Frequency of abdominal aortic expansion after thoracic endovascular repair of type B aortic dissection

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Abstract

Purpose: To determine abdominal aortic expansion after thoracic endovascular aortic repair (TEVAR) in patients with aortic dissection type B and 36 months minimum follow-up.

Methods: Retrospective study of 18 TEVAR patients with follow-up >36 months. Abdominal aortic diameters at celiac trunk (location B) and infrarenal aorta (location C) were recorded on the first and last imaging after TEVAR. False lumen thrombosis was determined at level of endograft (A) and at B and C. Aortic expansion was defined as diameter increase of 5 mm or 15%. Correlation analyses were performed to investigate potential determinants of expansion.

Results: Median follow-up was 75.2 months. Sixteen of 18 patients (88.9%) demonstrated abdominal expansion. Mean expansion was 9.9 ± 6.1 mm at B and 11.7 ± 6.5 mm at C, without a difference between acute and chronic dissections. Critical diameters of 55 mm were reached in two patients treated for chronic dissection (11.1%). Annual diameter increase was significantly greater at locations with baseline diameters >30 mm (2.1 ± 1.1 mm vs. 1.0 ± 0.6 mm, $p = 0.009$). Baseline diameters were greater in patients with chronic dissections.

Conclusion: Abdominal aortic expansion can be frequently recognized after TEVAR for aortic dissection type B and occurs independently from thoracic false lumen thrombosis. Clinical significant abdominal aortic expansion may occur more frequently in patients treated with TEVAR for chronic dissection.

Keywords

Aortic dissection, endovascular repair, expansion, follow-up, imaging

Introduction

Thoracic endovascular aortic repair (TEVAR) has developed into the standard of care concerning treatment of complicated aortic dissections of type Stanford B in cases where aortic reconstruction is indicated.[1] This is valid for both acute symptomatic (AD) and chronic expanding (CD) dissections. Endovascular graft (EVG) implantation aims at sealing off the primary intimal tear (entry) in order to stop the inflow of blood into the false lumen and to give rise to aortic remodeling by inducing false lumen thrombosis. Closure of the primary entry by the EVG with absence of rupture and endoleakage type 1 a or 3 according to White[2] is often considered technical success.[3–5] Because of additional intimal tears (reentries), which almost obligatorily occur especially at the ostia of the visceral arteries in the abdominal aorta, perfusion of the false lumen may be yet maintained after the EVG has been deployed. This implies that aortic expansion potentially leading to aortic rupture may arise after TEVAR for AD and CD independently from successful closure of the primary entry and the degree of thoracic false lumen thrombosis.

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The aim of this study was to determine both the frequency and magnitude of abdominal aortic expansion in a retrospectively analyzed single center cohort of patients with a minimum follow-up length of 3 years after TEVAR for type Stanford B aortic dissections. Moreover, we sought to analyze the relationship between abdominal aortic expansion and potential influencing variables.

Methods

Study design

For this institutional review, board-approved, retrospective, monocentric, exploratory study data of all patients treated with TEVAR for AD or CD between April 2000 and May 2013 were extracted from an institutional database ($n = 90$). Only those individuals were considered for enrollment that met the following inclusion criteria: first, aortic dissection involving the infrarenal abdominal aorta at initial presentation; second, two available postinterventional imaging studies (computed tomography (CT) angiography or magnetic resonance angiography) with a minimum time interval between the first postinterventional (t1, baseline) and the last postinterventional (t2) imaging of 36 months. The primary endpoint was evidence of expansion of the abdominal aorta (at the level of the celiac trunk or at the infrarenal aorta) at t2. Aortic expansion was defined as an increase of the maximum aortic diameter at t2 compared with t1 of at least 5 mm or 15%.

Study population

Seventy-two patients had to be excluded because of early death during postinterventional hospital stay ($n = 4$), unrelated death before reaching minimum follow-up period of 36 months ($n = 1$), follow-up period shorter than 36 months ($n = 40$), lack of sufficient follow-up or follow-up imaging ($n = 19$), aortic dissection not involving the infrarenal aorta ($n = 5$), and previous or concomitant abdominal aortic repair ($n = 3$). No interim aortic ruptures were reported. Thus, 18 patients (50.5 ± 12.3 years, 11 men) matched the inclusion criteria (Tables 1 and 2). The median interval between intervention and t1 was four days (range, 0–882 days). The median follow-up length (t1–t2) was 75.2 months (range, 39.6–142.4 months). Eight of these patients were treated with TEVAR for CD because of thoracic aortic expansion and 10 for AD because of symptomatic dissection. One of the procedures was a distal extension after TEVAR for AD 5 years earlier. Comorbidities are

Table 1. Patients' clinical features and comorbidities.

	Age at TEVAR	HCTD	ASA	AH	DL	CAD	PAD	COPD	Smoking	RI	DM
1	36	1	3	1	0	0	0	0	0	0	0
2	61	0	3	1	0	0	0	0	1	0	0
3	50	0	3	1	1	1	0	0	1	1	1
4	78	0	3	1	1	1	0	0	0	0	0
5	65	0	3	1	0	0	0	0	1	0	0
6	65	0	3	1	1	0	0	0	0	0	0
7	40	1	3	1	0	0	0	0	0	1	0
8	54	0	4	1	0	0	0	0	1	0	0
9	35	0	3	1	0	0	0	0	0	0	0
10	44	0	3	1	0	0	0	0	0	1	0
11	52	0	3	1	0	0	0	0	1	0	0
12	45	0	4	1	0	0	0	0	0	0	0
13	63	0	3	1	0	0	0	0	1	0	0
14	47	0	3	1	0	0	0	0	0	0	0
15	53	0	3	1	1	0	0	0	1	0	0
16	30	1	3	1	0	0	0	0	1	0	0
17	45	0	2	1	0	0	0	0	1	0	0
18	46	0	3	1	0	0	0	0	0	0	0

AH: arterial hypertension; ASA: *American Society of Anesthesiologists* risk index; CAD: coronary artery disease; COPD: chronic obstructive pulmonary disease; DL: dyslipidemia; DM: diabetes mellitus; HCTD: hereditary connective tissue disorder; PAD: peripheral artery disease; RI: renal insufficiency; TEVAR: thoracic endovascular aortic repair. In columns other than ASA, the given number indicates presence (1) or absence (0) of clinical feature or comorbidity.

Table 2. Summary of details on vascular procedures and aortic remodeling in 18 patients after TEVAR for aortic dissection.

Patient	Diagnosis	TL (mm)	RI	AE (mm) (relative diameter increase)	Location of AE	BL (mm)	RE	FLT	FU (months)	OP-t1 (days)
1	CD	130	1[a]	5.9 (20.1%)	C	29.3	2	2	118.6	882
2	CD	150		10.8 (32.0%)	C	33.8	4	2	100.8	5
3	CD	200		8.7 (22.7%)	C	38.3	3	2	41.5	4
4	CD	200		9.8 (27.0%)	C	36.3	3	2	97.7	6
5	CD	150		0	–	25.9	0	3	44.2	4
6	CD	235	1[a]	13.2 (30.8%)	C	42.8	1	1	97.9	2
7	CD	190		6.3 (21.2%)	C	29.7	8	0	92.2	4
8	CD	299		18.7 (43.9%)	C	42.6	7	2	66.2	2
9	AD	100		4.7 (21.5%)	C	21.9	2	1	61.9	722
10	AD	200		5.1 (25.9%)	C	19.7	2	1	149.2	36
11	AD	256		8.8 (34.6%)	C	25.4	3	1	142.4	2
12	AD	150		3.3 (13.4%)	–	24.6	0	0	41.5	5
13	AD	232		14.0 (54.3%)	C	25.8	1	2	124.2	1
14	AD	150		15.3 (60.0%)	C	25.5	2	1	101.1	6
15	AD	150		9.3 (31.0%)	B	30	3	1	46.5	5
16	AD	183		27.0 (115.4%)	C	23.4	2	1	66.9	0
17	AD	228	1[b]	9.9 (37.4%)	B	26.5	3	1	65.7	1
18	AD	221	1[b]	22.6 (107.1%)	C	21.1	0	1	57.3	1

AD: acute aortic dissection; AE: maximum abdominal expansion; BL: baseline abdominal aortic diameter at location of subsequent maximum diameter increase; CD: chronic aortic dissection; FLT: grade of false lumen thrombosis; FU: length of follow-up period; RE: number of reentries; RI: reintervention during study period; TEVAR: thoracic endovascular aortic repair; TL: treatment length (as assessed by centerline analysis if more than one EVG had been implanted).

[a]Transposition of the left subclavian artery.

[b]Distal endograft extension.

listed in Table 1, e.g., all patients were under treatment for arterial hypertension.

Imaging and image evaluation

At t1, all 18 scans were CT angiographies. The slice thicknesses at t1 were 3 mm ($n = 11$), 1 mm ($n = 6$), and 5 mm ($n = 1$). At t2, 14 of the 18 scans were CT angiographies as well. The slice thicknesses were 1 mm ($n = 11$), 3 mm ($n = 2$), and 1.5 mm ($n = 1$). In four patients, magnetic resonance angiographies using a contrast-enhanced three-dimensional fast low-angle shot angiographic sequence protocol were performed on a 1.5 T system (Siemens Magnetom Avanto, Siemens Medical Systems, Erlangen, Germany) with slice thickness of 1.8 mm.

Images were uploaded on a workstation equipped with dedicated postprocessing software (3mensio Vascular, 3mensio Vascular Imaging BV, Bilthofen, The Netherlands). Two readers with multiyear experience in vascular imaging and/or TEVAR (one radiologist, one vascular surgeon) analyzed the imaging data in consensus concerning aortic diameters, extent of false lumen thrombosis, number of reentries, and presence of endoleakages. The studies were presented randomly, and the readers were blinded to patient identity and follow-up time point. Maximum aortic diameters were determined on manually adjusted orthogonal double-oblique multiplanar reformations (MPR) at three locations: thoracic aorta at the level of the maximum diameter along the EVG (location A), abdominal aorta at the origin of the celiac trunk (location B), abdominal aorta at the level of the maximum diameter below the renal arteries (location C).

The grade of false lumen thrombosis was assessed at either location using a 4-point ordinal scale for t2: no thrombosis (grade 0), partial thrombosis of less than 50% extent (grade 1), subtotal thrombosis of more than 50% extent (grade 2), and complete thrombosis (grade 3). The number of reentries between the end of the EVG and the aortic bifurcation was counted at t1. For t2, we investigated whether a secondary tear of the dissection membrane at the distal end of the EVG maintaining abdominal false lumen perfusion developed that was not evident at t1 (distal intimal erosion). A third reader measured the length of the thoracic aortic segment covered by the EVG (treatment length) using a centerline tool at t1 if more than one EVG had been

implanted (Aquarius Intuition, TeraRecon Inc., Foster City, USA). In case of a single EVG, the treatment length equated to the length of the implanted device.

Thoracic endovascular repair

Closure of the primary entry tears was achieved in all patients. Aortic rupture and endoleakages of type 1 a and 3 did not occur (technical success rate 100%). Endoleakages of type 2 were present at t1 in four cases. These endoleakages resolved and were not detectable at t2 in any case. In two patients with postinterventional development of subclavian steal syndrome, secondary transposition of the left subclavian artery was necessary in the early period (7 and 107 days, respectively) after TEVAR with initial coverage of the left subclavian artery. In two other patients, secondary distal extension was performed during the early period (14 and 137 days, respectively). The indications for these reinterventions were symptomatic true lumen collapse with visceral malperfusion and early expansion of the distal descending thoracic aorta. Details on the individual TEVAR procedures are provided in Table 2.

Statistics

Comparisons of diameter changes between aortic locations and differences concerning expansion rates were performed using Mann–Whitney U tests. Pearson's correlation coefficients were determined to investigate the association between potential cofactors of abdominal expansion and abdominal diameter changes (Microsoft Excel, Microsoft Inc., Redmond, Washington, USA with Real Statistics Resource Pack, http://www.realstatistics.com). Aortic diameter changes were visualized using box–whisker plots. All data are presented as mean ± standard deviation unless otherwise indicated.

Results

Abdominal aortic diameter changes

Abdominal aortic expansion according to the study definition occurred in 16 of 18 patients after TEVAR at B (celiac trunk) and/or C (infrarenal aorta) (88.9%, $n=9$ for AD, $n=7$ for CD). In these 16 patients, abdominal aortic expansion was observed at 22 of the 32 analyzed aortic locations: aortas of six patients were expanding at B ($n=5$ for AD, $n=1$ for CD) and of 16 patients at C ($n=9$ for AD, $n=7$ for CD). Six patients demonstrated expansion at both B and C. In 14 of 16 patients with abdominal aortic expansion, the site of maximum diameter increase was location C. Thus, abdominal aortic expansion was limited to B in 0 patients and to C in 10 patients. In three patients, with abdominal aortic expansion, expansion at A was documented as well ($n=1$ for AD, $n=2$ for CD). One of the two patients without abdominal aortic expansion ($n=1$ for AD, $n=1$ for CD) expanded at A (13.3 mm, 35.5%, initially treated for AD).

Diameter increases of the abdominal aorta were in total significantly higher for C (10.6 ± 6.9 mm) than for B (3.8 ± 6.2 mm) ($p=0.001$). When taking only patients with abdominal expansion into account, absolute diameter increases were 9.9 ± 6.1 mm (3.5–20.8 mm) at B and 11.7 ± 6.5 mm (4.7–27.0 mm) at C. Relative diameter increases were 37.3 ± 24.0% (15.3–81.3%) at B and 43.2 ± 29.2% (20.1–115.4%) at C. In nine abdominal aortic locations, the diameter increase was at least 10 mm. A maximum abdominal aortic diameter of at least 40 mm, 50 mm, and 55 mm was reached at t2 in nine, three, and two patients, respectively. See Tables 2 and 3 and Figure 1 for a synopsis on aortic expansion results.

The annual diameter increase was significantly smaller in patients with abdominal baseline diameters below 30 mm at the location of maximum diameter change at t2 (1.0 ± 0.6 mm per year) compared with patients with abdominal baseline diameters above 30 mm at the location of maximum diameter change at t2 (2.1 ± 1.1 mm per year, $p=0.009$). Baseline diameters were significantly higher for patients treated in the setting of CD than of AD for both location B (39.3 ± 5.0 mm vs. 29.4 ± 4.7 mm, $p=0.001$) and C (34.8 ± 6.3 mm vs. 23.1 ± 2.6 mm, $p<0.001$).

In those two patients who had reached critical abdominal aortic diameters of 55 mm, abdominal reinterventions have been indicated after the study period (patient #6 and patient #8). Both patients were primarily treated with TEVAR in the setting of CD and reached more than 55 mm at location C (56.0 mm after 97.9 months follow-up and 61.3 mm after 66.2 months, respectively). Baseline diameters at location C were 42.8 mm and 42.6 mm, respectively. Annual diameter increase was 1.6 mm for patient #6 and 3.4 mm for patient #8. Patient #8 had the second most reentries in our series ($n=7$).

Figure 2 shows box–whisker plots of aortic diameter changes at B and C. These indicate that the interquartile ranges of diameter changes were 1.1–4.9 mm for B and 6.0–13.8 mm for C. Thus, diameter increases of 75%, 50%, and 25% of individuals were 6.0 mm, 9.0 mm, and 13.8 mm at C, respectively. Mean diameter changes per year were 0.7 ± 1.3 mm (range 0–4.9 mm) for B and 1.7 ± 1.4 mm (range −1.9 to 3.8 mm) for C.

Analysis of potential cofactors

Complete false lumen thrombosis along the thoracic aortic segment covered by the EVG was evident at t2

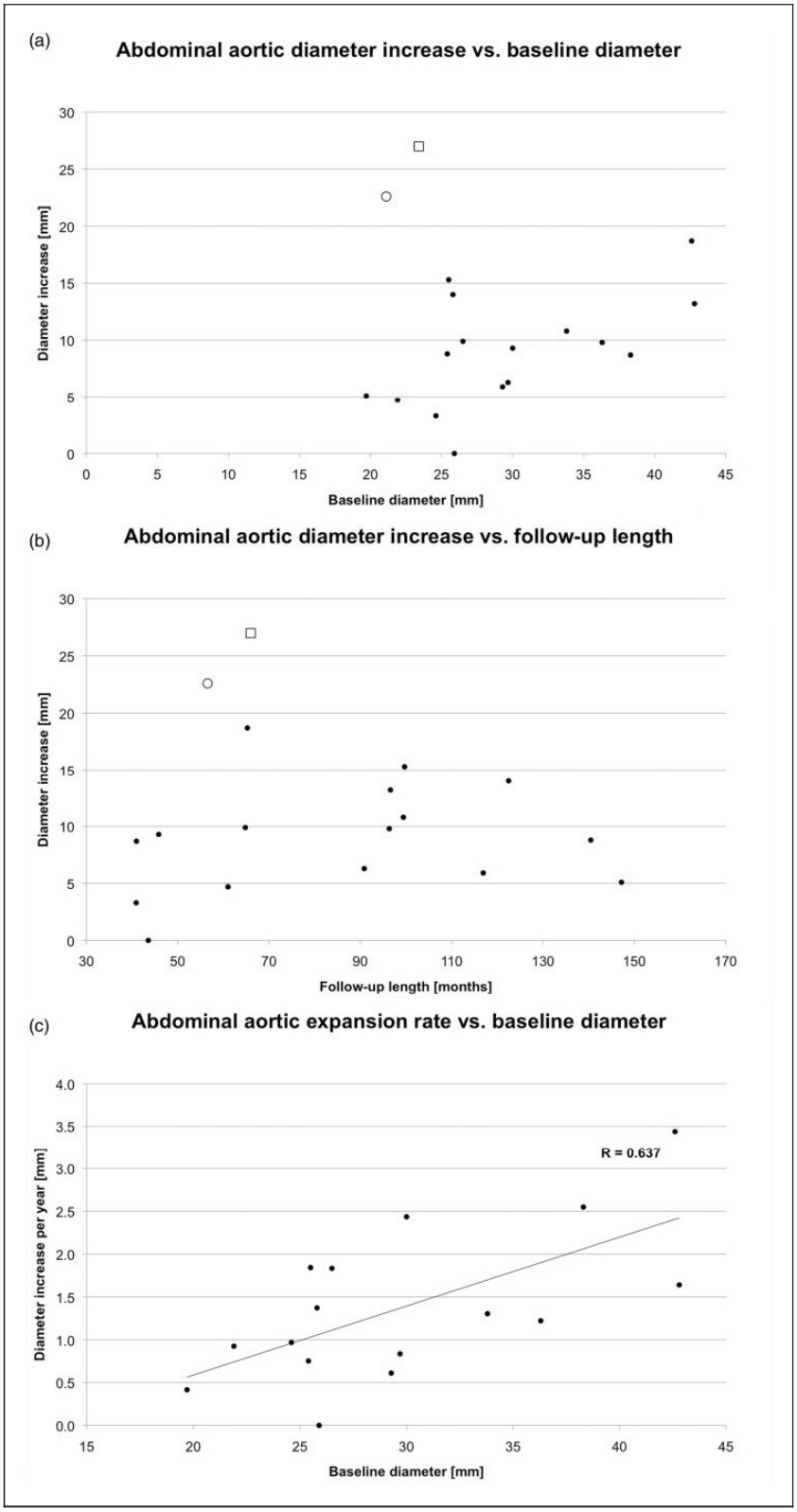

Figure 1. Plots of abdominal aortic diameter development in all 18 patients. Upper two plots show abdominal aortic diameter increase in relationship to baseline diameter (a) and follow-up length (b), respectively. Lower plot illustrates relationship between annual abdominal aortic diameter increase and baseline diameter after exclusion of two outliers (c). Diameters refer to location B (celiac trunk) or C (infrarenal aorta) depending on which one was larger at end of the follow-up period. Open markings indicate the two statistical outliers in upper two plots.

Table 3. Distribution of expansion in patients with evidence of abdominal aortic expansion.

	AD	CD	Total
Number of patients (*n*)	10	8	18
Median age (years)	45.5 (30–63)	57.5 (36–78)	48.5 (30–78)
Median follow-up (months)	66.3 (41.5–149.2)	94.9 (41.5–118.6)	79.5 (41.5–149.2)
AE (*n*)	9	7	16
Expansion at location A if AE			
Number of patients (*n*)	1	2	3
Mean absolute expansion (mm)	6.4	13.1 ± 4	10.8 ± 4.8
Mean relative expansion (%)	23.3	23.0 ± 1.6	23.1 ± 1.2
Expansion at location B if AE			
Number of patients (*n*)	5	1	6
Mean absolute expansion (mm)	10.8 ± 6.3	5.1	9.9 ± 6.1
Mean relative expansion (%)	41.7 ± 24	15.3	37.3 ± 24.0
Expansion at location C if AE			
Number of patients (n)	9	7	16
Mean absolute expansion (mm)	12.6 ± 7.9	10.5 ± 4.4	11.7 ± 6.5
Mean relative expansion (%)	54.9 ± 34.7	28.3 ± 8.3	43.2 ± 29.2

AD: acute aortic dissection; AE: abdominal expansion; CD: chronic expanding aortic dissection; location A: thoracic aorta along endovascular graft; location B: abdominal aorta at celiac trunk; location C: infrarenal abdominal aorta.

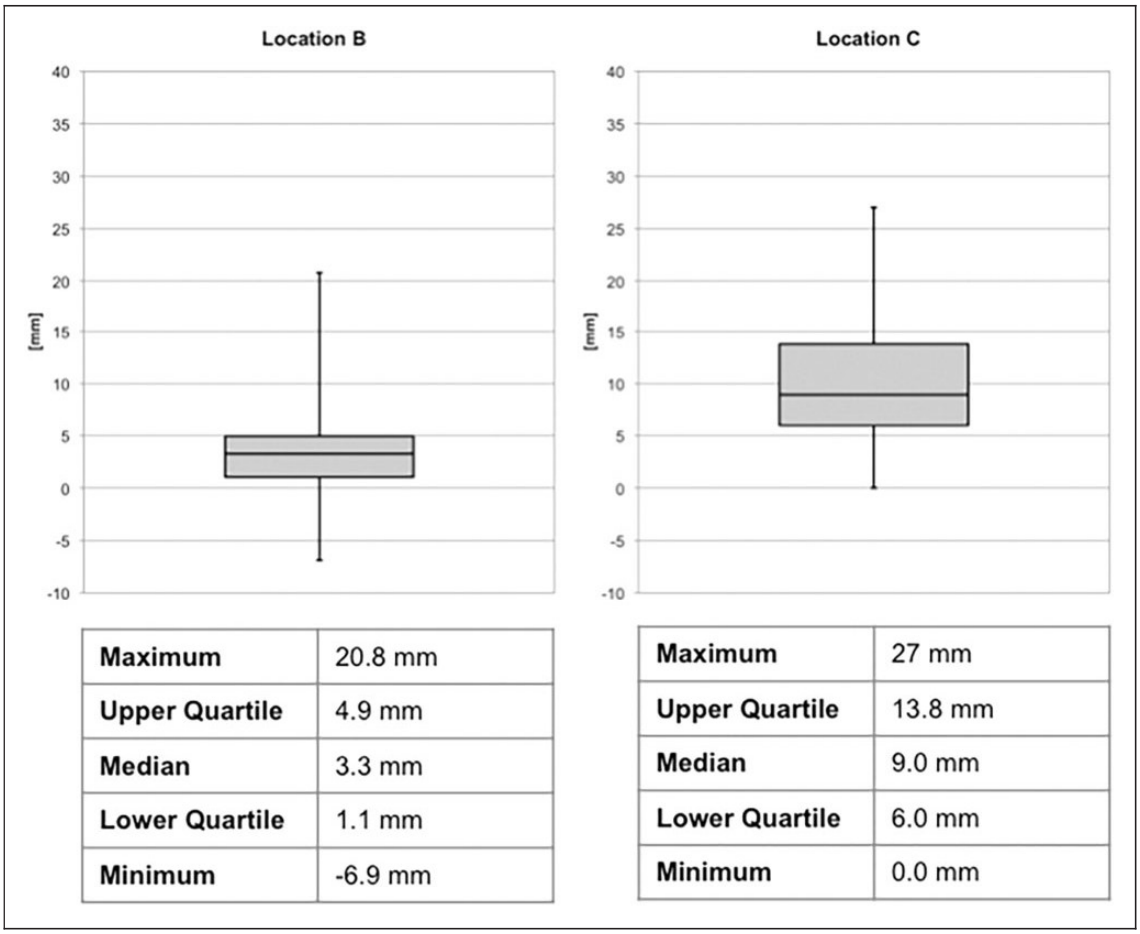

Figure 2. Box–whisker plots of abdominal aortic diameter changes at location B and C. Left and right sections provide quartiles of aortic diameter changes at location B (celiac trunk) and C (infrarenal aorta).

in all patients with abdominal aortic expansion. Complete false lumen thrombosis at B was evident in three patients with abdominal expansion. In these patients, none had complete false lumen thrombosis at C. At B and C, the majority of patients showed grade 0 or grade 1 false lumen thrombosis ($n = 10$ for either location). Both patients without abdominal aortic expansion had subtotal false lumen thrombosis along the thoracic aortic segment covered by the EVG. One of the two patients without abdominal aortic expansion had complete false lumen thrombosis along the abdominal aorta (locations B and C). See Table 4 for a synopsis on the distribution of false lumen thrombosis. See Figures 3 and 4 for examples of abdominal aortic expansion occurring despite favorable development of the thoracic aorta at A.

The number of thoracoabdominal reentries distal to the EVG visible at t1 was 2.6 ± 2.1 averaged over all patients. In patients with abdominal aortic expansion, the number of thoracoabdominal reentries was 2.9 ± 2.1. In both patients without abdominal aortic expansion, there were no reentries evident on postinterventional imaging.

Reviewing the source data of absolute abdominal expansion, it becomes apparent that two individuals show remarkably differing growth tendencies than the other study subjects (patients #16 and #18, Table 2, Figure 1a). These two individuals developed extraordinary abdominal expansion despite follow-up length below median (27.0 mm in 66.9 months and 22.6 mm in 57.3 months, respectively, Figure 1b). Critical abdominal diameters of 55 mm had not been reached during the study period. In patient #18, who was treated in the setting of AD and received distal endograft extension because persisting abdominal true lumen collapse two weeks after primary TEVAR, there was evidence of distal intimal erosion at t2 with a newly formed secondary entry tear immediately distal to the EVG sustaining antegrade abdominal false lumen perfusion (Figure 3). Distal intimal erosion or other procedure-related findings reinforcing false lumen perfusion were not present in the other patients. Patient #16, who was treated in the setting of AD as well, was the youngest individual in our cohort and was suspected to suffer from hereditary connective tissue disorder (Figure 4). She developed an acute dissection of the ascending aorta after the end of this study and 6 years after primary TEVAR at our institution. Two more subjects were suspected to suffer from hereditary connective tissue disorder, too (patients #1 and #7). These two individuals belong to the group of CD patients in which abdominal expansion was present, but have shown only moderate abdominal expansion during the study period (5.9 mm in 118.6 months and 6.3 mm in 92.2 months, respectively, Table 2).

Table 4. False lumen thrombosis at late follow-up (t2) in presence of abdominal aortic expansion.

	AD	CD	Total
FLT at location A if AE			
Grade 0	0	0	0
Grade 1	0	0	0
Grade 2	0	0	0
Grade 3	9	7	16
FLT at location B if AE			
Grade 0	2	3	5
Grade 1	3	2	5
Grade 2	1	2	3
Grade 3	3	0	3
FLT at location C if AE			
Grade 0	2	1	3
Grade 1	6	1	7
Grade 2	1	5	6
Grade 3	0	0	0

AD: acute aortic dissection; AE: abdominal expansion; CD: chronic expanding aortic dissection; FLT: false lumen thrombosis; location A: thoracic aorta along endovascular graft; location B: abdominal aorta at celiac trunk; location C: infrarenal abdominal aorta.

Pearson's correlation coefficients for age, treatment length, baseline diameter, and follow-up length in relation to absolute diameter changes at the abdominal location of maximum diameter increase did not show substantial linear dependencies if all patients were considered. The same was the case for the annual absolute diameter change in relation to the baseline diameter. If subgroups of patients with postintervential imaging features clearly favoring expansion (patient #18 with distal intimal erosion) and with suspicion of hereditary connective tissue disorders (patients #1, #7, and #16) were secondarily excluded from correlation analysis, Pearson's correlation coefficients for treatment length and baseline diameter versus absolute diameter increase ($r > 0.5$ each) and annual absolute diameter increase versus baseline diameter ($r > 0.6$) increase and indicate good linear correlation (Table 5).

The annual diameter increase was significantly smaller in patients with abdominal baseline diameters below 30 mm at the location of maximum diameter change at t2 (1.0 ± 0.6 mm per year) compared with patients with abdominal baseline diameters above 30 mm at the location of maximum diameter change at t2. (2.1 ± 1.1 mm per year, $p = 0.009$). Baseline diameters were significantly higher for patients treated in the setting of CD than of AD for both location B (39.3 ± 5.0 mm vs. 29.4 ± 4.7 mm, $p = 0.001$) and C (34.8 ± 6.3 mm vs. 23.1 ± 2.6 mm, $p < 0.001$).

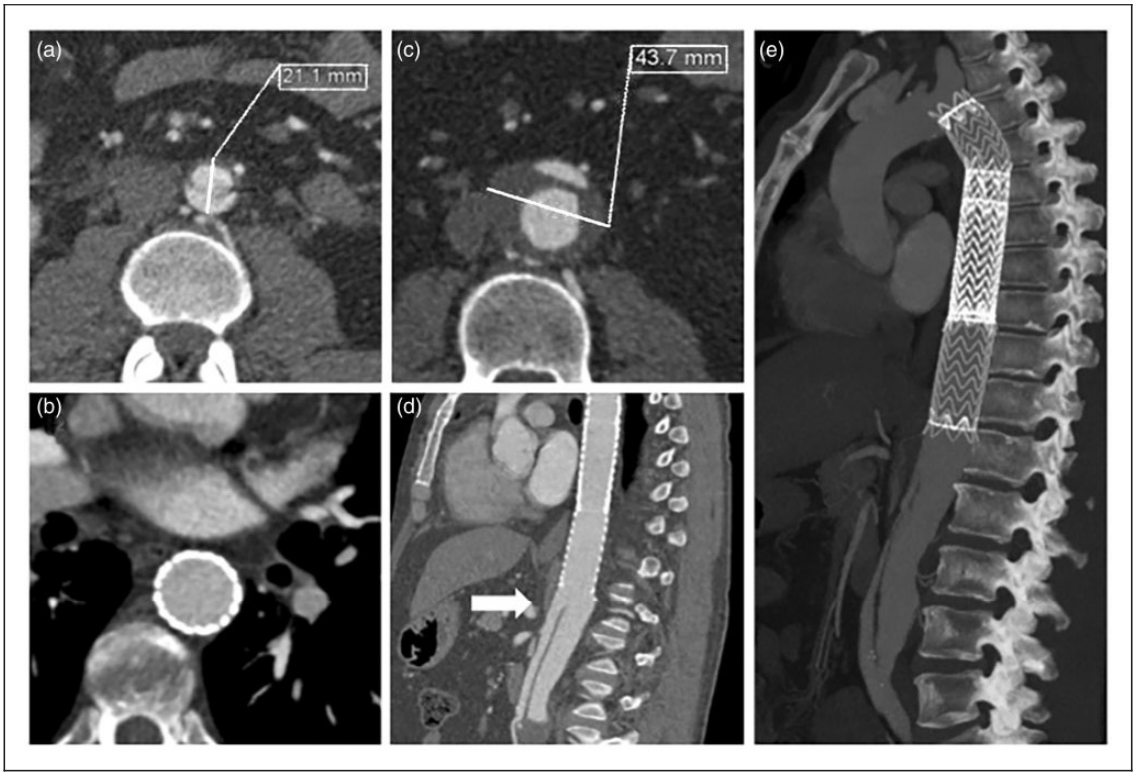

Figure 3. Infrarenal abdominal aortic expansion despite complete thoracic false lumen thrombosis in a 46-year-old male patient. (a) MPR of location C (infrarenal aorta) with baseline postinterventional infrarenal aortic diameter 1 day after thoracic endovascular repair at t1. (b) Complete remodeling of descending thoracic aorta along segment covered by endograft after a follow-up period of 57.3 months at t2. (c) MPR of location C demonstrating aortic expansion with diameter increase of 22.6 mm or 107.1%, respectively, at t2. (d) MPR showing formation of a wide reentry at distal end of endograft suggestive of distal intimal erosion maintaining false lumen perfusion (arrow). (e) Maximum intensity projection of aorta illustrating thoracic aortic remodeling and abdominal expansion. MPR: multiplanar reformation.

Neither at location B nor at location C there was a linear correlation between baseline false lumen diameters or baseline false lumen to total diameter ratios and diameter changes ($-0.3 < r > 0.3$ each).

Discussion

We found that almost 90% of our patients treated with TEVAR for aortic dissections have developed an abdominal aortic diameter increase of at least 5 mm or 15% during a follow-up period of at least 3 years despite technical success and despite complete false lumen thrombosis at the level of the EVG. Fifty percent of individuals have shown diameter increases of at least 9 mm along the abdominal aorta. In 11% of patients, critical abdominal diameters of at least 55 mm have been reached. The infrarenal abdominal aorta was at higher risk for expansion than the visceral abdominal aorta with a mean diameter increase per year of 1.7 mm. The annual abdominal aortic diameter increase was significantly higher when the diameter in the visceral or infrarenal segment was at least 30 mm at baseline. As abdominal baseline diameters were larger in patients treated with TEVAR in the setting of CD, the probability of abdominal aortic expansion gaining clinical significance may be higher for CD. Otherwise, the frequency of abdominal expansion was comparable between patients treated in the setting of AD and CD. Other strong predictors of abdominal aortic expansion aside from baseline diameter could not be identified.

After introduction of TEVAR as a feasible treatment option for aortic dissection, several publications have reported on morphology changes or remodeling of the aorta during the postinterventional period. However, most of these publications predominantly provide ambiguous data on the fate of the descending thoracic aorta. For example, Schoder et al.[6] have reported that persisting false lumen perfusion distal to the EVG resulted in diameter increases of the distal thoracic aorta in several patients (9.4 mm in the mean after 2 years). The patients in the TEVAR arm of the INSTEAD-XL trial have developed in the mean a

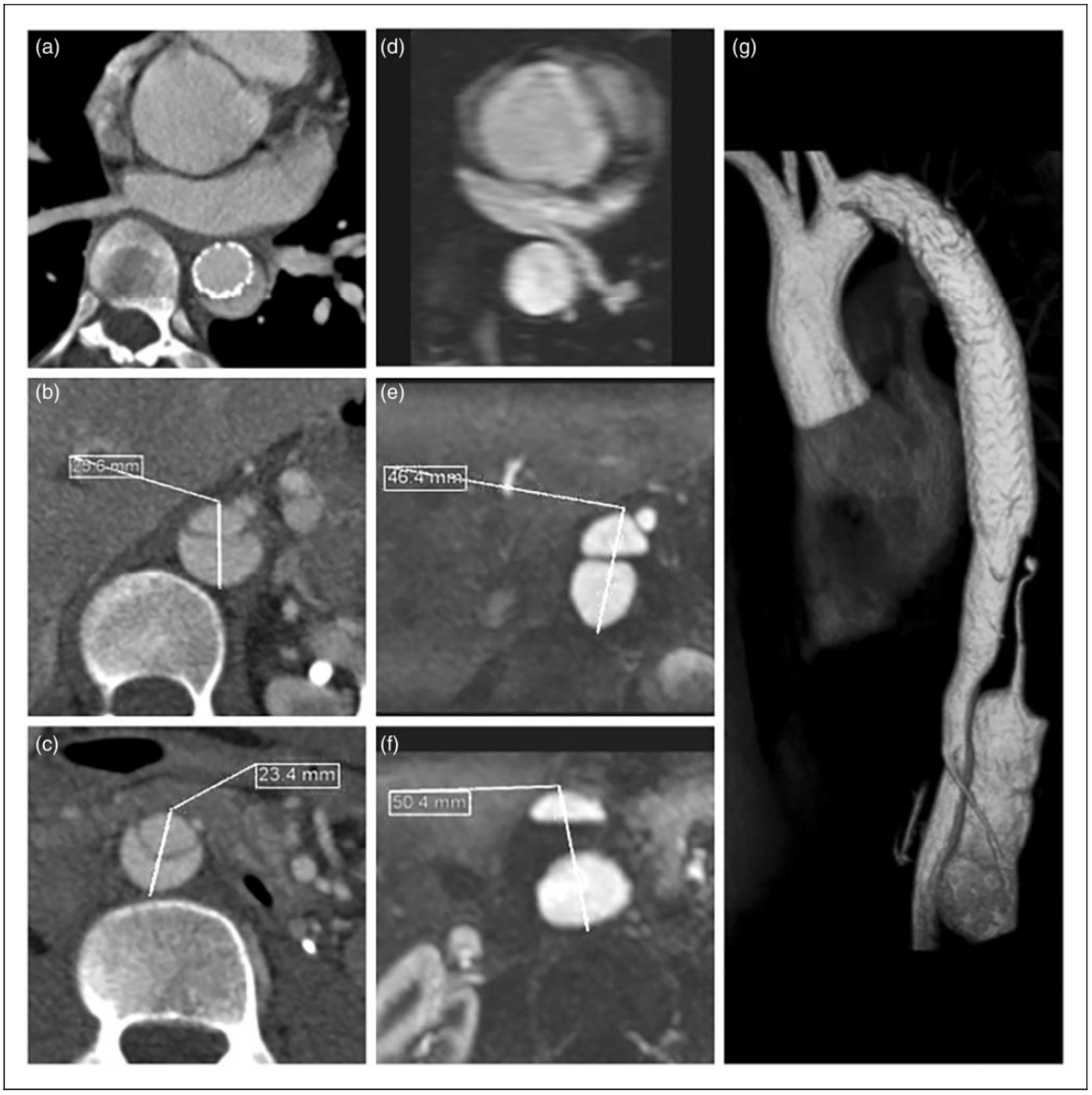

Figure 4. Visceral and infrarenal abdominal aortic expansion despite complete false lumen thrombosis along the endovascular graft in a 30-year-old female patient. (a) Persistent false lumen perfusion in thoracic aorta along endovascular graft 1 day after thoracic endovascular repair at t1. (b) MPR for baseline diameter assessment of location B (celiac trunk) at t1. (c) MPR for baseline diameter assessment of location C (infrarenal aorta) at t1. (d) Magnetic resonance angiography with MPR through descending thoracic aorta at a level comparable to (a) demonstrating complete remodeling along endovascular graft 66.9 months after implantation at t2. (e) MPR at location B showing aortic diameter increase of 20.8 mm or 81.3%, respectively, at t2. (f) MPR at location C showing aortic diameter increase of 27.0 mm or 115.4%, respectively, at t2. (g) Volume rendering of late follow-up magnetic resonance angiography illustrating thoracic aortic remodeling along endovascular graft and abdominal expansion. MPR: multiplanar reformation.

diameter decrease of the descending thoracic aorta at the hiatus level after 5 years follow-up, despite critical thoracic aortic expansion in 20.8% of TEVAR patients.[7]

Explicit data on the morphological evolution of the abdominal aorta and especially of the infrarenal aorta are scarce. Our data indicate that the infrarenal aorta is in general subject to a significantly greater diameter increase than the aorta at the origin of the visceral arteries. Schoder et al.[6] reported on a mean diameter increase of the aorta at the level of the celiac trunk of 7.6 mm averaged over all 23 subjects after 2 years follow-up. Park et al.[8] have published a series of 20 patients, in which diameter increases at the visceral and infrarenal level were 11.8% and 12.6% after approximately 2 years, respectively. Moreover, Sayer et al.[9] reported on three of eight patients demonstrating abdominal aortic expansion

Table 5. Pearson's correlation coefficients *r* for potential cofactors impacting on abdominal expansion.

Dependent variable	Independent variable	*r* for all patients	*r* for subgroup[a]
Absolute diameter increase	Age	−0.13	0.20
Absolute diameter increase	Treatment length	0.43	0.59
Absolute diameter increase	Baseline diameter	0.09	0.57
Absolute diameter increase	Follow-up length	−0.04	0.10
Baseline diameter	Annual diameter increase	0.07	0.66

aAfter exclusion of subjects with imaging features associated with endograft implantation favoring aortic expansion (patient #18) and with suspicion of hereditary connective tissue disorder (patients #1, #7, and #16).

of at least 5 mm in their cohort after 3 years follow-up.

The frequency of expansion appears to be considerably higher in our cohort. This may be attributed to differences concerning the follow-up length, which was notably greater in our study than in the studies mentioned earlier. In a preliminary analysis of our cohort after a median follow-up length of 27 months including all patients without the prerequisite of a minimum follow-up length, the expansion frequency was 22%.[10] Thus, the length of the follow-up period seems to be a relevant determinant of the abdominal aortic diameter increase, although a linear association between follow-up length and abdominal aortic diameter change could not be identified in our analysis.

Several publications have focused on true and false lumen volumetry before and after TEVAR for aortic dissection and concluded that a volume increase of the true lumen and a volume decrease of the false lumen are generally observable after successful TEVAR.[11–13] In the volumetric analysis by Qing et al.,[12] a total aortic volume increase was observed in 8 of 25 patients with CD, but no information was given on the site of volume increase or evidence of aneurysm formation or progression. A similar result is provided by Stanley et al.,[13] who have reported on a significant total aortic volume increase in 10 patients with a persistent false lumen perfusion after TEVAR (compared with 13 patients with postinterventional false lumen thrombosis). In a series published by Kim et al., a 10% reduction of the aortic volume distal to the EVG occurred over 5 years in the absence of endoleakages. However, aneurysmal expansion and potential concern for rupture were seen in the endoleakage group, since a total aortic volume increase of 25% was observable in two patients after 2 years.[11]

However, sole volumetry of total aortic true and false channels may lead to underdiagnosis of aortic expansion at distinct aortic locations because different locations are of different susceptibility to false lumen thrombosis, are obviously at different risk for expansion, and thus may expand independently from each other. In contrast to the studies mentioned earlier, e.g., Huptas et al.[14] and Andacheh et al.[15] provided separate volumetric data for the thoracic and abdominal aorta. Huptas et al.[14] found that shrinkage of the false lumen was generally limited to the thoracic aorta, while two patients have shown a false lumen expansion during short-term follow-up. Andacheh et al.[15] have reported that 46 patients with initial extension of the dissection into the infrarenal aorta had an increase of the mean aortic diameter and volume of 21% and 17% after 1 year, respectively.

In concordance to other studies, we ascertained that complete remodeling of the whole dissected aorta is rare. In our study population, there were only three cases of complete false lumen thrombosis or false lumen remission at the visceral abdominal segment (all in patients with AD) and none at the infrarenal abdominal segment. Moreover, complete false lumen thrombosis of the thoracic aorta along the EVG did not reliably induce false lumen thrombosis of the abdominal aorta and did not prevent expansion of the abdominal aorta. Interestingly, the only two cases in which we observed incomplete false lumen thrombosis along the EVG were those without evidence of abdominal aortic expansion. Thus, the amount of false lumen thrombosis along the EVG cannot be regarded as a sufficient indicator of prospective abdominal diameter development, although it has been suggested in the past that in patients with partial thrombosis of the false lumen the aneurysmal aorta continues to enlarge.[16] The observation that larger initial false lumen diameters may be predictive of aortic enlargement cannot be affirmed for the abdominal aorta according to our data.[17]

Our statistical analysis supposes that the baseline diameter of the expanding aortic location is an important determinant of the postinterventional abdominal diameter development. This is comparable to atherosclerotic nondissecting abdominal aneurysms, which tend to grow in a nonlinear fashion with acceleration of growth as the aneurysm enlarges.[18,19] The expansion rate was significantly higher in our patients with

baseline diameters greater than 30 mm at the visceral or infrarenal abdominal aortic segment. Baseline diameters were larger in the group of patients treated for CD than for AD. The probability of abdominal aortic expansion gaining clinical significance should be considered higher for CD than for AD. On this note, both patients in whom abdominal reintervention has been indicated in our cohort were treated in the setting of CD and had baseline diameters greater than 30 mm. As we did not perform serial measurements of aortic diameters during the available follow-up period, it is not possible to derive any information concerning the linearity of aortic expansion from our data. Thus, the computed yearly growth rates of 0.7 ± 1.3 mm and 1.7 ± 1.4 mm for the visceral and infrarenal aorta, respectively, should be interpreted with caution.

Concerning the potential impact of reentries on aortic remodeling, it is remarkable that both of our patients without abdominal aortic expansion did not have visible reentries distal to the EVG in postinterventional imaging. In contrast, among the patients with abdominal aortic expansion, a strong influence of the number of reentries on aortic diameters could not be derived. Thus, other individual factors that determine the fate of the abdominal aortic diameter and may help to prospectively identify patients at risk for abdominal expansion remain uncertain.

The magnitude of abdominal expansion was distributed heterogeneously in our study cohort. Statistical coefficients did not show substantial linear associations between potential expansion cofactors and parameters of expansion if all patients were considered. However, review of our source data suggested to reconsider the data after exclusion of four patients of which one had distal intimal erosion and three had probable hereditary connective tissue disorder. The patient with distal intimal erosion and one of the patients with probable hereditary connective tissue disorder had above-average abdominal aortic expansion after below-average follow-up length. The two other patients with probable hereditary connective tissue disorder did not have above-average expansion rates. Other patients with EVG-associated findings favoring false lumen perfusion and aortic expansion, such as distal intimal erosion, were not identified in our cohort. Thus, undesirable findings associated to EVG implantation and predisposing genetic background are supposed to alter the natural course of dissections after TEVAR compared with other individuals and may be surveyed differently.

As our data indicate that 50% of patients have developed abdominal aortic diameter increases of at least 9 mm after a median follow-up period of 7 years, we support the recommendation for lifelong follow-up imaging of the whole thoracic and abdominal aorta in these patients. To reduce radiation exposure, magnetic resonance angiography nowadays is a feasible alternative to CT for aortic imaging especially in young patients[20].

Aortic repair in aneurysm formation in aortic dissections is generally recommended if critical aortic diameters of 55 mm are reached.[21] A relevant proportion of 2 patients of our series (11.1%) exceeded this threshold diameter. The clinical significance of expansion in several other patients with small growth rates and small baseline abdominal aortic diameters may be equivocal, because diameters requiring intervention may not be surpassed. Abdominal reinterventions in patients with abdominal aortic expansion have to be indicated in due consideration of the individual aortic morphology and risk profile. Repair of the thoracoabdominal aorta in dissections is technically challenging, because revascularization of the visceral arteries is necessary, too. Treatment options include open repair, hybrid procedures and total endovascular techniques using custom-made fenestrated or branched EVG.[22,23] One of the major complications is paraplegia, whose risk grows with increasing aortic coverage.[24]

Limitations

There are several limitations that should be considered when interpreting our retrospective data. First, the sample size is small, because the gross majority of our patients did not reach the required minimum follow-up length or was lost during follow-up. The required minimum follow-up length was chosen, in due consideration of losing includable subjects to compile a study cohort providing data for an above-average follow-up period compared with other publications dealing with this topic. The small sample size, however, did not allow multivariate analysis of potential influence of certain clinical features, imaging findings and comorbidities on aortic expansion in depth. The heterogeneity of follow-up lengths ranging from 3 years to more than 11 years necessitates interpreting the magnitude and clinical significance of abdominal expansion in relation to the given follow-up length on an individual basis. Second, we did not analyze a control group of patients with aortic dissections treated conservatively. It might be possible that TEVAR actually is slowing down growth in the abdominal compartment compared to untreated patients. However, a recent publication on aortic expansion in uncomplicated type B dissections reported on comparable annual expansion rates of 1.7 mm during a median follow-up of 33 months without presenting data on the site of expansion.[25] Third, imaging was performed using a variety of scanners providing different slice thicknesses. Nevertheless, all but one CT scan and all MRI scans provided slice

thicknesses of 3 mm or less, which is generally deemed acceptable for performing MPR.[26]

Conclusion

Despite successful sealing of the primary entry tear and induction of thoracic false lumen thrombosis, chronic expansion of the abdominal aorta is a phenomenon frequently encountered during long-term follow-up after TEVAR for both AD and CD aortic dissections. Especially in patients with abdominal aortic diameters larger than 30 mm on first postinterventional imaging and status post TEVAR for chronic dissection, physicians should draw special attention to the long-term diameter development of the abdominal aorta. As individual factors promoting abdominal aortic expansion still have not been fully understood, lifelong imaging surveillance under due consideration of the abdominal aorta is still suggested to be necessary in all patients.

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References

1. Eggebrecht H, Nienaber CA, Neuhauser M, et al. Endovascular stent-graft placement in aortic dissection: a meta-analysis. *Eur Heart J* 2006; 27: 489–498.
2. White GH, Yu W, May J, et al. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. *J Endovasc Surg* 1997; 4: 152–168.
3. Faure EM, Canaud L, Agostini C, et al. Reintervention after thoracic endovascular aortic repair of complicated aortic dissection. *J Vasc Surg* 2014; 59: 327–333.
4. Thrumurthy SG, Karthikesalingam A, Patterson BO, et al. A systematic review of mid-term outcomes of thoracic endovascular repair (TEVAR) of chronic type B aortic dissection. *Eur J Vasc Endovasc Surg* 2011; 42: 632–647.
5. Torsello GB, Torsello GF, Osada N, et al. Midterm results from the TRAVIATA registry: treatment of thoracic aortic disease with the valiant stent graft. *J Endovasc Ther* 2010; 17: 137–150.
6. Schoder M, Czerny M, Cejna M, et al. Endovascular repair of acute type B aortic dissection: long-term follow-up of true and false lumen diameter changes. *Ann Thorac Surg* 2007; 83: 1059–1066.
7. Nienaber CA, Kische S, Rousseau H, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv* 2013; 6: 407–416.
8. Park KB, Do YS, Kim SS, et al. Endovascular treatment of acute complicated aortic dissection: long-term follow-up of clinical outcomes and CT findings. *J Vasc Interv Radiol* 2009; 20: 334–341.
9. Sayer D, Bratby M, Brooks M, et al. Aortic morphology following endovascular repair of acute and chronic type B aortic dissection: implications for management. *Eur J Vasc Endovasc Surg* 2008; 36: 522–529.
10. Weber TF, Hyhlik-Dürr A, Böckler D, et al. ECR 2010 book of abstracts. *Insights Imaging* 2010; 1: 233.
11. Kim KM, Donayre CE, Reynolds TS, et al. Aortic remodeling, volumetric analysis, and clinical outcomes of endoluminal exclusion of acute complicated type B thoracic aortic dissections. *J Vasc Surg* 2011; 54: 316–324. discussion 24–25.
12. Qing KX, Yiu WK and Cheng SW. A morphologic study of chronic type B aortic dissections and aneurysms after thoracic endovascular stent grafting. *J Vasc Surg* 2012; 55: 1268–1275. discussion 75–76.
13. Stanley GA, Murphy EH, Knowles M, et al. Volumetric analysis of type B aortic dissections treated with thoracic endovascular aortic repair. *J Vasc Surg* 2011; 54: 985–992. discussion 92.
14. Huptas S, Mehta RH, Kuhl H, et al. Aortic remodeling in type B aortic dissection: effects of endovascular stent-graft repair and medical treatment on true and false lumen volumes. *J Endovasc Ther* 2009; 16: 28–38.
15. Andacheh ID, Donayre C, Othman F, et al. Patient outcomes and thoracic aortic volume and morphologic changes following thoracic endovascular aortic repair in patients with complicated chronic type B aortic dissection. *J Vasc Surg* 2012; 56: 644–650. discussion 50.
16. Gaxotte V, Thony F, Rousseau H, et al. Midterm results of aortic diameter outcomes after thoracic stent-graft implantation for aortic dissection: a multicenter study. *J Endovasc Ther* 2006; 13: 127–138.
17. Song JM, Kim SD, Kim JH, et al. Long-term predictors of descending aorta aneurysmal change in patients with aortic dissection. *J Am Coll Cardiol* 2007; 50: 799–804.
18. Brady AR, Thompson SG, Fowkes FG, et al. Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. *Circulation* 2004; 110: 16–21.
19. Sherer EA, Bies RR, Clancy P, et al. Growth of screen-detected abdominal aortic aneurysms in men: a Bayesian analysis. *CPT Pharmacomet Syst Pharmacol* 2012; 1: e12.
20. Rengier F, Geisbüsch P, Vosshenrich R, et al. State-of-the-art aortic imaging: part I – fundamentals and perspectives of CT and MRI. *Vasa* 2013; 42: 395–412.
21. Akin I, Kische S, Rehders TC, et al. Thoracic endovascular stent-graft therapy in aortic dissection. *Curr Opin Cardiol* 2010; 25: 552–559.
22. Böckler D, Kotelis D, Geisbüsch P, et al. Hybrid procedures for thoracoabdominal aortic aneurysms and chronic aortic dissections – a single center experience in 28 patients. *J Vasc Surg* 2008; 47: 724–732.
23. Verhoeven EL, Paraskevas KI, Oikonomou K, et al. Fenestrated and branched stent-grafts to treat post-dissection chronic aortic aneurysms after initial treatment in the acute setting. *J Endovasc Ther* 2012; 19: 343–349.

24. Kotelis D, Geisbüsch P, von Tengg-Kobligk H, et al. [Paraplegia after endovascular repair of the thoracic and thoracoabdominal aorta]. *Zentralbl Chir* 2008; 133: 338–343.
25. Kotelis D, Grebe G, Kraus P, et al. Morphologic predictors of aortic expansion in chronic type B aortic dissection. *Vascular* 2016; 24: 187–193.
26. von Tengg-Kobligk H, Weber TF, Rengier F, et al. Image postprocessing of aortic CTA and MRA. *Radiologe* 2007; 47: 1003–1011.