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PERIPHERAL

Endovascular Repair of Acute and Chronic Aortic Type B Dissections

Main Factors Affecting Aortic Remodeling and Clinical Outcome

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ABSTRACT

OBJECTIVES The aim of this study was to assess factors influencing the clinical outcome and morphological changes of acute and chronic type B aortic dissection after thoracic endovascular aortic repair (TEVAR).

BACKGROUND Aortic remodeling after TEVAR may be associated with clinical outcome, complications, and endoleak development.

METHODS Sixty cases of TEVAR for complicated type B acute aortic dissection (AAD) (n = 29) and chronic aortic dissection (CAD) (n = 31) with a minimum follow-up of 3 years were retrospectively reviewed. Using computed tomography images, we assessed true lumen, false lumen, and total aortic short-axis diameters. Six procedural factors were analyzed in relation to aortic remodeling and other outcomes. Analysis of variance was used to compare short-axis, false lumen, and true lumen diameters during the follow-up period. Univariate and multivariate analyses were used to assess the relationship between procedural factors and multiple outcomes.

RESULTS A total of 100 stent grafts were implanted in 60 consecutive patients with acute aortic dissection (AAD) and CAD. Aortic remodeling consisting of false lumen thrombosis and shrinkage was more prominent in AAD than in CAD, especially within the first 18 months. Of note, the entire aortic diameter increased significantly cephalad to the stent graft in AAD. Only in the AAD group there was increased aortic remodeling related to post-dilation of the stent graft. Type I and II endoleaks occurred in 17 patients (28%); in AAD, embolization of the left subclavian artery after stent graft deployment was significantly associated with a lower risk of endoleak development, but this was not evident in CAD.

CONCLUSIONS Aortic remodeling and clinical outcome after TEVAR can be influenced by procedural techniques (post-dilation and embolization of the left subclavian artery in patients with acute but not chronic aortic dissection). (J Am Coll Cardiol Intv 2016;9:183–91) © 2016 by the American College of Cardiology Foundation.

Thoracic endovascular aortic repair (TEVAR) is an effective treatment for complicated type B aortic dissection in either the acute or chronic phase, with the aims of excluding the false lumen (FL), covering the primary entry tear, and stimulating remodeling of the thoracic aorta (1–4).

Morphological remodeling of the aorta consists of a progressive thrombosis of the FL and contemporary

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ABBREVIATIONS AND ACRONYMS

AAD = acute aortic dissection

CAD = chronic aortic dissection

CTA = computed tomography angiography

FL = false lumen

LSA = left subclavian artery

MAE = major adverse event(s)

TEVAR = thoracic endovascular aortic repair

TL = true lumen

enlargement of the true lumen (TL) without enlargement of the total aortic diameter.

A higher survival rate has been reported in those patients who achieved aortic remodeling (5-mm reduction in the maximal descending thoracic aortic diameter) from pre-operative imaging to final follow-up (1,5).

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The efficacy of TEVAR in facilitating aortic remodeling was highlighted recently by the INSTEAD-XL (Investigation of Stent-grafts in Aortic Dissection) trial. In this study, TEVAR in uncomplicated dissection produced aortic remodeling and reduced aorta-related mortality compared with medical therapy (2). A new frontier in the endovascular treatment of thoracic aortic dissection is to explore the morphological changes that occur in the stented segment and in the adjacent aorta when followed over a long-term follow-up period. It has been suggested that acute aortic dissections (AADs) show a higher degree of remodeling compared with chronic aortic dissections (CADs); however, certain procedural factors that may affect further clinical outcome, morphological changes, and endoleaks have not been fully explored (1-6).

Our study aimed to retrospectively evaluate the procedural factors that may affect morphological changes of type B AAD and CAD after TEVAR as well as endoleak development and clinical outcome.

METHODS

A retrospective study analysis was performed in 60 consecutive patients (age, 69 ± 3.5 years; male/female, 40/20) who underwent endovascular treatment for acute and chronic Stanford type B aortic dissection from November 2009 to January 2011.

Dissections were classified according to the American College of Cardiology/American Heart Association guidelines and of the Expert Consensus of the Society of Thoracic Surgeons Endovascular Surgery Task Force: acute was defined as ≤14 days from the onset of symptoms; chronic dissection (CAD) was defined by an elapsed time ≥14 days from symptom onset (2,4).

Indications for TEVAR in patients with AAD were aortic rupture (hemothorax, hemomediastinum), malperfusion syndrome (acute limb ischemia, bowel ischemia, renal ischemia), persistent pain for >3 days or hypertension refractory to medical treatment.

Chronic dissections were considered for intervention in the presence of complications as impending rupture and organ ischemia or unstable blood pressure, maximal short-axis thoracic aortic diameter >55 mm, or rapid growth (10 mm/year) of the thoracic aorta. Patients with underlying inherited (e.g., Marfan syndrome) and inflammatory disease (e.g., Takayasu syndrome) were not included in the study.

All patients who presented with acute onset of symptoms (<14 days) not responding to medical therapy were treated within a few days (1 to 6 days) from admission. If an acute patient is stable with no complications, we wait 14 days to decide whether TEVAR is indicated.

Each patient was informed of the intended treatment and possible complications, and written consent was obtained before the procedure.

The study was approved by the local ethics committee and the internal review board.

Before the procedure and during the follow-up (5 days and 6, 12, 24, and 36 months), all patients were evaluated with clinical assessment and computed tomography angiography (CTA). In case of anomalous findings, digital subtraction angiography was performed.

Clinical success was defined when pre-operative pathological condition and symptoms (i.e., malperfusion syndrome) were resolved by TEVAR.

Using CTA in the true axial plane, we evaluated the short-axis diameter of the whole aorta, the TL and TL at 7 different levels: 3 cm and 1 cm above the stent graft, superior edge of the stent graft, midlevel of the stent graft, inferior edge of the stent graft, and 1 cm and 3 cm below the stent graft.

The aortic changes regarding the morphology and diameter of the TL and FL were assessed and compared with the pre-treatment CTA findings.

Univariate and multivariate analyses were used to assess the following factors that have been associated with endoleaks and major adverse events (MAE) (defined as mortality, lack of clinical success, and major complications): stent graft oversizing; balloon dilation after stent graft deployment; number of stent grafts implanted; length of covered thoracic aorta; covering of the left subclavian artery (LSA); and embolization of the LSA. All these procedural details were analyzed and correlated with the presence of aortic remodeling, endoleaks, or MAE.

STATISTICAL ANALYSIS. A statistical analysis was done using SPSS version 14.0 (SPSS Inc., Chicago, Illinois) and Excel for Mac 2008. Continuous variables were presented as mean with 95% confidence intervals. Comparison of continuous variables was made by the Student *t* test. Procedural factors associated with absence of aortic remodeling, occurring endoleaks, or complications were explored using a

Cox proportional hazards model. Factors being found statistically significant ($p < 0.05$) in the univariate analysis were included in the multivariate analysis. Results are represented as hazard ratios and 95% confidence intervals. Hierarchical analysis was performed for the number of stent grafts because in some patients more than 1 stent graft was implanted.

Categorical variables were analyzed by the chi-square and Fisher exact tests. Analysis of variance was used to compare short axis, FL, and TL diameters over the follow-up period. Regression analysis was also used to analyze the FL thrombosis in relation to the lack of MAE and endoleaks. Significance was assumed at $p < 0.05$.

RESULTS

Patient cardiovascular risk factors and clinical presentation are presented in Table 1. Twenty-nine patients (48.3%) were included in the AAD group, and 31 patients (51.7%) in the CAD group.

Clinical success was obtained in 27 patients with AAD (93.1%) and in all patients with CAD. In 2 patients with AAD, malperfusion syndrome was not immediately resolved after the first TEVAR, and therefore an additional stent graft has to be deployed to resolve the malperfusion syndrome within 7 days. Clinical outcomes are reported in Table 2. Technical success was achieved in all patients with correct deployment of the stent grafts and complete exclusion of the primary entry tear.

Different commercially available stent grafts were used: Thoracic Excluder Graft (W.L. Gore & Associates, Flagstaff, Arizona) (n = 57); Talent LPS (Medtronic, Minneapolis, Minneapolis) (n = 22); Valiant-Captivia (Medtronic) (n = 13); Zenith TX (Cook Medical, Inc., Bloomington, Indiana) (n = 6), Relay (Bolton Medical Inc., Sunrise, Florida) (n = 2).

The total number of implanted stent grafts was 100: 43 in the AAD group and 57 in the CAD group. Mean number of stent grafts per patient in the AAD group was 1.5 ± 0.5 and 1.9 ± 0.8 in the CAD group (AAD vs. CAD; $p = 0.01$).

A summary of the technical features of all procedures is given in Table 3. In the AAD group, the caliber of the proximal implanted stent graft ranged from 28 mm to 40 mm (mean, 34 ± 4.4 mm), while the length ranged between 10 and 20 cm (mean, 15 ± 4.3 cm).

In the CAD group, the stent graft diameters ranged from 31 to 47 mm (mean, 39 ± 5.8 mm) with a length ranging between 10 and 40 cm (mean, 25 ± 11 cm). The mean proximal neck diameter was 30.8 ± 4.4 mm in AAD patients and 32.5 ± 3.1 mm in CAD patients ($p = 0.04$).

Stent graft oversizing (mean value) in both groups ranged between 6% and 24% (mean, of 14.9 ± 6.3%), 13.8% ± 3.5% in the AAD group and 15.5 ± 3.3% in the CAD group, with no significant statistical difference between the 2 groups ($p = 0.31$). The degree of stent graft oversizing was less in those patients in whom a proximal endoleak subsequently developed (type Ia) during the follow-up (mean, 4.3 ± 1.7 mm) than in those patients who did not (mean, 5.3 ± 2.2 mm). However, final values were not significantly different from a statistical point of view in the 2 groups ($p = 0.09$).

Univariate and multivariate analyses confirmed that stent graft oversizing in both AAD and CAD did not influence aortic remodeling, endoleak, or MAE (Tables 4 and 5). The proximal and distal stent graft diameters were, respectively, 32.5 ± 1.7 mm and

TABLE 1 Clinical Features of AADs and CADs Included in the Study

Clinical Features	AADs	CADs	p Value
Age, yrs	68 ± 5.5	71 ± 3.5	0.7
Male/female	21/11	19/9	0.9
Hypertension	18 (62)	27 (87)	0.03
Diabetes	9 (31)	10 (32)	0.1
Obesity	6 (20)	6 (19)	0.1
Ischemic heart disease	5 (26)	4 (12)	0.1
Peripheral vascular disease	4 (13)	7 (22)	0.1
Smoking	2 (6.8)	6 (19)	0.03
Drug/alcohol abuse	2 (6.8)	1 (3)	0.07
Clinical presentation			
Signs of aortic rupture	5 (17)	0 (0)	0.01
Malperfusion syndrome	17 (58.6)	3 (9.6)	0.01
Impaired blood pressure	7 (24.1)	17 (54.8)	0.01
Aortic diameter (≥55 mm)	0 (0)	4 (12.9)	0.04
Rapid growth (≥10 mm/yr)	0 (0)	7 (22.5)	<0.01

Values are mean ± SD, n, or n (%).
AADs = acute aortic dissections; CADs = chronic aortic dissections.

TABLE 2 Comparative Evaluation of the Outcomes Post-TEVAR in Patients With AAD and CAD

Outcome	AAD (n = 29)	CAD (n = 31)	p Value
Major adverse events	10 (34.4)	4 (12.9)	0.05
Lack of clinical success	2 (6.9)	0 (0)	0.35
Mortality, 36 months	4 (13.7)	2 (6.4)	0.35
Endoleak	8 (14)	5 (17)	0.28
Major complications	4 (14) (1 pseudoaneurysm of the left axillary artery; 2 retrograde extensions of the dissection; 1 temporary paraparesis)	2 (3) (1 thoracic aortic aneurysm formation; 1 dissection of the left external iliac artery)	0.35

Values are n (%).
TEVAR = thoracic endovascular aortic repair; other abbreviations as in Table 1.

TABLE 3 Technical Data for All Procedures in Patients With AAD and CAD

Procedural Data	AAD	CAD	p Value
Stent grafts	43	57	0.048
Distance of aorta covered, mm	174 ± 18	198 ± 20	0.02
Intentional coverage of the LSA	14 (48)	19 (61)	0.23
Intentional LSA embolization after stent graft deployment	6 (20.6)	5 (16.1)	0.9
Stent graft dilation	18 (62)	18 (58)	0.1
Mean oversizing, mm	4.5 ± 2.0 (13.8)	6.8 ± 2.1 (15.5)	0.07

Values are n, mean ± SD, n (%), or mean ± SD (%).
LSA = left subclavian artery; other abbreviations as in Table 1.

26.9 ± 1.2 mm in AAD and 33.9 ± 1.3 mm and 29.7 ± 2.0 mm in CAD.

The mean length of the covered thoracic aorta was 174 ± 18 mm in cases of AAD and 198 ± 20 mm in cases of CAD.

To obtain the complete exclusion of the FL, an additional stent graft was necessary in 12 cases of AAD (41%) and in 16 cases of CAD (51%), all of them were DeBakey type IIIb with the dissection extending into the abdominal aorta. The overlapping segment between the 2 stent grafts was at least 5 cm (range, 5.0 to 8.3 cm; mean, 6.6 cm).

The distal landing zone of the stent grafts was between the T5 and L1 vertebral bodies. Univariate analysis showed that neither the number of stent grafts implanted nor the length of aorta covered had any significant relationship with improved FL thrombosis, endoleaks, or MAE in both AAD and CAD (Tables 4 and 5).

To reduce the risk of paraplegia in those patients (n = 20) whose thoracic descending aorta was totally covered by the stent graft, cerebral spinal fluid pressure was monitored by the insertion of a lumbar catheter before intervention.

The endovascular treatment of AAD resulted in a significant increase in TL diameter over time (from a mean of 20.4 mm calculated from all indicated aorta levels to 28 mm after 36 months, p = 0.02) and a significant decrease in the FL diameter (from a mean value calculated from all indicated aorta levels of 9.1 to 5 mm at 36 months; p = 0.01).

However, the total maximum diameter of the aorta in AAD was not significantly altered at level of the stent graft and below (from level 4 to 7) from a mean value of 28.8 mm to 29.3 mm at 36 months (p > 0.05), with a mean overall increase of +0.47 mm. However, we noted a significant increase in the aorta diameter above the stent graft (from level 1 to 3) from a mean value of 31.5 mm to 35.5 mm at 36 months (p = 0.05), with a mean overall evolution of +4.5 mm (Figure 1).

In CAD, TEVAR did not significantly modify the total aortic caliber (mean at all levels: from 31.8 mm at discharge to 33.3 mm at 36 months; mean increase of +1.1 mm; p > 0.05), also regarding the TL and FL either at the level of the stent graft (TL mean increase of +3 mm at 36 months [p > 0.05]; FL mean decrease of −0.73 mm [p > 0.05]) or in the aorta below the endograft (mean increase in the TL of +2.5 mm [p > 0.05]; mean decrease in the FL of −1.5 mm [p > 0.05]).

Measurement changes of TL, FL, and total diameter at different levels over time are shown in Figures 1 to 3.

Over 36 months, the decrease in the FL corresponded to greater FL thrombosis in the AAD group than in the CAD group (AAD, 95% vs. CAD, 70%; p < 0.01).

In AAD, FL thrombosis occurred more frequently within the first 18 months after the procedure (Figure 4).

Regression analysis showed that in AAD, FL thrombosis was directly related to freedom from MAE and endoleaks (p = 0.01) (Figure 4); however, this was not observed for CAD (p = 0.1) (Figure 5).

Type I and II endoleaks were detected in 17 of 60 patients (28%): 3 type I (17.6%) and 14 type II (82.3%). In the AAD group, 2 type I and 6 type II endoleaks occurred, whereas in the CAD group, there was 1 type I endoleak and 8 type II endoleaks. Type II endoleaks were present in 14 cases: in 3 of 14 (2.1%) cases, a type II endoleak was evident at CTA before discharge. All of them originated from the excluded LSA (proximal neck <2 cm). Two endoleaks sealed spontaneously during follow-up and were no longer apparent at 6 and 9 months CTA. In the other 12 cases, deliberate embolization of the FL and of the origin of the excluded LSA was performed using a variety of agents.

Endoleak characteristics and embolization techniques are shown in Table 6. However, after reintervention, all endoleaks were successfully sealed.

TABLE 4 Univariate and Multivariate Analysis of Procedural Factors in Relation to Improved Remodeling and Complications in AADs (Endoleak, Mortality, and Others)

	Univariate		Multivariate	
Factors	p Value	HR (95% CI)	p Value	HR (95% CI)
Stent graft oversizing	0.915	1.12 (0.41–2.24)		
Balloon dilation after stent deployment	0.031	0.36 (0.12–0.94)	0.754	0.81 (0.25–0.83)
No. of stent grafts implanted*	0.845	0.89 (0.51–3.21)		
Length of aorta covered	0.120	1.01 (0.95–2.32)		
Covering of the LSA	0.45	1.05 (0.22–1.09)		
Embolization of the LSA during TEVAR	0.04	0.42 (0.18–1.10)	0.05	0.51 (0.31–0.88)

*Hierarchical analysis.
CI = confidence interval; HR = hazard ratio; other abbreviations as in Tables 1 to 3.

MANAGEMENT OF THE LSA. The origin of the LSA was intentionally occluded in 14 of 29 patients (48%) in the AAD group and in 19 of 31 patients (61%) in the CAD group. In 11 patients (18%, 6 AADs and 5 CADs), the origin of the occluded LSA was embolized during the initial procedure, immediately after stent graft deployment to avoid FL revascularization. In 6 patients (4 AADs and 2 CADs), in whom the LSA was excluded only by the stent graft (without direct embolization), a type II endoleak developed. A secondary intervention was performed, via a left brachial approach, to exclude the LSA origin, and complete sealing of the leak was successfully achieved in all of them. As the univariate analysis showed, LSA coverage had no bearing on the development of type II endoleaks, in either AAD or CAD ($p > 0.05$); instead embolization of the LSA was observed to confer some protection against endoleak development in AAD ($p = 0.04$), but not in CAD ($p = 0.39$). Moreover, embolization of the LSA had a direct influence on improvement of FL thrombosis and a reduction in endoleak development but not MAE ($p = 0.05$) (Table 4). On multivariate analysis, either covering or embolization of the LSA in CAD did not demonstrate an association with other outcomes such as improved aortic remodeling, mortality, and other MAE. Postoperative carotid-subclavian surgical bypass was needed in only 2 patients with AAD (2 of 14, 14.2%) who experienced neurological symptoms. No spinal cord ischemia associated with intentional coverage of the LSA has been observed.

POST-DILATION. Dilation of the stent graft was performed to obtain a better seal to the aortic wall at the level of the proximal and distal landing zones. Moreover, it was also performed when multiple devices were inserted at the level of the overlapping segments in 18 patients (62%) in the AAD group and in 18 patients (58%) in the CAD group.

In both groups, the rate of endoleaks was not significantly different between patients who underwent stent graft dilation and those who did not. In fact, in patients with AAD, 8 endoleaks occurred; endoleaks occurred in 3 of 18 patients who underwent post-dilation and in 5 of 11 in patients who did not ($p = 0.09$). In CAD, 9 endoleaks occurred: 4 in patients with post-dilation and 5 in patients without ($p = 0.9$).

In AAD over 36 months, FL thrombosis rates were higher in patients who had undergone stent graft dilation compared with those patients who did not (17 of 18 vs. 7 of 11; $p < 0.05$). On univariate and multivariate analyses, post-stent deployment dilation was correlated only with improved FL thrombosis (univariate analysis, $p = 0.031$) but not with a greater higher freedom from MAE or endoleaks (Table 3) (multivariate analysis, $p = 0.754$).

In the CAD group, the FL thrombosis rate, observed at 36 months of follow-up, was constant over that period but was not directly related to freedom from MAE or endoleaks (Figure 5). Moreover, multivariate analysis showed different results for patients who underwent stent graft dilation and those who did not (Table 4).

However, the 2 cases of antegrade dissections were not associated with dilation of the stent graft as the complication, which, in both cases, occurred immediately after stent graft deployment.

TABLE 5 Univariate and Multivariate Analysis of Procedural Factors in Relation With Endoleak and Other Outcomes (Endoleak, Mortality, and Others) in CADs

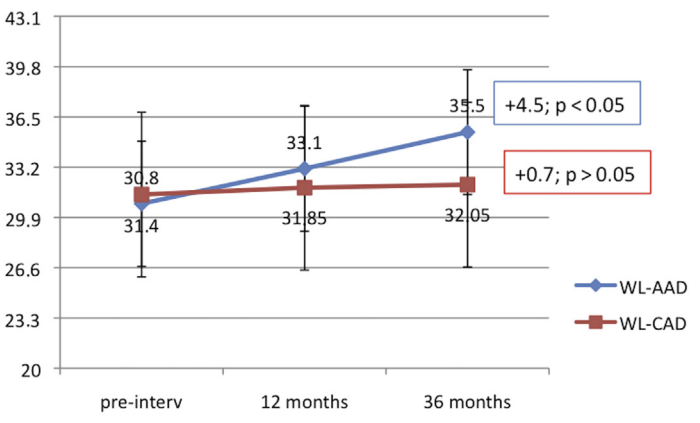
Factors	Univariate		Multivariate	
	p Value	HR (95% CI)	p Value	HR (95% CI)
Stent graft oversizing	0.971	1.08 (0.40–4.03)		
Balloon dilation after stent deployment	0.11	0.31 (0.35–6.21)		
No. of stent grafts implanted*	0.789	1.18 (0.26–4.23)		
Length of aorta covered	0.581	1.37 (0.55–5.15)		
Covering of the LSA	0.87	0.91 (0.47–2.88)		
Embolization of LSA during TEVAR	0.39	0.57 (0.44–3.24)		

*Hierarchical analysis.
Abbreviations as in Tables 1 to 4.

DISCUSSION

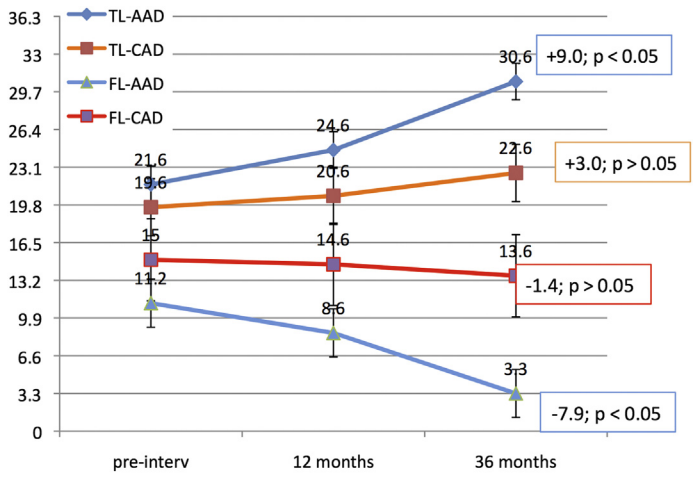
Several articles have outlined that the rationale of endovascular treatment for type B dissections relies on the exclusion of the primary entry tear inducing

FIGURE 1 Aortic Remodeling Above the Stent-Graft



Whole lumen (WL) changes over time in acute aortic dissection (AAD) and chronic aortic dissection (CAD) above the stent graft (levels 1 and 2). Interv = intervention.

FIGURE 2 Aortic Remodeling at the Level of the Stent-Graft



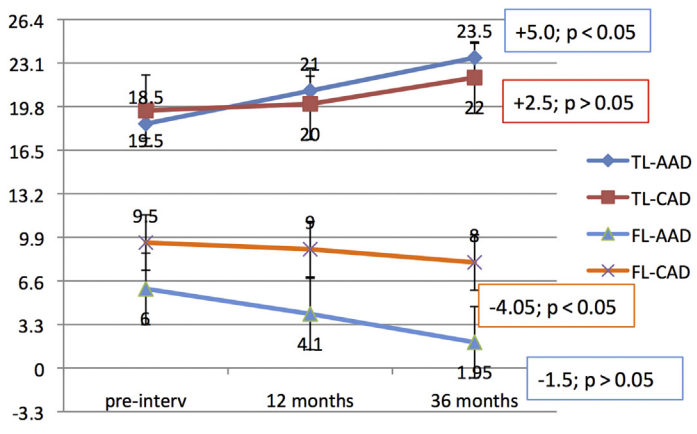
True lumen (TL) and false lumen (FL) changes over time in acute aortic dissection and chronic aortic dissection at the level of the stent graft (levels 3 to 5). Abbreviations as in Figure 1.

aortic remodeling and avoiding major complications either in an acute or chronic condition (1-5,7).

As reported, aortic remodeling consists of FL thrombosis with progressive reduction of its diameter and concomitant progressive enlargement of the TL (5,8-11).

However, in chronic dissections, some reports indicate that endovascular repair should not significantly alter the aortic morphology (10). This behavior reflects the lower FL thrombosis rate that we observed in CAD compared with AAD (**Figures 1 to 5**).

FIGURE 3 Aortic Remodeling Below the Stent-Graft



TL and FL changes over time in AAD and CAD below the stent graft (levels 6 and 7). Abbreviations as in Figures 1 and 2.

In our study, FL thrombosis was more evident in AAD within the first 18 months after treatment. These outcomes are similar to the results of other recent studies that stress the higher capacity of remodeling of acute rather than chronic dissection after TEVAR (5,9,11,12).

Our results confirmed that in AAD, FL thrombosis and shrinkage is directly proportional to the absence of MAE and endoleaks (**Figure 4**). By comparison, FL thrombosis in CAD is less prominent initially and at the same time has a weak correlation with MAE and endoleak occurrence. This finding is probably related to the fact that in our cohort mainly type II endoleaks from the LSA occurred (**Figure 5**). Apart from FL and TL changes in size, which are well known, the entire lumen of the aorta above the stent graft has not yet been analyzed (13,14). Our results showed that the whole aortic lumen tends to increase significantly above the stent graft (+4.5 mm in 36 months) in AAD, but not in CAD. This may be related to the increased active remodeling of the aortic wall that is typical of AAD and also associated with increased shear stress forces above the stent graft, which may lead to aneurysm formation or retrograde type A dissection.

We also analyzed some procedural factors that may affect the fate of the aorta after stent graft deployment. As far we are aware, these procedural factors in relation to aortic remodeling have not previously been clearly examined.

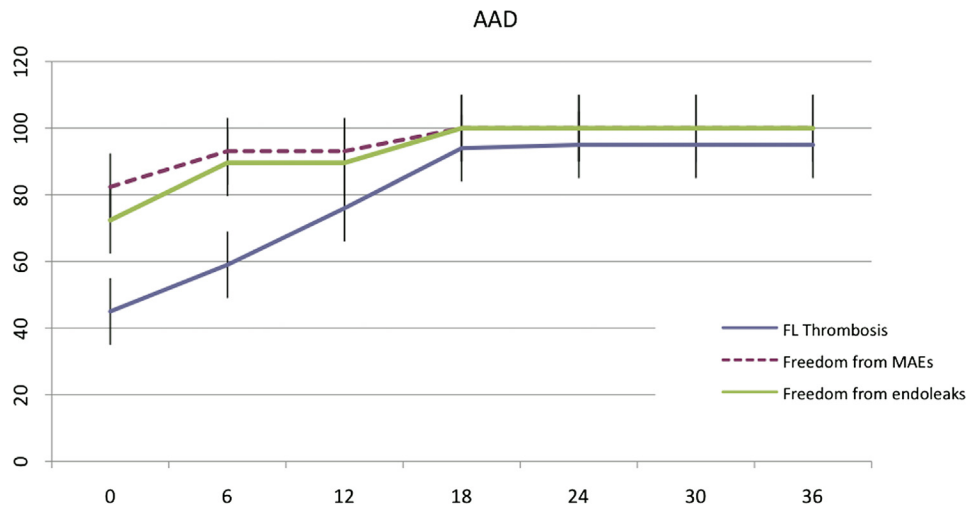
For example, stent graft oversizing is still contentious (15-18). Multiple factors must be evaluated, especially with regard to the patient's age and nature of the aortic disease (e.g., acute dissection, chronic dissection, trauma).

Stent graft oversizing has been recommended to be minimal (≤20%), using the nondissected midaortic arch as the reference vessel diameter, in those patients with a history of trauma (2,15,16). In fact, greater oversizing may be associated with endograft collapse (infolding) and consequent incomplete sealing (17,18). Moreover, in the case of AAD, a decrease in the degree of stent graft oversizing potentially reduces the barotrauma on the fragile aortic wall, reducing the risk of antegrade dissection or aortic rupture. In 1 study, stent graft oversizing ≥20% was a significant predictive factor for reintervention (19).

In our experience, the degree of stent graft oversizing was observed to be slightly higher in CAD (15.5%) than in AAD (13.8%) ($p = 0.07$). What we observed is that stent graft oversizing has a bearing on either endoleak or MAE in either AADs or CADs (**Tables 3 and 4**).

There is a paucity of literature regarding outcomes and long-term morphological changes in

FIGURE 4 Clinical Outcomes and False Lumen Thrombosis in AAD

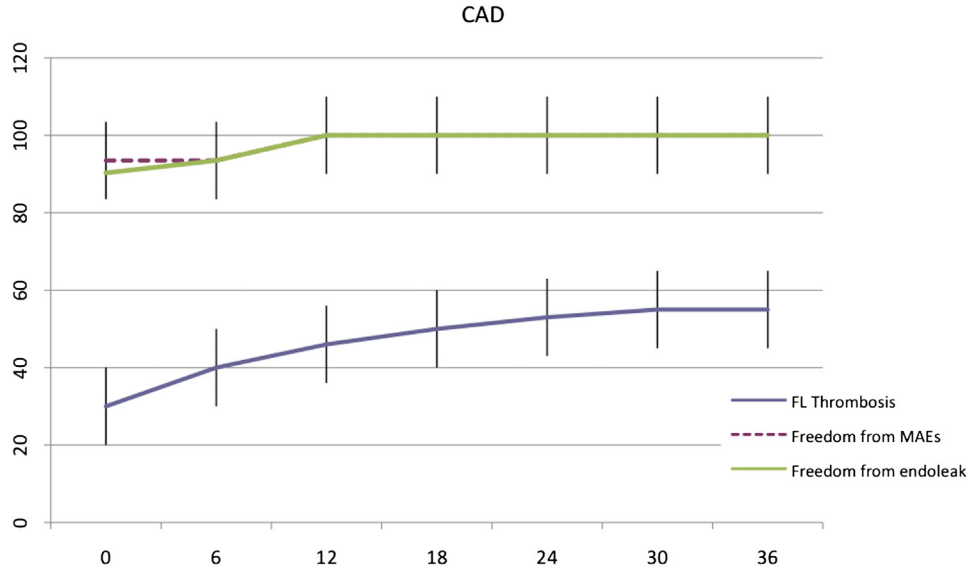


Kaplan-Meier curves showing the direct relationship between freedom from major adverse events (MAEs)/endoleaks and FL thrombosis over the time in AAD ($p = 0.01$). Abbreviations as in Figures 1 and 2.

both AAD and CAD in relation to stent graft dilation (5,8-18,20).

In our study, we investigated the relationship between stent graft dilation, clinical outcomes, incidence of complications, and aortic remodeling in both AAD and CAD. In AADs, at both 18 and 36 months, higher FL thrombosis rates were observed in those patients in whom dilation was performed. From these results, we might suggest that stent graft dilation improves FL thrombosis, especially in AAD (95% AAD vs. 70% CAD of patients with complete FL thrombosis; $p < 0.01$). Hence, improved FL

FIGURE 5 Clinical Outcomes and False Lumen Thrombosis in CAD



Kaplan-Meier curves showing nonsignificant relationship between freedom of MAEs/endoleaks and FL thrombosis over time in CAD ($p = 0.1$). Abbreviations as in Figures 1, 2, and 4.

TABLE 6 Description and Treatment of the Endoleaks

Patient #	Type of Endoleak	Dissection	Origin	Treatment	Follow-up (Months)
1	II	AAD	LSA	Embolization with coils and Onyx	62
2	II	AAD	LSA	Embolization with coils and Onyx	36
3	II	AAD	LSA	Embolization with coils	38
4	I	AAD	Distal (low flow)	Embolization with coils and Onyx	41
5	II	AAD	LSA	Embolization with coils and Onyx	39
6	II	AAD	LSA	Embolization with coils	36
7	I	AAD	Distal (low flow)	None	36
8	II	CAD	LSA	Embolization with coils	39
9	II	AAD	LSA	None	36
10	II	CAD	LSA	None	51
11	II	CAD	LSA	Embolization with Onyx	39
12	II	CAD	LSA	Embolization with coils	37
13	I	CAD	Distal (low flow)	None	49
14	II	CAD	LSA	Embolization with coils and Onyx	36
15	II	CAD	LSA	Embolization with coils and Onyx	52
16	II	CAD	LSA	Embolization with Onyx	36
17	II	CAD	LSA	Embolization with coils and Onyx	38

Abbreviations as in Tables 1 and 3.

thrombosis has been related to improved freedom from endoleak and MAE in AAD (Figure 4). On the contrary, in the CAD group, there was no significant difference in the FL thrombosis rate between patients who underwent stent graft dilation and those who did not (72% dilation vs. 68% no dilation $p > 0.05$).

To explain these differences, one might consider the greater stiffness of the aorta in chronic dissections that prevents stent graft dilation from influencing the aortic remodeling. This concept can be also considered when we evaluate the number of stent grafts implanted. In CAD patients, more stent grafts ($n = 57$) were implanted than in AAD patients ($n = 43$) ($p = 0.048$).

All CAD patients were successfully treated by covering a significantly longer segment of the aorta than in AAD patients (Table 2). This may indicate the need to extend the stent grafts more distally to obtain complete exclusion of the FL and to increase the TL diameter. By comparison, in AAD, shorter distances can be covered given the high clinical success rate and remodeling and the higher risk of paraplegia (Table 2, Figure 4). We observed that stent graft extension was required in 2 AAD patients to treat malperfusion syndrome: from this small experience, we could presume that in some cases, a single module could not be enough, even though this is not statistically significant in either AAD or CAD (Tables 3 and 4). Regarding endoleaks, Sze et al. (6) investigated factors portending endoleaks after TEVAR. Significant factors found were coverage of the LSA, a small radius of aortic arch curvature, and incomplete proximal apposition of the stent graft. However, those factors were significantly associated with complex endoleaks (combination of types Ia and II) but not with pure type II endoleaks, which are mainly present in our population. With regard to coverage of the LSA, intentional exclusion of the LSA without surgical revascularization had been described as a feasible technique (9,21–23). However, according to the results of both our study and that of Sze et al. (6), coverage of the LSA does not seem to be strongly correlated with a different incidence of pure type II endoleaks compared with patients without coverage of the LSA. Furthermore, in our study, we analyzed the effect of embolization of the covered LSA, and this has been significantly inversely related to the development of an endoleak; because this technique significantly reduces endoleak development, it has also been correlated with improved FL thrombosis ($p = 0.05$) (Table 4, Figure 4).

Hence, embolization of the LSA origin might be performed during the initial procedure. Noncritical arm ischemia was reported in several studies (9,22,23). A complete evaluation of the blood flow at the level of the vertebral and mammary arteries is mandatory to avoid neurological complications. The other factors, i.e., aortic arch radius and incomplete apposition of the stent graft, have not been considered in our study because in our case series, we did not find significant variability in aortic arch radius and proximal apposition of the different stent grafts.

STUDY LIMITATIONS. This is a retrospective observational study with a relatively small number of patients and relatively small number of MAEs and endoleaks. In addition, a wide range of different types of stent grafts were used.

CONCLUSIONS

Aortic remodeling after TEVAR can be associated with the characteristics of the dissection and the procedural technique. In particular, TEVAR of AAD results in a greater degree of aortic remodeling than in CAD.

According to the type of aortic dissection, different techniques such as post-dilation, embolization of the LSA, and distal extension of the stent graft may be considered.

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PERSPECTIVES

WHAT IS KNOWN? Beside certain evidence that AADs show a higher degree of remodeling compared with CADs, procedural factors that may affect further clinical outcomes have not been fully explored.

WHAT IS NEW? Aorta remodeling after TEVAR can be related to the characteristics of the dissection and to the procedural technique. Acute dissections seem to be more sensitive than chronic.

WHAT IS NEXT? The most appropriate endovascular techniques may be selected according to the type of aortic dissection.

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KEY WORDS aorta remodeling, aortic dissection, stent graft, TEVAR