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How Does the Ascending Aorta Geometry Change When It Dissects?

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Objectives The purpose of this study is to delineate changes in aortic geometry and diameter due to dissection.

Background Aortic diameter is the major criterion for elective ascending aortic replacement for dilated ascending aortas to prevent aortic dissection. However, recommendations are made on the basis of clinical experience and observation of diameters of previously dissected aortas.

Methods Six tertiary centers on 2 continents reviewed their acute aortic dissection type A databases, which contained 1,821 patients. Included were all non-Marfan patients with nonbicuspid aortic valves who had undergone computed tomography angiography <2 years before and within 12 h after aortic dissection onset. Aortic geometry before and after dissection onset were compared.

Results Altogether, 63 patients were included (27 spontaneous and 36 retrograde dissections, median age 68 [57; 77] years; 54% were men). In all but 1 patient, maximum ascending aortic diameter was <55 mm before aortic dissection onset. The largest increase in diameter and volume induced by the dissection were observed in the ascending aorta (40.1 [36.6; 45.3] mm vs. 52.9 [46.1; 58.6] mm, +12.8 mm; $p < 0.001$; 124.0 [90.8; 162.5] cm^3 vs. 171.0 [147.0; 197.0] cm^3, +47 cm^3; $p < 0.001$). Mean aortic arch diameter increased from 39.8 (30.5; 42.6) mm to 46.4 (42.0; 51.6) mm (+6.6 mm; $p < 0.001$) and descending thoracic aorta diameter from 31.2 (27.0; 33.3) mm to 34.9 (30.9; 39.5) mm (+3.7 mm; $p < 0.001$). Changes in thoracic aorta geometry were similar for spontaneous and retrograde etiology.

Conclusions Geometry of the thoracic aorta is affected by aortic dissection, leading to an increase in diameter that is most pronounced in the ascending aorta. Both spontaneous and retrograde dissection result in similar aortic geometry changes. (J Am Coll Cardiol 2014;63:1311–9) © 2014 by the American College of Cardiology Foundation

In the current guidelines, aortic diameter is a major criterion for elective ascending aortic replacement for dilated ascending aortas for prevention of acute aortic dissection type A (AADA) (1). However, recommendations are based on data of aortic diameters obtained from previously dissected aortas (2,3), as aortic dimensions prior to dissection onset are commonly unknown. Given the thinning of the outer aortic wall with separation of the media by the dissection process, it is conceivable that the dissection process itself leads to changes of the aortic diameter and geometry. Up-to-date data quantifying the extent of these acute changes is not

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Abbreviations and Acronyms

AADA = acute aortic dissection type A

CTA = computed tomography angiography

STJ = sinotubular junction

yet available, as the vast majority of patients has not undergone cross-sectional imaging prior to dissection onset. Furthermore, the few experimental studies on AADA merely mimic the complexity of acute dissection (4,5).

We hypothesized that the dissection itself leads to acute changes in aortic geometry and diameter. The aim of this study was to assess the extent of changes in aortic geometry induced by the dissection process by means of computed tomography angiography (CTA) obtained before and after AADA in a retrospective, multicenter fashion.

Methods

Study population. Six tertiary centers in the United States and Europe reviewed their AADA databases, which contained, overall, 1,821 patients operated on between January 1, 1994, and March 1, 2013 for patients with available CTA studies performed within 2 years prior to dissection onset and within 12 h after ascending aortic dissection onset. Included in this analysis were non-Marfan patients with nonbicuspid aortic valves with AADA as a primary aortic event (spontaneous dissection) and AADA secondary to type B dissection (retrograde dissection). This retrospective study was approved by all institutional review committees. The need for informed consent was waived.

Image analysis. DICOM (Digital Imaging and Communications in Medicine standard) data of eligible patients were transferred to a core laboratory for further analysis in an anonymized fashion. One observer blinded to patient-identifying information performed the image analysis, using Aquarius Intuition (TeraRecon, Inc., Foster City, California). A centerline was created from the aortic valve annulus to the most distal available portion of the descending thoracic aorta. The thoracic aorta was divided into 3 segments by appropriate planes perpendicular to the centerline (Fig. 1A): 1) the ascending aorta, beginning at the plane corresponding to the nadirs of all 3 aortic cusps and extending to the plane immediately proximal to the origin of the brachiocephalic artery; 2) the aortic arch, beginning immediately proximal to the origin of the brachiocephalic artery and extending to a plane immediately distal to the origin of the left subclavian artery; and 3) the proximal descending thoracic aorta, beginning at a plane immediately distal to the origin of the left subclavian artery and extending to a plane at the transverse level of the left main coronary artery orifice. Length, tortuosity, and volume were assessed in each aortic segment. Length was defined as the centerline distance between the previously-defined planes. Tortuosity (T) was calculated as the ratio of the incremental curve length (L_c) of the centerline to the linear distance (d) between its 2 endpoints, as assessed by an electronic caliper (Fig. 1B). Volumetric measurements were obtained in a semiautomated fashion preceded by manual aortic segmentation of the aortic wall's outer surface in cross sections. Once the aorta was segmented, the software automatically constructed a 3-dimensional model of its shape, which was inspected carefully and corrected manually as needed. From this model, the aortic volume was computed automatically. The total volume of each segment included the true and false lumen and was computed in reference to the aortic wall's outer surface.

Planimetric measurements yielding luminal area and maximum and minimum diameter by semiautomated polygonal border tracing were obtained by contour tracking at the previously-mentioned planes and at the following additional planes perpendicular to the centerline (Fig. 2): 1) the sinus of Valsalva—defined as the plane depicting the largest sinus dimension; 2) the sinotubular junction (STJ); 3) the mid-ascending aorta—with equal distance to the STJ and brachiocephalic artery orifice; and 4) the proximal descending thoracic aorta—5 cm distal to the left subclavian artery orifice. All reported diameters are maximum diameters. Minimum diameters were obtained for aortic ellipticity index calculation.

The ellipticity index, defined as maximum diameter divided by minimal diameter, was calculated for each plane. Circularity was defined as an ellipticity index $\leq$1.1.

For the analysis of geometry changes due to AADA onset, only newly dissected segments were considered.

Statistical analysis. Continuous data are reported as median (first quartile; third quartile), and categorical variables are reported as counts and percentages. For comparison of continuous variables, the Student t test was applied when equal distribution was present as tested by the Kolmogorov-Smirnov test. For unequally distributed variables, the Mann-Whitney rank sum test was employed. Paired t test and paired Mann-Whitney rank sum test were applied to compare pre- and post-dissection values. Categorical variables were compared using the chi-square test. In cases of small group sizes ($n < 5$), the Fisher exact test was employed. The association between ascending aortic diameter change and continuous variables was assessed by the Pearson correlation coefficient and linear regression analysis. All statistical calculations were performed using SigmaPlot (version 12, Systat Software, San Jose, California).

Results

Of 1,821 patients, we identified 63 patients with available CTA data obtained within 2 years prior to dissection onset and immediately after dissection onset before surgery. Indications for the CTA studies prior to AADA onset are listed in Table 1. The median time interval between both CTs was 3.8 (0.7; 8.5) months (52 patients with <1 year). Complete demographic data and cardiovascular risk profiles were available in 1,344 of 1,821 patients (73.8%). Compared with these, patients with pre-dissection imaging were older (median: 68 years

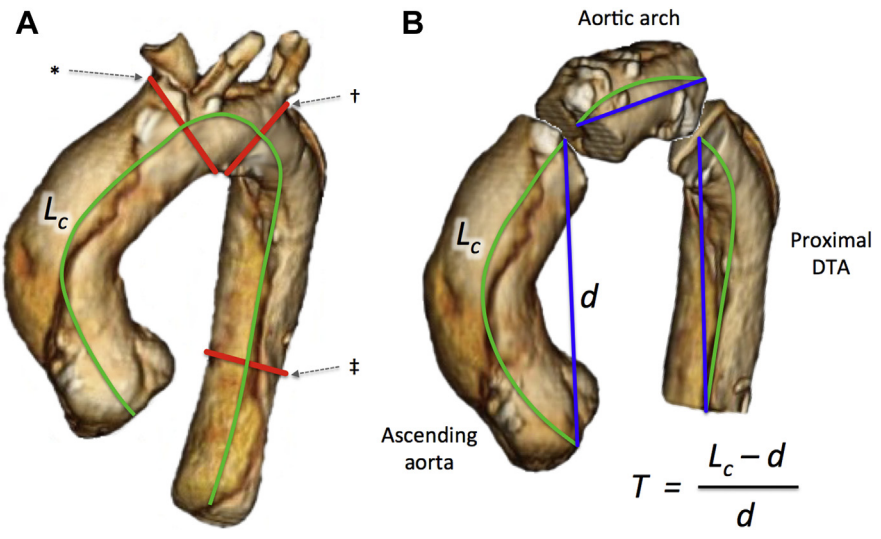

Figure 1 Thoracic Aorta Segmentation

(A) Illustration of the pre-defined segmentation planes at the origin of the brachiocephalic artery **(*)**, at the origin of the left subclavian artery **(†)**, and at the level of the left main coronary artery orifice **(‡)**. L_c indicates the centerline. **(B)** Geometric assessment of thoracic aortic segments. Tortuosity (T) is expressed as a ratio of the incremental curve length (L_c) to the linear distance (d) between its 2 endpoints. Volumetric measurements included the true and false lumen. DTA = descending thoracic aorta.

[57; 77] years vs. 62 [51; 71] years; $p < 0.001$) and had higher rates of hypertension (91% vs. 76%; $p = 0.011$), dyslipidemia (51% vs. 29%; $p < 0.001$), and previous cardiac surgery (19% vs. 10%; $p = 0.023$) than did patients without pre-dissection imaging (Table 2).

In patients with pre-dissection imaging, AADA was of spontaneous etiology in 27 patients (median age: 69 [60; 78] years) and of retrograde etiology in 36 patients (median age: 68 [54; 76] years). Detailed etiology of retrograde AADA is presented in Table 3. Except for patient sex, both groups

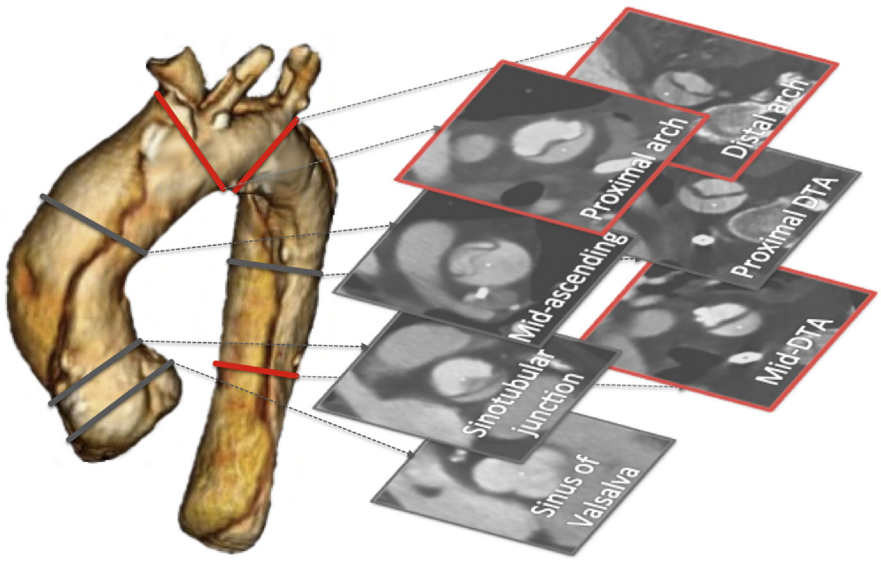

Figure 2 Predefined Segmentation Planes

Red planes divided the thoracic aorta into the 3 segments of interest: the ascending aorta, aortic arch, and proximal descending thoracic aorta. **Gray planes** indicate additional segmentation planes for further analysis. Aortic lumen area, maximum and minimal diameters, aortic ellipticity, and area-derived diameter were analyzed at all planes. DTA = descending thoracic aorta.

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Table 1 Indications for CTA Studies Prior to AADA Onset

Spontaneous AADA (n = 27)	
Thoracic aortic dilation follow-up	23 (36.5)
Cancer staging	3 (4.8)
Evaluation of anomalous coronary arteries	1 (1.6)
Retrograde AADA (n = 36)	
Type B aortic dissection medical treatment follow-up	14 (22.2)
Thoracic endovascular aortic repair follow-up	22 (34.9)

n = 63. Values are n (%).
AADA = acute aortic dissection type A; CTA = computed tomography angiography.

presented similar cardiovascular risk factor profiles and demographics. A higher prevalence of male sex was observed in retrograde AADA than in spontaneous AADA (79% vs. 39%; p = 0.013). Clinical and demographic characteristics are summarized in Table 4. Ascending aorta was newly dissected in all patients, aortic arch in 55, and proximal descending thoracic aorta in 19 patients.

Aortic diameters prior to AADA onset. Thoracic aortic diameters prior to AADA onset are presented in Table 5 for all 63 patients and stratified by AADA etiology. Prior to AADA, the largest aortic dimensions were observed at the level of the mid-ascending aorta with a median diameter of 40.1 (36.6; 45.3) mm. All but 1 patient exhibited an ascending aortic diameter of <55 mm. There was no significant difference in baseline aortic geometry in regard to the ascending aorta and aortic arch between patients with spontaneous and retrograde AADA. In patients with retrograde AADA, descending thoracic aorta dimensions were significantly larger than in spontaneous AADA patients, given the presence of the initial type B dissection prior to onset of AADA.

Table 2 Demographics and Cardiovascular Risk Factors in Patients With and Without Pre-Dissection Aortic Imaging

	Pre-Dissection Imaging (n = 63)	No Pre-Dissection Imaging (n = 1,344)	p Value
Demographics			
Age, yrs	68.0 (56.5;77.0)	62.0 (51.1;71.3)	<0.001
Male	34 (54.0)	869 (64.7)	0.111
Race			
Caucasian	54 (85.7)	1,110 (82.6)	0.556
Black	5 (7.9)	143 (10.6)	0.636
Asian	2 (3.2)	10 (0.7)	0.177
Hispanic	1 (1.6)	11 (0.8)	0.958
Cardiovascular risk factors			
Hypertension	57 (90.5)	1,018 (75.7)	0.011
Dyslipidemia	32 (50.8)	385 (28.6)	<0.001
Diabetes	10 (15.9)	127 (9.4)	0.143
COPD	10 (15.9)	134 (10.0)	0.194
Renal failure	7 (11.1)	75 (5.6)	0.120
Current smoking	5 (7.9)	95 (7.1)	0.991
CAD	15 (23.8)	190 (14)	0.052
PAD	5 (7.9)	64 (4.8)	0.409
Prior cardiac surgery	12 (19.0)	127 (9.5)	0.023

Values are median (first quartile; third quartile) or n (%).
CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; PAD = peripheral artery disease.

Aortic dimensions change due to dissection. Thoracic aortic dimensions of primarily nondissected segments were generally larger after AADA onset. The most pronounced increase in dimensions was observed in the ascending aorta (Figs. 3 and 4).

ASCENDING AORTA. Median mid-ascending aorta area and maximum diameter increased from 1,159.0 (988.5; 1,493.0) mm^2 to 1,870.0 (1,511.3; 2,435.5) mm^2 (+711 mm^2; +61%; p < 0.001) and from 40.1 (36.6; 45.3) mm to 52.9 (46.1; 58.6) mm (+12.8 mm; +32%; p < 0.001), respectively (Figs. 3 and 5). After onset of AADA, ascending aortic diameters ≥55 and ≥50 mm were observed in 44% (28 of 63) and in 60% (38 of 63) of patients, respectively. A similar but less pronounced increase in dimensions was observed for the level of the STJ (+6.9 mm; p < 0.001). In contrast, sinus of Valsalva diameter did not increase significantly. The aorta's ellipticity increased significantly (median: 1.09 [1.07; 1.12] vs. 1.14 [1.09; 1.16]; p < 0.001), but only at the mid-ascending level. The ascending aortic volume increased (124.0 [90.8; 162.5) cm^3 vs. 171.0 [147.0; 197.0] cm^3; p < 0.001), as did tortuosity (0.18 [0.12; 0.26] vs. 0.22 [0.16; 0.37]; p = 0.044) (Table 6).

Spontaneous and retrograde AADA led to similar aortic diameter increases at the level of the STJ (+5.8 mm vs. +6.7 mm; p = 0.881) and mid-ascending aorta (+10.7 mm vs. +13.6 mm; p = 0.991). Mid-ascending aortic diameter increase stratified to spontaneous and retrograde AADA is depicted in Figure 6. There was no significant correlation between patients' age, weight, height, body mass index, body surface area, and ascending aortic diameter change. There was no significant difference in ascending aortic diameter changes among patients with and without cardiovascular risk factors.

AORTIC ARCH. Proximal and distal aortic arch diameters increased on average by +6.6 mm (39.8 [30.5; 42.6] mm vs. 46.4 [42.0; 51.6] mm; p < 0.001) and +4.2 mm (31.1 [27.9; 33.8] mm vs. 35.3 [31.5; 40.8] mm; p = 0.025) (Figs. 3 and 4). Neither ellipticity nor tortuosity changed significantly, whereas median aortic arch volume increased from 41.1 (36.5; 58.5) cm^3 to 48.0 (38.0; 63.0) cm^3 (p = 0.042) (Table 6). Increase in diameters of the proximal and distal aortic arch did not differ between patients with retrograde and spontaneous AADA (p = 0.251 and p = 0.452, respectively).

PROXIMAL DESCENDING THORACIC AORTA. In patients with spontaneous AADA, descending thoracic aorta diameter increased by +3.7 mm (31.2 [27.0; 33.3] mm vs. 34.9 [30.9; 39.5] mm; p = 0.034) and +5.0 mm (27.9 [26.0; 30.2] mm vs. 32.9 [29.5; 41.4] mm; p = 0.013) at the proximal and mid-descending thoracic aorta, respectively (Fig. 4). Changes

Table 3 Mechanisms of Retrograde Aortic Dissection Type A

During medical treatment of type B dissection	14 (38.9)
Post-TEVAR for type B dissection	14 (38.9)
Post-TEVAR for aortic arch or descending thoracic aortic aneurysm	8 (22.2)

n = 63. Values are n (%).
TEVAR = thoracic endovascular aortic repair.

in volume, ellipticity, and tortuosity did not reach statistical significance (Table 6).

Discussion

Ascending aorta dilation is a well-described risk factor for the development of acute aortic dissection (6). Replacement of the ascending aorta is currently the recommended preventive procedure to avoid aortic dissection and is indicated whenever the ascending aorta diameter reaches 55 mm in patients with degenerative thoracic aneurysm, or 40 to 50 mm in patients with genetically-mediated connective tissue disease, according to the current 2010 American College of Cardiology Foundation/American Heart Association guidelines (1). Although applied to patients with nondissected dilated ascending aortas, these recommendations are made on the basis of studies in which the diameters of already dissected aortas were evaluated. We hypothesized that the geometry of a dissected ascending aorta differs from the geometry of the same nondissected aorta.

The present study is the first to address the aortic geometry changes after acute onset of aortic dissection type A in humans by means of CT. The findings of this study can be summarized as follows:

1. Acute aortic dissection leads to significant diameter, ellipticity, tortuosity, and volume alterations of the originally nondissected ascending aorta. The induced average increase in the mid-ascending aortic diameter is 13 ± 7 mm (+32%).
2. Dimensions of the sinus of Valsalva do not change significantly.
3. Spontaneous and retrograde AADA mechanisms result in similar changes in aortic geometry.
4. Induced increase in ascending aortic diameter is not associated with patients' age, height, and weight.

Clinical characteristics. Older age among patients with pre-dissection imaging might be due to the fact that older patients are more likely to undergo CT. In accordance with older age, hypertension and dyslipidemia were more frequently observed in patients with pre-dissection aortic imaging. Additionally, patients with pre-dissection imaging had more frequent history of cardiac surgery (19%), which was slightly more than the 14% (71 of 507) reported by the IRAD (International Registry of Acute Aortic Dissection) investigators (7), 15% (49 of 330) reported by Estrera et al. (8), and 16% (31 of 190) demonstrated by the University of Florida group (9). The observed difference might be due to

Table 4 Demographics and Cardiovascular Risk Factors in Patients With Pre-Dissection Aortic Imaging

	Spontaneous AADA (n = 27)	Retrograde AADA (n = 36)	p Value
Demographics			
Age, yrs	69.0 (59.5;77.5)	68.0 (54.0;75.5)	0.445
Male	9 (33.3)	25 (69.4)	0.006
Weight, kg	77.5 (67.3;95.3)	92.5 (75.5;108.8)	0.185
Height, cm	168.0 (162.8;173.5)	175.5 (165.5;173.5)	0.121
BMI, kg/m^2	28.8 (25.2;32.2)	30.4 (24.9;35.1)	0.519
BSA, m^2	1.8 (1.8;2.1)	2.1 (1.9;2.2)	0.138
Race			
Caucasian	24 (88.9)	30 (83.3)	0.450
Black	1 (3.7)	4 (11.1)	0.388
Asian	1 (3.7)	1 (2.8)	1.000
Hispanic	0	1 (2.8)	1.000
Cardiovascular risk factors			
Hypertension	23 (85.2)	34 (94.4)	0.388
Dyslipidemia	13 (48.1)	19 (52.8)	0.913
Diabetes	5 (18.5)	5 (13.9)	0.733
COPD	5 (18.5)	5 (13.9)	0.733
Renal failure	2 (7.4)	5 (13.9)	0.689
Current smoking	2 (7.4)	3 (8.3)	1.000
CAD	10 (37.0)	5 (13.9)	0.066
PAD	2 (7.4)	3 (8.3)	1.000
Prior cardiac surgery	3 (11.1)	9 (25.0)	0.287

Values are median (first quartile; third quartile) or n (%).
BMI = body mass index; BSA = body surface area; other abbreviations as in Tables 1 and 2.

Table 5 Thoracic Aorta Diameters Prior to Aortic Dissection Type A Onset

	All (n = 63)	Spontaneous AADA (n = 27)	Retrograde AADA (n = 36)	p Value
Sinus of Valsalva	40.4 (37.4;43.0)	40.5 (36.7;46.2)	40.3 (37.9;42.1)	0.522
Sinotubular junction	37.2 (34.8;40.8)	38.3 (34.9;42.2)	36.7 (34.8;39.3)	0.309
Mid-ascending aorta	40.1 (36.6;45.3)	43.1 (36.8;47.9)	39.9 (36.1;43.9)	0.117
Proximal aortic arch	39.8 (36.1;42.6)	38.8 (36.8;43.9)	40.9 (35.9;42.0)	0.838
Distal aortic arch	33.8 (30.5;40.6)	31.1 (27.9;33.8)	35.6 (32.8;42.0)	0.002
Proximal descending thoracic aorta	36.0 (32.0;43.0)	32.4 (28.0;34.3)	41.4 (36.3;48.1)	<0.001
Mid-descending thoracic aorta	32.5 (28.6;40.0)	28.3 (26.0;30.2)	39.1 (34.2;46.3)	<0.001

Values are median (first quartile; third quartile) and are given in millimeters.
Abbreviations as in Tables 1 and 2.

the fact that patients who undergo cardiac surgery are more likely to have imaging scans of the nondissected aorta than patients previously not operated. On the other hand, the overall incidence of previous cardiac surgery in patients without pre-dissection imaging was lower than in the previously-mentioned series.

Aortic geometric alterations due to dissection. Several working groups have addressed the conflict of the ascending aortic size with regard to prophylactic surgery and optimal timing for ascending aortic replacement (10,11). However, few have considered that the rapid separation within the aortic media may cause acute weakness of the aortic wall, which should theoretically trigger an immediate aortic diameter increase. In an animal model of endovascular-created aortic dissection, Okuno et al. (4) observed a tendency toward increased aortic diameter after dissection onset (from 10.9 ± 2.9 mm to 12.9 ± 3.8 mm). Assuming that the circumference of the true lumen is similar to the pre-dissection dimensions, Neri et al. (12) retrospectively measured the perimeter of the intimal flap in AADA patients and calculated the virtual pre-event aortic diameter. Unfortunately, the calculated pre-dissection diameter was not confirmed by any imaging modality. However, they demonstrated that AADA occurs rarely in the setting of true ascending aortic aneurysms.

To date, no investigation has quantified the acute changes in aortic geometry due to aortic dissection in humans. To address this issue, we assessed thoracic aortic anatomic parameters by CTA coincidentally performed before aortic dissection and immediately after aortic dissection onset. Most importantly, we observed the greatest increase in diameter, cross-sectional area, tortuosity, ellipticity, and volume in the ascending aorta. This significant increase in diameter warrants careful interpretation of published data on assumed aortic diameters at the time of aortic dissection onset, as all studies so far have assessed already dissected aortas. In a study by Parish et al. (10) on 177 patients with AADA, the mean ascending diameter in non-Marfan, tricuspid aortic valve patients was 53 ± 10 mm after dissection onset. In IRAD, the mean diameter was 53 mm in 591 AADA patients, ranging from 20 to 100 mm (11), which is similar to our post-dissection diameter of 52.9

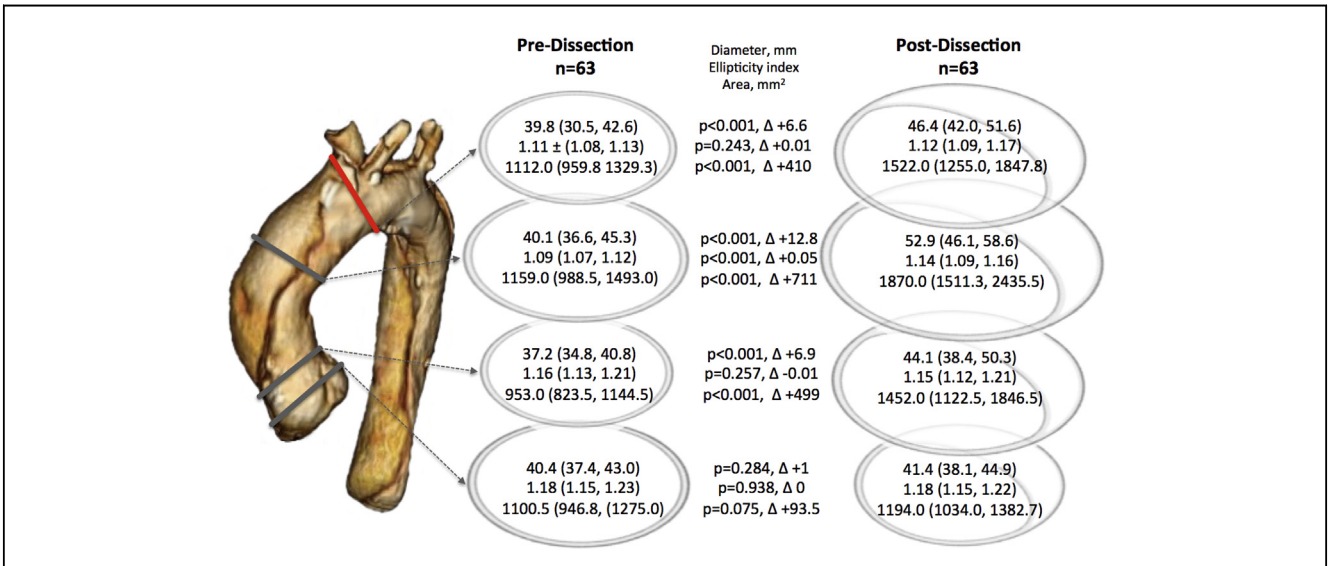

Figure 3 Ascending Aortic Dimension Changes Due to Dissection

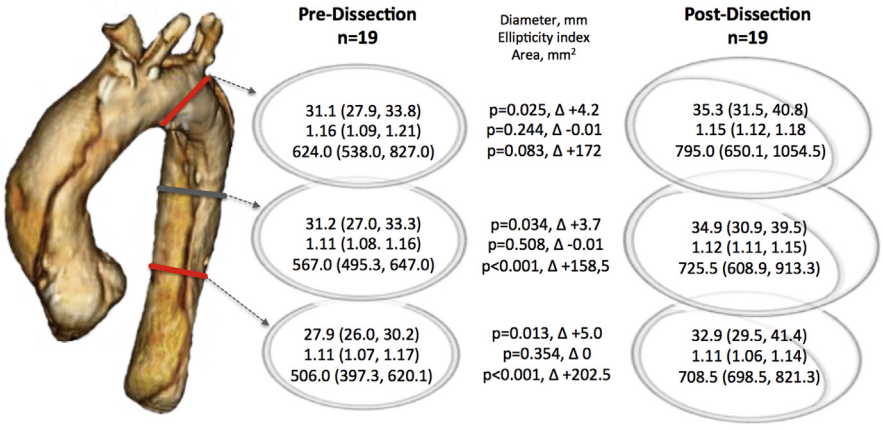

Figure 4 Descending Aortic Dimension Changes Due to Dissection

± 10.4 mm. Based on our findings, it can be assumed that the average ascending aortic diameter prior to dissection is lower, 40.1 ± 5.9 mm in our study. We observed only 1 patient with a pre-dissection ascending aorta diameter >55 mm. However, this is a retrospective study and the real prevalence of ascending aneurysm prior to dissection onset remains unknown. In both studies (10,11), the observed post-dissection diameter was <50 mm in about one-half of the AADA patients. This concurs well with our findings, as we found ascending aortic diameters of ≥50 mm in 60% of patients after dissection onsets.

Interestingly, aortic geometry did not change in the entire thoracic aorta, sparing the sinus of Valsalva. This might be due to the close anatomic relation to the aortic annulus,

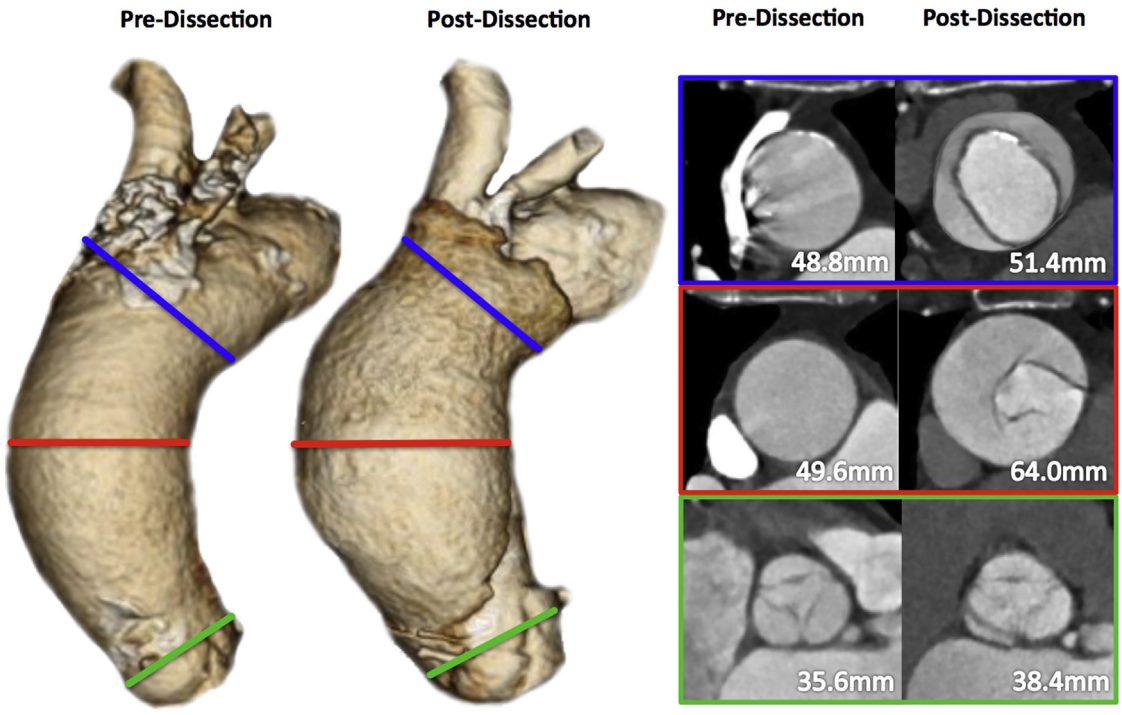

Figure 5 Pre- and Post-Dissection CT Angiography of an 85-Year-Old Male Patient

Volume-rendered images of the ascending aorta and cross-sectional images at sinus of Valsalva **(green),** mid-ascending aorta **(red),** and area immediately proximal to the origin of the brachiocephalic artery **(blue)** with maximum diameters are provided. CT = computed tomography.

Table 6 Aortic Geometry Change Due to Dissection in Primarily Nondissected Segments

	Pre-Dissection	Post-Dissection	Δ	p Value
Ascending aorta (n = 63)				
Centerline length, mm	99.6 (88.5;112.3)	105.0 (94.9;114.3)	+5.4	0.090
Tortuosity	0.18 (0.12;0.26)	0.22 (0.16;0.37)	+0.04	0.044
Volume, cm^3	124.0 (90.8;162.5)	171.0 (147.0;197.0)	+47	<0.001
Aortic arch (n = 55)				
Centerline length, mm	39.8 (33.9;47.9)	41.0 (36.6;45.7)	+1.2	0.677
Tortuosity	0.11 (0.02;0.18)	0.11 (0.02;0.23)	0	0.831
Volume, cm^3	41.1 (36.5;58.5)	48.0 (38.0;63.0)	+6.9	0.042
Proximal descending thoracic aorta (n = 19)				
Centerline length, mm	123.2 (105.3;140.5)	125.8 (106.2;142.0)	+2.5	0.912
Tortuosity	0.08 (0.01;0.15)	0.07 (0.01;0.13)	−0.01	0.253
Volume, cm^3	149.3 (132.1;165.4)	162.1 (139.2;177.1)	+12.8	0.877

Values are median (first quartile; third quartile). Δ indicates pre-dissection – post-dissection change.

which prevents progression of dissection by acting as a natural anatomic barrier.

Spontaneous versus retrograde dissection. The present study reports similar aortic geometry changes in patients with spontaneous and retrograde AADA. Comparison of demographics and cardiovascular risk factors between both groups revealed no relevant differences other than unequal sex distribution. The percentage of women was low (21%) among the retrograde AADA patients. As most of them had AADA after primary aortic dissection type B, the sex distribution we observed concurs with the lower incidence of type B aortic dissection in women (13–16). Retrograde and spontaneous AADA represent 2 distinct pathophysiological entities with regard to entry localization, direction of dissection extension, and, usually, different blood flow orientation in the false lumen.

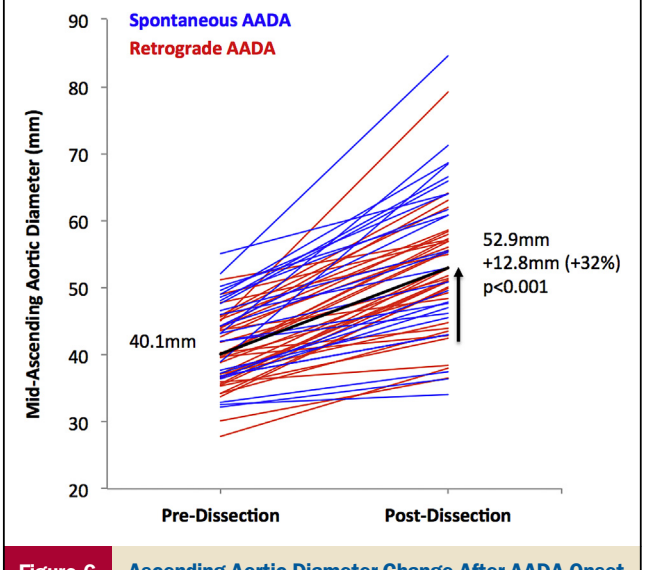

Figure 6 Ascending Aortic Diameter Change After AADA Onset

Blue indicates patients with spontaneous AADA; **red,** patients with retrograde AADA; and **black**, the overall median diameter increase. AADA = acute aortic dissection type A.

Whereas hypertension, aortic dilation, wall abnormalities, and the dynamics of aortic root are known risk factors for spontaneous AADA onset (17,18), retrograde propagation of type B dissection tends to depend on primary entry tear localization, which may cause different means of propagation (19). These mechanisms do not appear to significantly influence the change in geometry of primarily nondissected aortic segments. Most likely, changes of aortic geometry are rather caused by the separation between the adventitia and media, which resembles the final pathway of both spontaneous and retrograde AADA, finally destabilizing the aorta's 3-tiered architecture. However, our results must be interpreted with caution, because our subgroups are too small to draw definitive conclusions from.

Aortic elasticity. There is growing evidence on the elastic properties of the aortic wall and the risk of aortic dissection. Abnormal aortic elasticity (e.g., stiffness) is a known risk factor for aortic dilation in patients with Marfan syndrome (20). Other investigators have suggested that the presence of bicuspid aortic valve is associated with a stiffer sinus of Valsalva and abnormal ascending aortic distensibility (21,22). Furthermore, there is a strong relation between aging and the stiffening of the proximal aorta (23,24). However, static CTA does not allow for assessment of aortic elastic properties.

Study limitations. This is a retrospective study on patients who are being followed in tertiary centers. As the majority of patients with ascending diameter >55 mm undergo elective ascending replacement, the incidence of ascending aortic aneurysm prior to dissection onset cannot be elucidated from our data. Furthermore, older age, higher incidence of hypertension, dyslipidemia, and history of cardiac surgery in patients with versus without pre-dissection imaging should be considered when extrapolating these results to the global AADA population.

Conclusions

Aortic dissection changes the geometry of affected aortic segments with different patterns. The greatest diametric increase is observed in the ascending aorta, whereas sinus of

Valsalva dissection does not lead to major changes in dimensions. Both spontaneous and retrograde AADA result in similar aortic geometry changes.

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