staged TEVAR (Table 3). This patient presented with contained aneurysmal rupture and died on postoperative day 6 due to free rupture before staged TEVAR. The remaining 3 patients with aneurysm achieved complete coverage. There was 1 incidence (2.6%) of type 1b endoleak. There were 2 (5.1%) late deaths at 10.3 months and 20.0 months, respectively, after HAR in patients with aortic dissection. One patient died of abdominal aortic aneurysm rupture, whereas the cause for the other is unknown. Acute-onset renal failure requiring hemodialysis was noted in 4 patients (10.5%) with permanent hemodialysis in 1 patient due to progression of baseline stage 3 chronic kidney disease to stage 4. There was 0 (0.0%) incidence of postoperative paralysis, myocardial infarction, or type 1a, II, III, or IV endoleak. Nine patients (23.1%) underwent further reinterventions after TEVAR (Table E1). The median duration from index TEVAR to reoperation was 116 days (interquartile range, 59-307). There was no mortality due to reinterventions (0.0%).

TABLE 3. Summary of postoperative clinical outcomes

30-day/in-hospital outcomes	No. of patients (%), N = 39
Mortality	1 (2.6)
Stroke	0 (0.0)
Paralysis	0 (0.0)
Renal failure, requiring hemodialysis	4 (10.5)
Reoperation	1 (2.6)
Type Ia endoleak	0 (0.0)
Type Ib endoleak	1 (2.6)
Type II endoleak	0 (0.0)
Type III endoleak	0 (0.0)
Type IV endoleak	0 (0.0)
Long-term outcomes (mean follow-up 14.9 ± 9.1 mo, total follow-up 566.2 mo)	**No. of patients (%), N = 38**
Mortality	3 (7.9)
Stroke	0 (0.0)
Paralysis	0 (0.0)
Renal failure, requiring hemodialysis	4 (10.5)
Reoperation	9 (23.7)
Days to reoperation, Median (IQR)	116 (59-307)
Type Ia endoleak	0 (0.0)
Type Ib endoleak	1 (2.6)
Type II endoleak	0 (0.0)
Type III endoleak	0 (0.0)
Type IV endoleak	0 (0.0)

IQR, Interquartile range.

Computed Tomography Angiography Assessment of Thoracoabdominal Aorta Remodeling

Imaging and clinical surveillance were completed in 35 patients (100.0%) with aortic dissection. Total follow-up duration for patients with dissection was 521.5 patient months. At a mean follow-up period of 14.9 months, there was a significant increase in the mean TLD ($P < .05$) at all anatomic levels, except at the aortic bifurcation and common iliac arteries. Thoracoabdominal aorta remodeling data for TLD are summarized in Table 4, Figures 1-3, and Table E2. We observed complete zone 0 and aortic arch coverage without endoleak in all patients, as well as a more than 40% mean increase in the TLD at the level of the LSCA (mean difference [MD] +9.71 mm, 95% CI, 6.93-12.49, $P < .01$). The largest increase in mean TLD ($P < .01$) was observed at the level of the left inferior pulmonary vein (MD +13.22 mm, 95% CI, 10.38-16.07), tracheal carina (MD +13.06 mm, 95% CI, 10.05-16.07), and inferior left atrium (MD +11.19 mm, 95% CI, 7.84-14.53). Complete false lumen thrombosis of the DTA down to zone 5 was achieved in 100% of patients with aortic dissection.

Conversely, there was a small but statistically significant increase in the mean FLD ($P < .05$) at all anatomic levels, except at the LSCA, carina, aortic bifurcation, and right common iliac artery. Thoracoabdominal aorta remodeling data for FLD are summarized in Table E2 and Figure 3, *B*. The largest increase in mean FLD ($P < .01$) was observed at the level of the diaphragm (MD +4.54 mm, 95% CI, 2.07-7.01), the inferior left atrium (MD +4.33 mm, 95% CI, 2.13-6.52), and the left inferior pulmonary vein (MD +3.50 mm, 95% CI, 1.12-5.88).

The left renal artery was the most common vessel originating from the false lumen (10 [28.6%] pre-TEVAR, 14 [40.0%] post-TEVAR), followed by the right renal artery (9 [25.7%] pre-TEVAR and 6 [17.1%] post-TEVAR). Visceral perfusion remained intact after TEVAR in all patients. Complete false lumen thrombosis post-TEVAR was achieved at the celiac trunk, superior mesenteric artery, right renal artery, and left renal artery in 7 patients (20.0%), 4 patients (11.4%), 3 patients (8.6%), and 2 patients (5.7%), respectively.

DISCUSSION

For patients with contraindications to conventional open repair, HAR with staged TEVAR is a viable alternative for repair of complex arch and descending thoracic aortic pathologies. Whereas conventional surgical repair entails performing 2 major open operations (total arch repair with the elephant trunk followed by open

TABLE 4. Summary of true lumen remodeling of the aorta at various anatomic levels for patients with aortic dissection

	Preoperative true lumen measurements (mm)			Post-TEVAR true lumen measurements (mm)						Remodeling	
Level	Minimum	Maximum	Mean (SD)	Minimum	Maximum	Mean (SD)	Vessel origin from false lumen	False lumen partial thrombosis	False lumen full thrombosis	Mean change (95% CI) [% change]	*P* value
LSCA	11.2	34.6	20.32 (6.90)	21.2	37.6	30.03 (3.82)	-	-	-	9.71 (6.93-12.49) [+47.8%]*	<.01*
Carina	3.4	35	17.60 (8.19)	21.6	45.3	30.66 (5.75)	-	-	-	13.06 (10.05-16.07) [+74.2%]*	<.01*
LIPV	6.5	28.5	14.95 (5.54)	14.7	41.5	28.17 (6.16)	-	-	-	13.22 (10.38-16.07) [+88.4%]*	<.01*
Left atrium	4.9	32.3	13.91 (5.86)	9.8	40.9	25.1 (7.58)	-	-	-	11.19 (7.84-14.53) [+80.4%]*	<.01*
Diaphragm	6.6	28.5	14.46 (6.00)	9	37.8	22.31 (7.76)	-	-	-	7.85 (5.28-10.41) [+54.3%]*	<.01*
Celiac trunk	2.8	28.1	13.13 (5.98)	6.0	29.6	16.26 (5.5)	3 (8.5)	6 (17.1)	7 (20.0)	3.13 (1.59-4.68) [+23.8%]*	<.01*
SMA	3.4	27.1	13.06 (5.62)	6.3	30.1	15.47 (5.09)	2 (5.7)	4 (11.4)	4 (11.4)	2.41 (1.36-3.46) [+18.5%]*	<.01*
RRA	3.1	26.3	12.31 (5.77)	6.7	28.6	14.35 (5.58)	6 (17.1)	1 (2.9)	3 (8.6)	2.04 (0.93-3.15) [+16.6%]*	<.01*
LRA	3.1	26.3	11.88 (5.69)	6.8	28.6	13.57 (5.11)	14 (40.0)	2 (5.7)	2 (5.7)	1.69 (0.73-2.65) [+14.3%]*	<.01*
Aortic bifurcation	7.3	28.1	14.23 (6.07)	7.7	26.1	14.50 (4.86)	-	-	-	0.27 (−1.47 to 2.01) [+1.9%]	.75
RCIA	3.5	17.5	11.26 (3.65)	4.3	17.9	11.45 (3.94)	-	-	-	0.19 (−1.40 to 1.77) [+1.7%]	.81
LCIA	3.3	15.6	10.17 (3.81)	3.1	20.6	11.49 (4.35)	-	-	-	1.32 (−0.21 to 2.85) [+13.0%]	.09

TEVAR, Thoracic endovascular aortic repair; *SD*, standard deviation; *CI*, confidence interval; *LSCA*, left subclavian artery; *LIPV*, left inferior pulmonary vein; *SMA*, superior mesenteric artery; *RRA*, right renal artery; *LRA*, left renal artery; *RCIA*, right common iliac artery; *LCIA*, left common iliac artery. *Statistically significant.

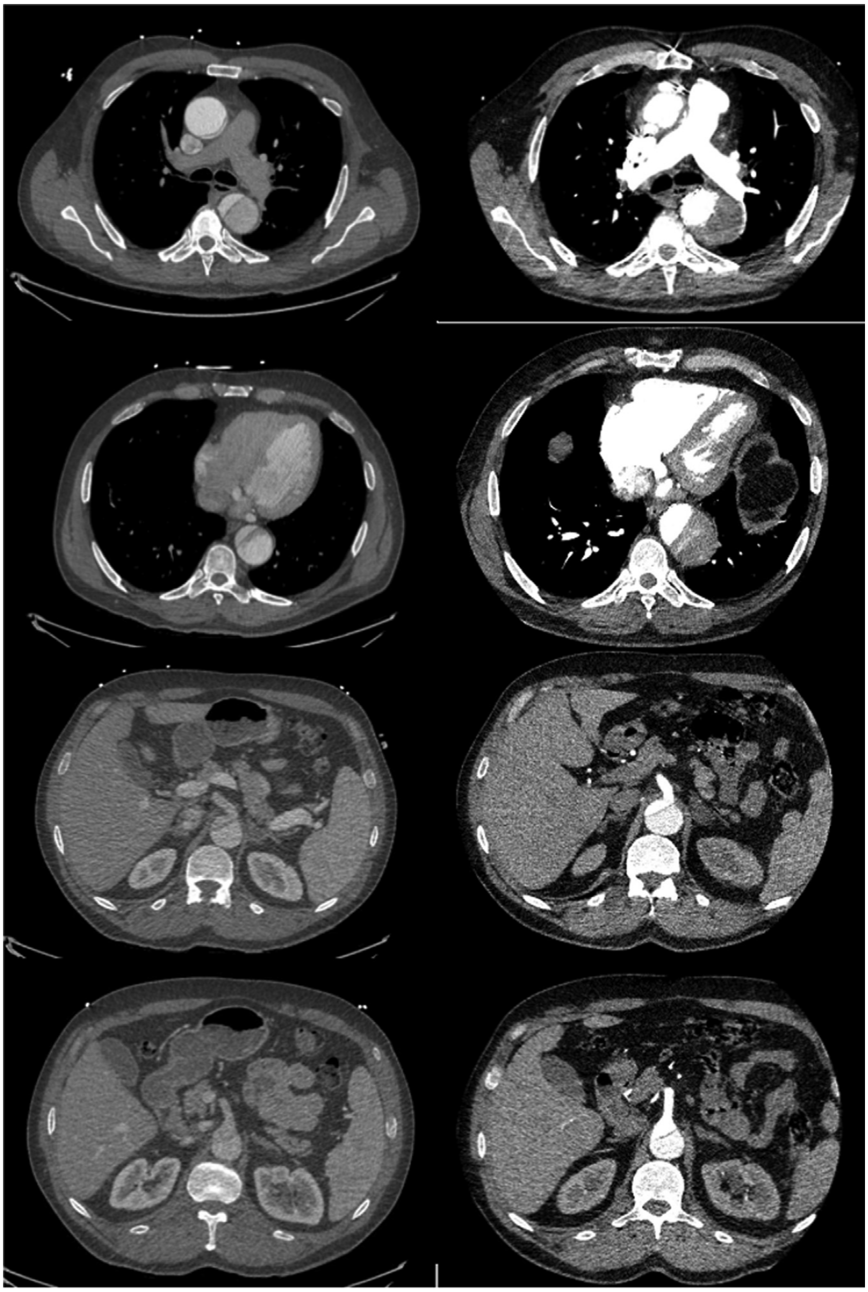

FIGURE 1. Computed tomography imaging showing preoperative and postoperative HAR with TEVAR.

thoracoabdominal repair in stage 2), HAR with staged TEVAR minimizes open surgical exposure and simplifies total arch replacement.[10,11] To our knowledge, our study is the first to comprehensively evaluate aortic remodeling of the aortic arch and thoracoabdominal aorta at different levels in patients undergoing HAR + TEVAR at an experienced center. At a mean follow-up of 14.9 months, we found HAR with zones 0 to 5 TEVAR to be associated with improved true lumen augmentation down to zone 8 and complete false lumen thrombosis down to zone 5 at 1-year follow-up.

Few centers have reported their experience with HAR + TEVAR in patients with aortic arch/descending thoracoabdominal aorta pathologies. Their data have been limited to clinical outcomes without information on aortic remodeling. In a previous report by our group in 2013, we reported a 100% successful great vessel debranching/stent graft deployment rate in a cohort of 28 patients undergoing type I HAR, 8 patients undergoing type II HAR, and 11 patients undergoing type III HAR.[5] The paraplegia rate was 2 (5.5%), with a stroke rate of 0 (0.0%). In-hospital/30-day mortality was 3 (8.0%). There were no postoperative

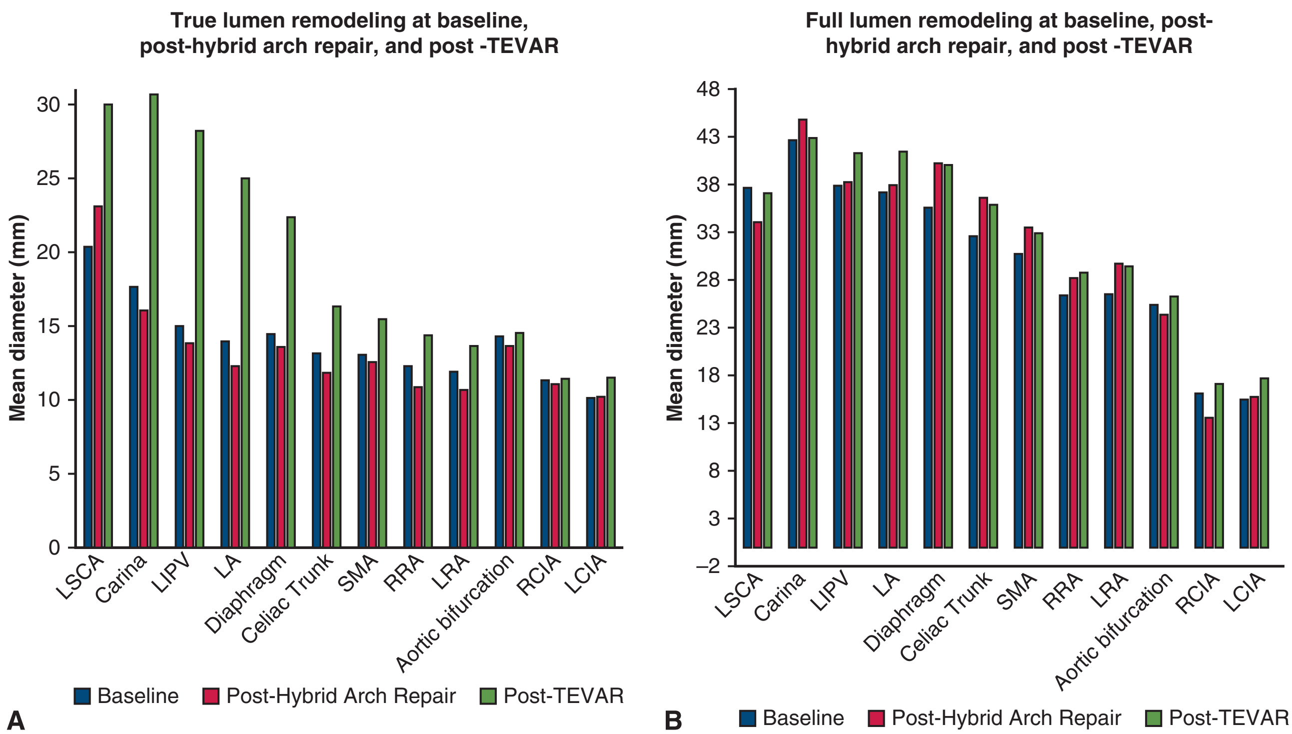

FIGURE 2. Graphical representation of (A) true lumen and (B) full lumen remodeling for each zone at baseline, after HAR, and post-TEVAR at long-term follow-up for patients with aortic dissection. *TEVAR*, Thoracic endovascular aortic repair; *LSCA*, left subclavian artery; *LIPV*, left inferior pulmonary vein; *LA*, left atrium; *SMA*, superior mesenteric artery; *RRA*, right renal artery; *LRA*, left renal artery; *RCIA*, right common iliac artery; *LCIA*, left common iliac artery.

endoleaks. A 2013 meta-analysis assessing the efficacy of hybrid techniques in patients with aortic arch pathologies including 26 studies with a total of 956 patients reporting aortic arch debranching procedures and 20 studies with 1316 patients undergoing the "frozen" or stented elephant trunk technique[12] reported a pooled 30-day/in-hospital mortality rate of 11.9% (95% CI, 9.4-14.9) for the arch debranching group and 9.5% (95% CI, 7.8-11.4) for the elephant trunk group. More recently, Joo and colleagues[13] assessed late adverse events after HAR in a cohort of 65 patients with aortic arch disease. At a median follow-up duration of 60.1 months, the authors reported overall survivals of 71.1% ± 7.4% and 57.2% ± 11.3% at 3 years and 6 years, respectively. Imaging surveillance was not reported in that study.

After invasive interventions, the thoracoabdominal aorta may undergo a series of intrinsic adaptations to reduce nonlaminar blood flow and stabilize the vessel walls. Such adaptions include an increase in TLD, reduction of false lumen diameter in patients with dissection, and complete thrombosis of the false lumen.[14] This may play an important role in promoting postinterventional survival, as well as reducing adverse complications, including endoleak.[14] In contrast to prior reports on HAR that have generally reported on patients' gross clinical outcomes,[15] we additionally evaluated aortic remodeling at distinct anatomic levels in high-risk patients after complex HAR + TEVAR receiving standardized postoperative medical regimen and computed tomography angiography follow-up. Of note, it is important to interpret aortic remodeling data in the context of clinical outcomes, especially mortality, stroke, and paraplegia. Currently, our series follow-up is not long enough to draw any correlations, given that we initiated the HAR + TEVAR strategy in 2020. However, midterm data assessing both clinical and imaging outcomes may help shed more light on the associated causes of improved survival associated with positive aortic remodeling.

As noted earlier, at a mean of 14.9 months follow-up, there was a significant increase in the mean TLD at each level ($P < .05$), except at the aortic bifurcation and common iliac arteries. The largest increases in mean TLD were observed at the levels of the left inferior pulmonary vein, carina, and left atrium, while also observing 100% false lumen thrombosis at these levels (zones 0-5). Although the exact pathophysiology underlying the lack of significant TLD expansion distal to aortic zone 8 is unclear, some prior studies have shown aortic dissections with infrarenal extension to be less amenable to remodeling.[16] Distal fenestrations below zone 5 and visceral vessel take-off from the false lumen likely contribute to this. In a 2014 systematic review of 16 articles describing

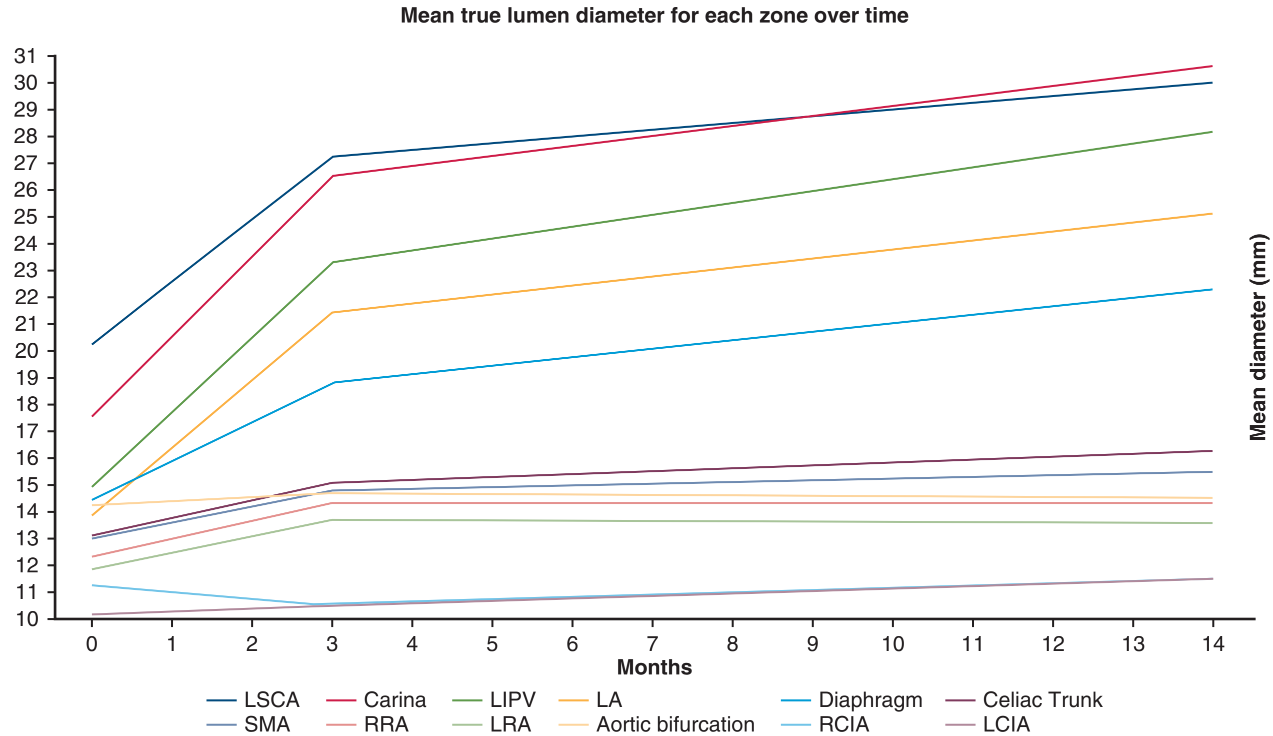

FIGURE 3. Progression of true lumen diameter for each zone over time for patients with aortic dissection. *LSCA*, Left subclavian artery; *LIPV*, left inferior pulmonary vein; *LA*, left atrium; *SMA*, superior mesenteric artery; *RRA*, right renal artery; *LRA*, left renal artery; *RCIA*, right common iliac artery; *LCIA*, left common iliac artery.

aortic remodeling after TEVAR,[17] Patterson and colleagues[17] reported false lumen thrombosis to occur more consistently in patients presenting with acute aortic dissection compared with patients with chronic dissection and positive remodeling to be less frequent distal to the diaphragm in both groups. Another possible cause for the absence of distal true lumen expansion in the thoracoabdominal aorta could be regional differences in aortic blood flow and wall shear stress. Prior studies have suggested that compared with the thoracic aorta, which possesses unidirectional laminar blood flow, the aortoiliac segment of the abdominal aorta possesses increased wall stress due to bidirectional blood flow and shear stress oscillation.[18] Moreover, the disruption of blood flow at the aortoiliac bifurcation may promote localized atherosclerosis/calcification, which has been shown to predict complications after endovascular interventions.[19] Consequently, zones 9 to 11, if involved, may require adjunctive treatment strategies for total aortic remodeling and complete false lumen obliteration. In line with this idea and findings, 9 patients (23.1%) in this study required reintervention after stage 2 TEVAR. Several entailed extension of the TEVAR or endovascular repair of iliac aneurysms for persistent abdominal aortic expansion or iliac artery enlargement.

Study Limitations

Our study was observational and limited to variables recorded in patient electronic health records/operative reports, which were not standardized. All HAR + TEVARs included in our analysis were performed at a single high-volume institution with experience in complex aortic repairs, and our findings may not be applicable to all facilities. Additionally, included patients all underwent comprehensive evaluation and were deemed to be inoperable or high risk for conventional repair. Further, patients had heterogeneous diagnoses/etiologies and underwent different types of HAR. The clinical and remodeling data seen in our study may not be reflective of all patient populations. The median time from index HAR to staged TEVAR was 38.5 days. In a few patients, this was delayed up to 120 days due to postoperative complications related to prolonged extracorporeal membrane oxygenation, organ malperfusion necessitating abdominal surgery, dialysis, and peripheral vascular interventions. This delay may have negatively impacted distal aortic remodeling in these patients, although we are unable to confirm this association because of our limited sample size. Finally, our follow-up duration is relatively short and premature to associate whether true lumen augmentation and false lumen thrombosis lead to improved clinical outcomes over follow-up.

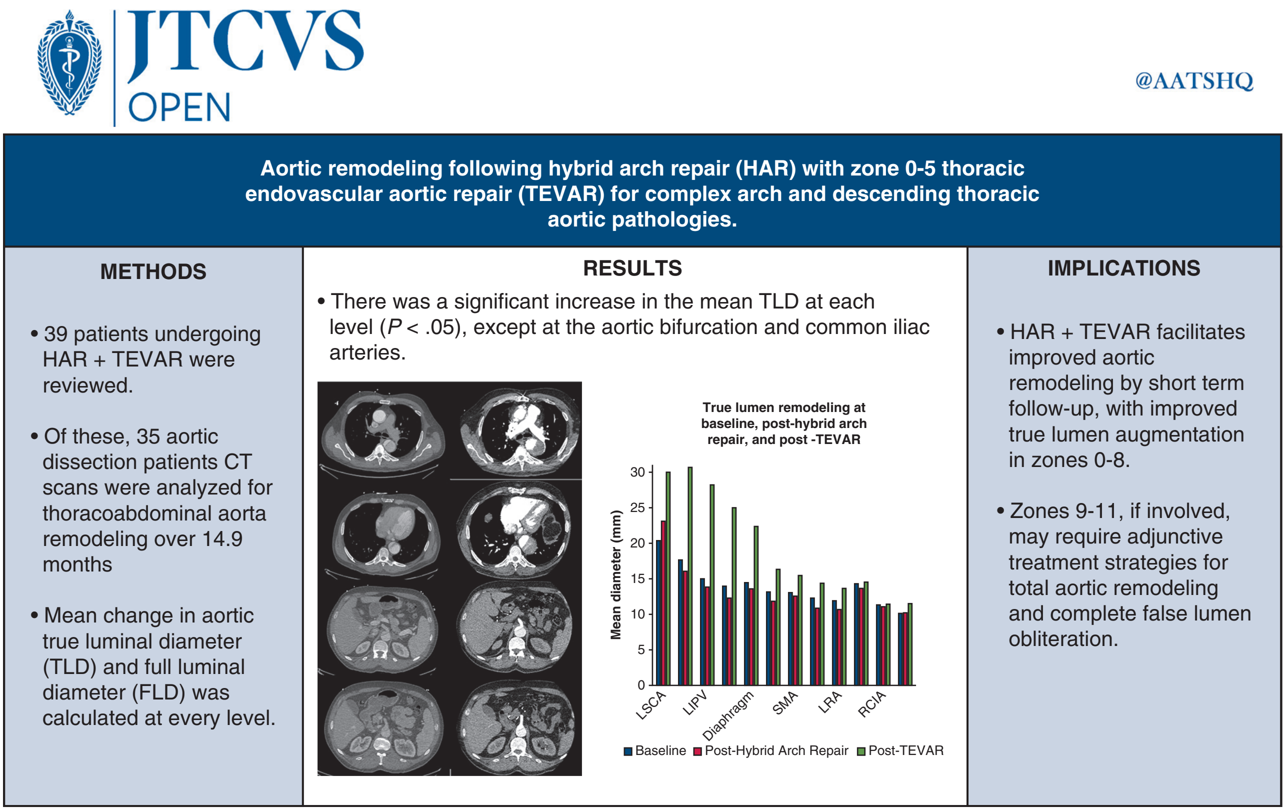

FIGURE 4. Graphical Abstract. Graphical summary of our analysis with representation of true and full lumen remodeling. *TLD*, True lumen diameter; *CT*, computed tomography; *LSCA*, left subclavian artery; *LIPV*, left inferior pulmonary vein; *SMA*, superior mesenteric artery; *LRA*, left renal artery; *RCIA*, right common iliac artery

CONCLUSIONS

HAR with zones 0 to 5 TEVAR provides excellent aneurysm exclusion and improved true lumen augmentation with a high rate of false lumen thrombosis down to zone 8 at 1-year follow-up (Figure 4). When involved, zones 9 to 11 may require adjunctive or alternative branched or fenestrated strategies for total aortic remodeling and complete false lumen obliteration in the abdominal aorta and iliac arteries.

Conflict of Interest Statement

Dr Nassiri is a consultant and proctor for Terumo Aortic and Medtronic Aortic. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: aneurysm, aortic arch, dissection, hybrid arch repair, thoracic endovascular repair

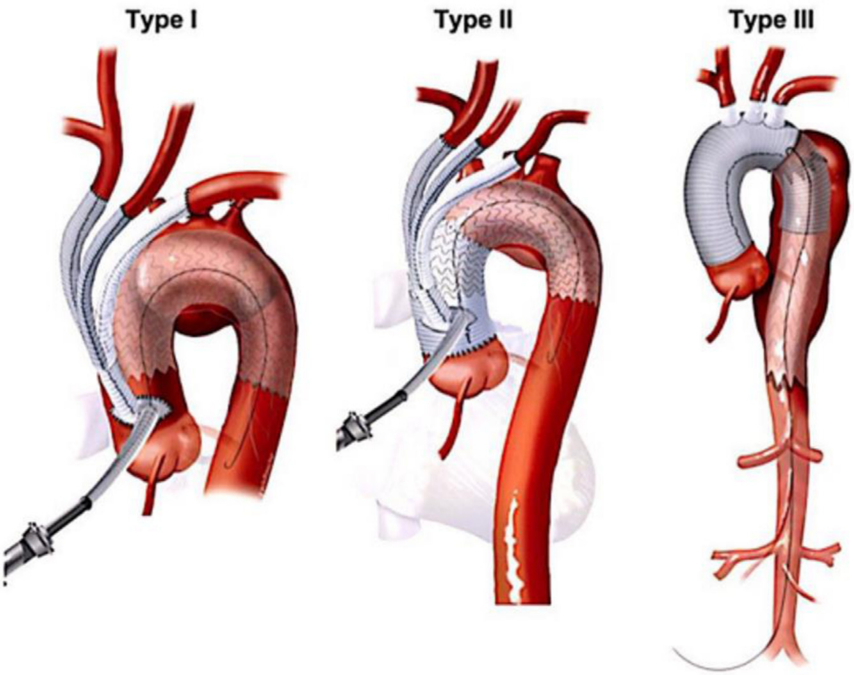

FIGURE E1. Schematic of major types of hybrid arch with zone 0 TEVAR. Adapted from Vallabhajosyula and colleagues.[4] https://doi.org/10.3978/j.issn.2225-319X.2013.05.09. PMID: 23977595; PMCID: PMC3741847.

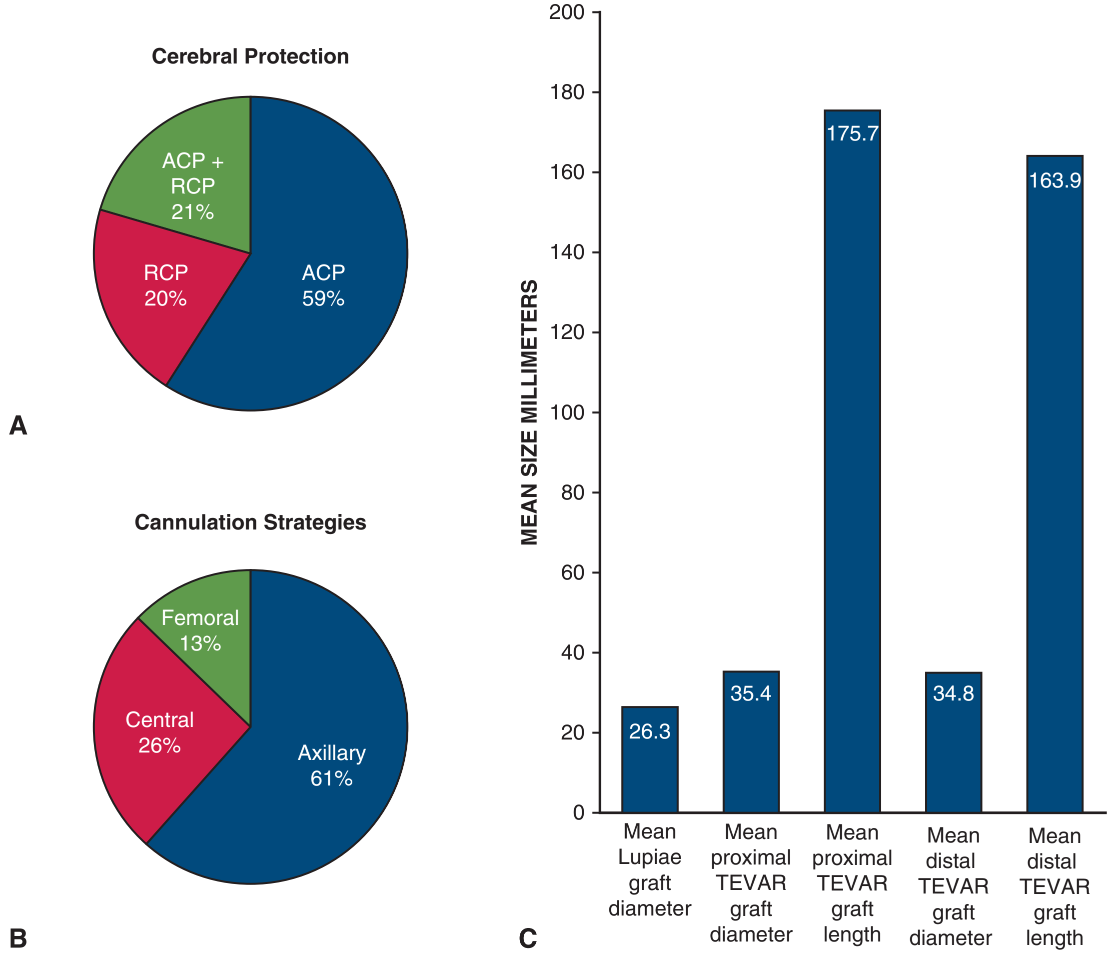

FIGURE E2. Summary of intraoperative cerebral protection strategies (A), cannulation strategies (B), and mean lengths/diameters of grafts used (C). *ACP*, Anterior cerebral protection; *RCP*, retrograde cerebral protection; *TEVAR*, thoracic endovascular aortic repair.

TABLE E1. Summary of reoperations

No.	Original etiology	Index operation	Days to reoperation	Reoperation indication
1	Acute type A	Type II HAR + simultaneous TEVAR	307	TEVAR for new type B aortic dissection
2	Chronic type A	Type III HAR + staged TEVAR	84	EVAR for new left common iliac aneurysm
3	Acute type A on B	Type II HAR + staged TEVAR	116	TEVAR for mycotic pseudoaneurysm at stent graft site
4	Chronic type A	Type II HAR + simultaneous TEVAR	32	TEVAR for rapidly growing aortic arch dissecting aneurysm
5	Acute type A	Type II HAR + staged TEVAR	148	TEVAR for stent graft–induced new entry tear/type B dissection
6	Chronic type A	Type III HAR + staged TEVAR	59	EVAR using a bifurcated unit body stent for dissecting thoracoabdominal aortic aneurysm and left common iliac artery dissecting aneurysm
7	Chronic type B	Type III HAR + staged TEVAR	7	Arterioplasty for infrarenal abdominal aortic intramural hematoma and left common iliac artery dissecting aneurysm
8	Acute type A	Type III HAR + staged TEVAR	379	Transverse arch replacement for postoperative hemolytic anemia secondary to sharp aortic graft kinking
9	Acute type A	Type III HAR + staged TEVAR	350	TEVAR for residual type B aortic dissection and right common iliac and common femoral pseudoaneurysm

HAR, Hybrid arch repair; *TEVAR*, thoracic endovascular aortic repair; *EVAR*, endovascular aortic repair.

TABLE E2. Summary of full lumen remodeling of the aorta at various anatomic levels for the 35 patients with aortic dissection

	Preoperative full lumen measurements (mm)			Post-TEVAR full lumen measurements (mm)						Remodeling	
Level	Minimum	Maximum	Mean (SD)	Minimum	Maximum	Mean (SD)	Vessel origin from false lumen	False lumen partial thrombosis	False lumen full thrombosis	Mean change (95% CI) [% change]	*P* value
LSCA	29.7	55.9	37.74 (6.35)	27.6	54.5	37.14 (6.65)	-	-	-	−0.60 (−3.36 to 2.15) [–0.2%]	.66
Carina	25.2	66.4	42.68 (9.44)	28.4	65.1	42.98 (10.41)	-	-	-	0.30 (−1.83 to 2.43) [+0.7%]	.77
LIPV	23.9	57.8	37.89 (9.16)	25.6	62.1	41.39 (9.44)	-	-	-	3.50 (1.12-5.88) [+9.2%]*	<.01*
Left atrium	26.7	57.3	37.23 (8.86)	26.3	60.2	41.56 (9.70)	-	-	-	4.33 (2.13-6.52) [+11.6%]*	<.01*
Diaphragm	25.5	74.5	35.62 (9.92)	22.7	99.7	40.16 (13.49)	-	-	-	4.54 (2.07-7.01) [+12.7%]*	<.01*
Celiac trunk	23.2	57.9	32.69 (6.86)	24.5	75.9	35.94 (9.32)	3 (8.5)	6 (17.1)	7 (20.0)	3.25 (1.74-4.75) [+9.9%]*	<.01*
SMA	21.8	56.5	30.78 (6.49)	20	70.2	32.91 (8.62)	2 (5.7)	4 (11.4)	4 (11.4)	2.13 (0.73-3.53) [+6.9%]*	<.01*
RRA	19.5	41.1	26.42 (4.60)	19.5	41.7	28.80 (4.57)	6 (17.1)	1 (2.9)	3 (8.6)	2.38 (3.23-5.74) [+9.0%]*	<.01*
LRA	19.5	44.8	26.52 (5.69)	19.2	69.6	29.48 (8.93)	14 (40.0)	2 (5.7)	2 (5.7)	2.96 (1.43-4.50) [+11.2%]*	<.01*
Aortic bifurcation	15.3	42.5	25.40 (5.90)	15.7	57.7	26.36 (8.18)	-	-	-	0.96 (−0.78 to 2.70) [+3.8%]	.27
RCIA	9.1	29.9	16.05 (4.99)	8.6	35.3	17.21 (5.61)	-	-	-	1.16 (−0.15 to 2.46) [+7.2%]	.08
LCIA	9.1	26.8	15.50 (4.08)	9.9	32.0	17.69 (5.33)	-	-	-	2.19 (1.02-3.35) [+14.1%]*	<.01*

TEVAR, Thoracic endovascular aortic repair; *SD*, standard deviation; *CI*, confidence Interval; *LSCA*, left subclavian artery; *LIPV*, left inferior pulmonary vein; *SMA*, superior mesenteric artery; *RRA*, right renal artery; *LRA*, left renal artery; *RCIA*, right common iliac artery; *LCIA*, left common iliac artery. *Statistically significant.