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Acute retrograde type A aortic dissection: morphologic analysis and clinical implications

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Abstract

Background: This study compares the morphology and outcomes of acute retrograde type A dissections (RTADs) with acute antegrade type A dissections (ATADs), and acute type B dissections.

Materials and methods: From 2000 to 2016, there were 12 acute RTADs, 96 ATADs, and 92 type B dissections with available imaging. Dissections were characterized using computerized tomography angiography images. We examined clinical features, tear characteristics, and various morphologic measurements.

Results: Compared with acute type B dissections, RTAD primary tears were more common in the distal arch (75% *versus* 43%, $P = 0.04$), and the false-to-true lumen contrast intensity ratio at the mid-descending thoracic aorta was lower (0.46 *versus* 0.71, $P = 0.020$). RTAD had less false lumen decompression because there were fewer aortic branch vessels distal to the subclavian that were perfused through the false lumen (0.40 *versus* 2.19, $P < 0.001$). Compared with ATAD, RTAD had less root involvement where root true-to-total lumen area ratio was higher (0.88 *versus* 0.76, $P = 0.081$). Furthermore, RTAD had a lower false-to-true lumen contrast intensity ratio at the root (0.25 *versus* 0.57, $P < 0.05$), ascending aorta (0.25 *versus* 0.72, $P < 0.001$), and proximal arch (0.39 *versus* 0.67, $P < 0.05$). RTAD were more likely to undergo aortic valve resuspension (100% *versus* 74%, $P = 0.044$).

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Disclosure

The authors have no financial or personal relationships with other people or organizations that could potentially and inappropriately influence (bias) their work and conclusions.

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Conclusions: RTAD tends to occur when primary tears occur in close proximity to the aortic arch and when false lumen decompression through the distal aortic branches are less effective. Compared with ATAD, RTAD has less root involvement, and successful aortic valve resuspension is more likely.

Keywords

Aortic dissection; Aortic operation; Computed tomography; Outcomes

Introduction

Spontaneous acute retrograde type A dissections (RTADs) have been described in 7%–25% of acute type A dissections.^{1–3} While most acute type A dissections have a tear in the aortic root or ascending aorta with antegrade propagation of the false lumen, RTAD originates from a primary tear in the aorta beyond the left subclavian where the false lumen propagates in a direction opposite to the direction of blood flow to the proximal arch and ascending aorta.⁴ It is well recognized that this can occur during medical treatment of type B dissections without aortic instrumentation. It can be viewed as a retrograde arch extension that extends to the ascending aorta.

Retrograde propagation of an acute type B dissection often manifests as recurrent pain that occurs at variable times during hospitalization.⁴ Acute hypertension such as with cocaine use with intense false lumen pressurization has been suggested as a mechanism for retrograde extension in some cases.^{4,5} Although endovascular therapies are an attractive option in the treatment of descending aortic pathologies, important considerations include proximity of the primary tear to the arch, need for arch coverage with possible compromise of branch vessels, as well as the degree of arch curvature.⁶ Indeed, even coverage of the left subclavian is associated with higher neurologic morbidity and death.^{7,8} Given an accommodating anatomy, thoracic endografting for the descending thoracic aorta with primary tear coverage in this scenario has been successfully used by groups such as Kato *et al.*⁹

This study focuses on spontaneous RTAD as opposed to the iatrogenic retrograde dissections from new intimal tears which complicates 1%–4% thoracic endovascular aortic repair procedures.^{10–12} Hemiarch replacement in acute type A dissection has been widely adopted. However, contemporary studies suggest that total arch replacement may also be a reasonable primary treatment strategy with subsequent advantages in addressing residual disease using endovascular techniques.¹³ This is of particular relevance in RTAD as extended arch resections will increase the likelihood of primary tear resection to decrease false lumen perfusion. Alternatively, it has been suggested that conservative medical therapy with close monitoring in patients with RTAD meeting specific anatomic criteria is a reasonable approach.^{1,14} It is therefore very relevant to examine the anatomic features and pathogenic mechanisms of RTAD and its implications for therapeutic strategies. In this study, we conducted a detailed morphologic analysis and comparison of both acute RTAD and antegrade type A dissections (ATADs) as well as classic type B dissections. We also examined presenting features and surgical outcomes of the two groups of type A dissections.

Materials and methods

Patients

This study was approved by the University of Wisconsin–Madison Institutional Review Board. A waiver of the need to obtain consent from patients was approved. We conducted a retrospective review of patients with RTAD ($n = 12$), ATAD ($n = 96$), and type B dissection ($n = 92$) who presented to the University of Wisconsin Hospitals and Clinics between January 2000 and February 2016. The incidence of RTAD in our series was 11.1% of all acute type A dissections. Dissection was defined as acute if the onset of symptoms was <14 d from the time of surgery. Diagnosis was made by computed tomography (CT) scans, echocardiography, and surgical findings. We included baseline demographics, presentation, operative data, and postoperative outcomes in acute type A dissection patients.

Imaging analysis

Three-dimensional reconstruction of CT scan images was performed using TeraRecon iNtuition image analysis software (TeraRecon Inc, Foster City, CA). Aortic measurements were made using a center-line method which yields aortic cross-sectional images orthogonal to the direction of blood flow. This avoids inaccurate measurements from inadvertent oblique images. CT scan measurements were performed by a faculty cardiothoracic surgeon with expertise in image analysis, clinical evaluations, and operative intervention for aortic disease. Corresponding to the aortic arch zones as previously described by Mitchell and Ishimaru,¹⁵ proximal arch is zone 0, distal arch is zone 3, proximal descending thoracic aorta is the upper third of zone 4, whereas mid-descending thoracic aorta corresponds to middle of zone 4.

For the true and false lumen, we measured the luminal areas, and fraction of the total perimeter that the true and false lumen occupies to quantify the degree of circumferential medial separation or injury. False lumen flow is quantified in the arterial phase of the CT scan as the contrast intensity ratio of the false lumen as a fraction of the true lumen. We also noted the location and size of the primary and secondary tears. The estimated area of the primary tear was calculated by multiplying the length by the width of the tear. We also corroborated the size and location of the tears with intraoperative findings on transesophageal echocardiography and surgical anatomy.

We determined the presence of branch dissection and evaluated the perfusion of noncoronary aortic branches including the innominate, right carotid, right subclavian, left carotid, left subclavian, celiac, superior mesenteric, left and right renal, inferior mesenteric, as well as left and right iliac arteries. Translational motion artifact at the aortic root precluded accurate in-vivo imaging of coronary perfusion.

Follow-up

Survival data were available for all 108 acute type A dissection patients. Mid-term survival data were obtained through detailed clinical follow-up. The maximum follow-up was 13.3 y with a total follow-up of 277.9 patient-years and a mean follow-up of 2.6 ± 3.1 y.

Statistical methods

Pearson -square test or Fisher's exact test was used to analyze categorical variables. KaplanMeier Survival curve with ManteleCox statistics was used to analyze survival data. Wilcoxon rank-sum test was used to compare continuous variables. The year of operation is expressed as median with interquartile range and analyzed with median test. Pearson's correlation coefficient was used to examine the relationship between false-to-true lumen contrast intensity ratio and the number of major aortic branches perfused by the false lumen. Statistics were performed using Statistical Package for the Social Sciences software (SPSS Inc, Chicago, IL).

Results

Determinants of retrograde false lumen extension

Compared with type B dissection, primary tears in RTAD were more often located in the distal arch (9/12 (75.0%) *versus* 40/92 (43.5%), $P=0.04$) as opposed to the rest of the descending thoracic and abdominal aorta. Therefore, 9 of 49 distal arch primary tears (18.4%) were associated with an RTAD. Comparing RTAD with type B dissections respectively, there were no differences in the primary tear area ($2.31 \pm 2.04 \text{ cm}^2$ *versus* $2.92 \pm 2.94 \text{ cm}^2$, $P=0.82$), the number of secondary tears (0.83 ± 0.83 *versus* 0.60 ± 0.73 , $P=0.23$), or the cumulative diameter of secondary tears ($1.11 \pm 1.65 \text{ cm}$ *versus* $0.59 \pm 0.83 \text{ cm}$, $P=0.55$). We found a lower false-to-true lumen contrast intensity ratio in the mid-descending thoracic aorta of RTAD *versus* type B dissections (0.46 ± 0.36 *versus* 0.71 ± 0.34 , $P=0.04$) indicating greater thrombosis or more sluggish flow at this location. Interestingly, the true lumen in RTAD occupies a smaller fraction of the total perimeter at the distal arch than type B dissections (0.34 ± 0.23 *versus* 0.51 ± 0.30 , $P=0.04$) indicating greater circumferential wall delamination and injury in RTAD at this location.

We next examined the perfusion characteristics of major aortic branches distal to the left subclavian (i.e., celiac, superior mesenteric, right and left renal, inferior mesenteric, as well as right and left iliac arteries). Evaluating RTAD and type B dissection respectively, there was no difference in the number of branches that were dissected (1.58 ± 1.38 *versus* 1.79 ± 1.79 , $P=0.94$). The RTAD false lumen was less decompressed through major aortic branches compared with type B dissections as there were less branches perfused either via the false lumen only or through both the false and true lumens (0.67 ± 0.98 *versus* 1.70 ± 1.76 , $P=0.04$). We then limited our evaluation only to RTAD and type B dissection patients with primary tears in the distal arch or proximal descending thoracic aorta. This resulted in an even larger difference and repeated our observation that RTAD patients have less major aortic branches that are perfused through the false or both lumens (0.40 ± 0.70 *versus* 2.16 ± 1.71 , $P=0.001$). These findings suggest less effective false lumen decompression in the acute period and increased retrograde false lumen pressure and fluid momentum.

We further examined the statistical correlation between mid-descending thoracic aorta false-to-true lumen contrast intensity ratio with the number of branches perfused by the false lumen only or by both the true and false lumen. There was a significant correlation in the

Pearson's correlation coefficient between these two parameters in type A dissections (Fig. 1, $r = 0.50$, $P < 0.01$) as well as RTAD and type B dissections with primary tears in the distal arch or proximal descending thoracic aorta (Fig. 2, $r = 0.57$, $P < 0.01$).

Morphology comparison between retrograde and ATAD

Compared with ATAD where the tear originates in the root, ascending aorta, or proximal arch, RTAD tended to have a larger total aortic area (Table 1) in the distal arch ($P = 0.02$), and mid-descending thoracic aorta ($P = 0.02$) likely corresponding to the primary tear at these sites causing abrupt expansion from acute aortic weakness. This is also illustrated by circumferential delamination and injury where the portion of the total perimeter occupied by the true lumen was lower in RTAD than ATAD at the distal arch (0.34 ± 0.23 versus 0.55 ± 0.31 , $P = 0.01$) and mid-descending thoracic aorta (0.39 ± 0.22 versus 0.60 ± 0.31 , $P = 0.02$). Furthermore, there was a trend toward a smaller RTAD true-to-total lumen ratio (Table 2) at the distal arch ($P = 0.13$) and mid-descending thoracic aorta ($P = 0.09$) indicating greater injury and a larger false lumen in the region of the primary tear site. Conversely, RTAD had a larger true-to-total lumen ratio at the root ($P = 0.11$) and ascending aorta ($P = 0.03$) consistent with less proximal aortic injury. Like other investigators,^{1,14} we found a lower false-to-true lumen intensity ratio at the root (0.26 ± 0.34 versus 0.57 ± 0.43 , $P = 0.01$), ascending aorta (0.25 ± 0.19 versus 0.72 ± 0.31 , $P < 0.01$), and proximal arch (0.39 ± 0.30 versus 0.67 ± 0.36 , $P = 0.01$) indicating more thrombosis and/or sluggish flow at these sites.

We then compared the arch branch (i.e., innominate, right and left carotid, and right and left subclavian arteries) perfusion of RTAD versus ATAD. RTADs are much less likely to have dissected arch vessels (0.50 ± 1.17 versus 1.44 ± 1.62 , $P = 0.04$), more likely to have true lumen only flow (4.83 ± 0.39 versus 3.81 ± 1.56 , $P = 0.02$), and much less likely to have false lumen only or both false and true lumen perfusion of branch vessels (0.17 ± 0.39 versus 1.03 ± 1.48 , $P = 0.04$). None of the RTAD had occlusion of arch branch vessels (0 versus 0.16 ± 0.60 , $P = 0.336$). No differences in branch perfusion characteristics beyond the left subclavian ($P > 0.05$) were identified between RTAD and ATAD. Figure 3 depicts the types of dissections and different proportions as outlined in the results.

Clinical presentation and outcomes comparison between retrograde and ATAD

There were no differences in the age, gender, weight, height, creatinine, left ventricular ejection fraction, or major comorbidities between RTAD and ATAD (Table 3). No differences in cardiogenic shock on presentation or operative year were seen (Table 4, $P > 0.05$). Consistent with the lack of differences in distal aortic branch perfusion between RTAD and ATAD, there was no difference in the presence of malperfusion (Table 4, $P = 0.85$). In the RTAD group (Table 4), aortic valve resuspension was more prevalent ($P = 0.044$) likely related to less root injury and probably contributed toward the trend for a shorter cross clamp time in the RTAD group ($P = 0.066$). No differences in major postoperative complications or length of hospital stay were seen (Table 5, $P > 0.05$). No statistical difference in 30-d operative mortality ($0/12$ (0%) versus $10/96$ (10.4%), $P = 0.241$) or mid-term survival ($P = 0.120$) was seen.

Discussion

Our RTAD incidence of 11.1% of all type A dissections is consistent with previous reports of 7%–20%.^{1,3,14} RTADs with primary tears in the descending thoracic aorta have distinct implications for clinical presentation and therapeutic strategies. While earlier experiences recommended an early surgery approach for RTAD,^{16–18} more contemporary authors suggest that conservative medical treatment may be appropriate for select patients.^{1,14} Features suggesting that a medical approach may be reasonable include hemodynamic stability, thrombosis of ascending false lumen, and an ascending aorta <5.5 cm.^{14,19} Close monitoring with serial CT scans and noting changes in symptoms will guide a cross over to surgery.¹⁴ Conversely, surgical intervention for RTAD is suggested for a nonthrombosed false lumen, enlargement of the ascending aorta and arch, and serious complications including aortic regurgitation, and aortic rupture.¹

Kaji *et al.* have also noted improved prognosis in patients with thrombosed ascending false lumens.¹ However, we tended to advocate surgery as the initial strategy for RTAD because of concern over reliability of patient follow-up as well as access to medical care in a more rural setting with patient transfers over long distances. This approach has worked well for our group as we had zero 30-d operative mortality for the RTAD patients. Perhaps, because of the low patient numbers, we were not able to demonstrate a long-term survival difference.

Consistent with prior findings,¹⁷ we did not find significant differences in the clinical profile of RTAD and ATAD patients. Although the series of RTAD described by Ryalski *et al.* included those resulting from thoracic endovascular aortic repair patients in addition to medically managed type B dissections, our findings are similar in that the RTAD had larger descending thoracic aortic dimensions compared with ATAD.²⁰ The same group also explains this phenomenon as resulting from acute aortic weakness that increases the aortic diameter and changes the ellipticity, tortuosity, and volume at the initial site of the dissection.²⁰

Surgical repair of RTAD has several favorable features compared with ATAD. Patients are more likely to undergo aortic valve resuspension because of less root disease. This translates into a shorter cross-clamp time. Indeed, the need to address the primary tear in RTAD to promote false lumen thrombosis and improved long-term outcomes presents unique challenges.¹⁸ Occasionally, resection of the primary tear can be achieved through a median sternotomy incision as part of a total arch replacement or a frozen elephant trunk procedure. This is facilitated by 75% of the primary tears being at the distal arch in RTAD. However, even if the primary tear cannot be resected through a median sternotomy approach, arch branch rerouting with proximalization can greatly facilitate future stent-graft interventions and reduce types I and II endoleaks because of a persistent false lumen in the arch.²¹ Our finding that arch branches are less likely to be dissected in RTAD makes debranching and associated total arch replacement procedures easier by being able to sew to a preserved branch vessel wall. Although total aortic arch replacement was reported to be associated with significant morbidity and a high-operative mortality of 13%–46%,^{2,22} more recent reports suggest similar short- and long-term survival outcomes between hemi and total arch replacement for acute type A dissection.²³

Our comparison of RTAD and type B dissection morphologies provides some insight into the possible mechanism of RTAD. Although retrograde progression in a medically treated type B dissection is occasionally captured on serial CT, the temporal sequence of events may vary from delayed retrograde extension to a simultaneous retrograde and antegrade extension during initiation of the primary tear. Similar to findings by Nauta *et al.* where more false lumen thrombosis occurred in patients with retrograde arch extension,⁴ our group also identified greater thrombosis or reduced flow in the distal false lumen. Our data from bivariate correlation analysis and enumeration of branch vessels perfused by the false lumen suggest that this may be from less effective decompression of the false lumen through the distal aortic branches. Decompression of the false lumen via aortic branches can occur when perfusion from the false lumen re-enters the true lumen via a distal branch re-entry point so the false lumen flow returns to the true lumen for effective tissue perfusion. The data suggest that the poor false lumen distal outflow leads to greater back pressure at the primary tear with greater circumferential delamination at this site. This also redirects blood flow in the false lumen in a retrograde direction thereby promoting an RTAD.

Conclusions

Based on our results, acute type B dissections are at higher risk for early retrograde extension with possible conversion to a type A if the primary tear is at the distal arch, presence of sluggish flow or thrombosis of the distal false lumen, and if there is not at least one major abdominal aortic branch that is perfused through the false lumen only or through both the false and true lumens. Careful follow-up imaging and attentions to changes in symptoms are likely important in this “at risk” group of patients.

The conclusion of our study is limited by its retrospective nature in a single institution with its inherent limitations and biases. Because of variation in the quality of the CT scans, we only included studies of adequate quality in the form of arterial phase CT scans with a resolution that defined the aortic anatomy adequately for our measurements. Our conclusions on survival outcomes are limited by a short mean follow-up of 2.6 y and the small number of RTAD patients. Branch perfusion is dynamic, especially in the acute phase, so relative perfusion by the true and false lumen is likely not fully reflected on a static image. However, static CT scan images are currently the most widely accessible imaging technique for dissection diagnosis.

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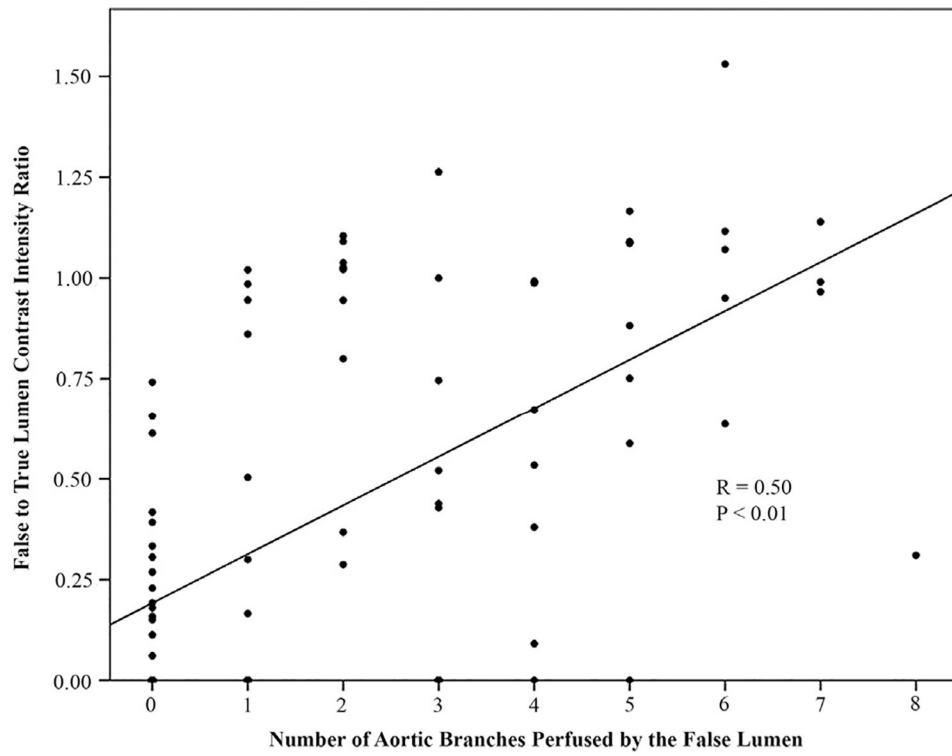


Fig. 1-
Contrast intensity ratio at mid-descending thoracic aorta *versus* false lumen perfusion of aortic branches in all type A dissection.

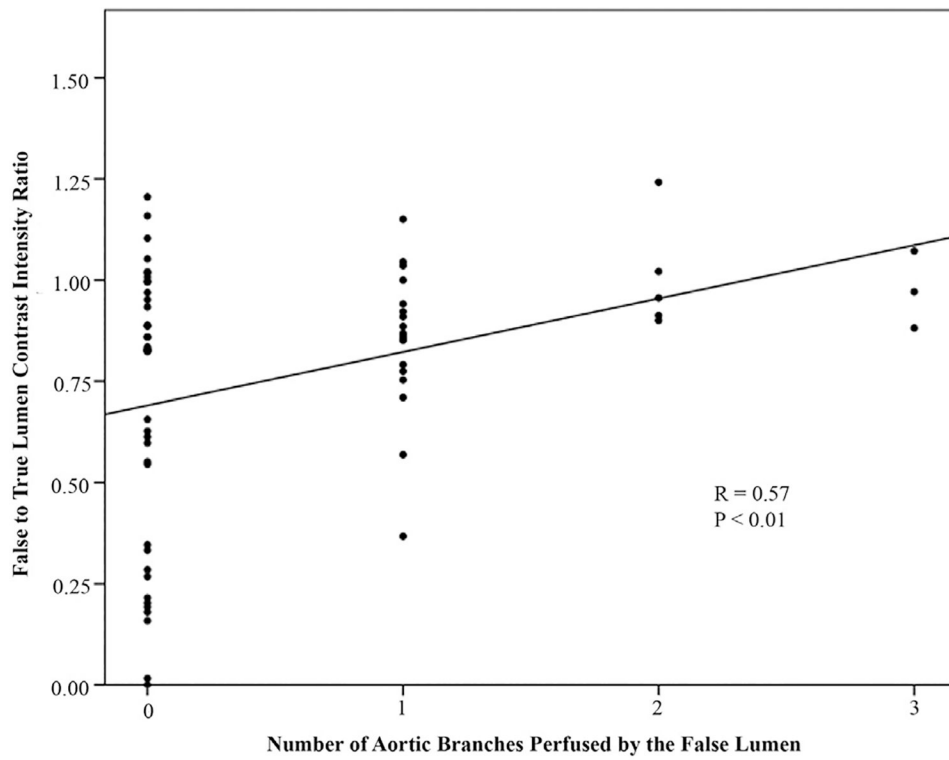


Fig. 2- Contrast intensity ratio at mid-descending thoracic aorta *versus* false lumen perfusion of aortic branches in dissections with primary tears in the proximal descending thoracic aorta.

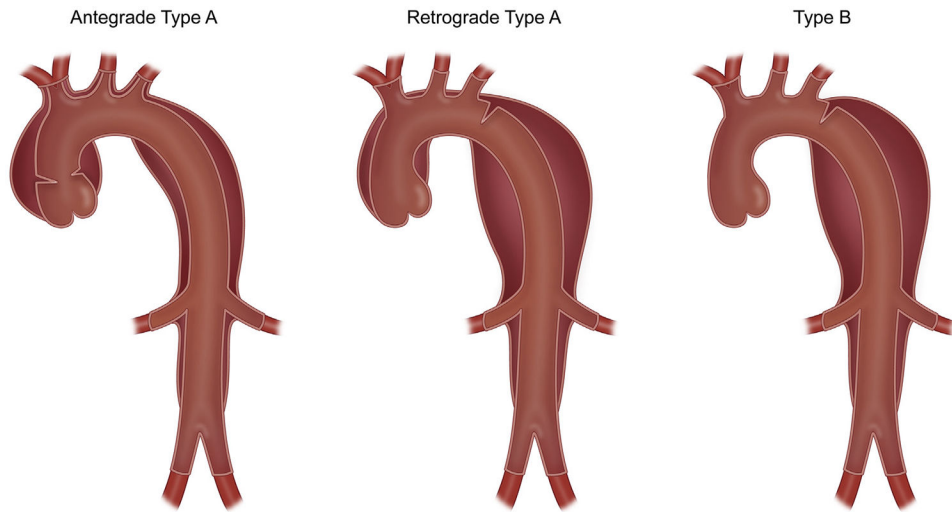


Fig. 3- Anatomic depiction of the morphologic differences between antegrade type A, retrograde type A and type B dissections.

Table 1–Total aortic cross-sectional area in RTAD *versus* ATAD.

Aortic segment areas (mm ²)	RTAD (n = 12)	ATAD (n = 96)	P value
Annulus	468.75 ± 78.81	436.88 ± 133.80	0.18
Root	1255.92 ± 465.15	1613.72 ± 711.84	0.04
Ascending aorta	1694.92 ± 490.85	1954.83 ± 705.73	0.34
Proximal arch	1346.58 ± 329.62	1412.64 ± 541.01	0.95
Distal arch	956.25 ± 290.31	782.09 ± 226.15	0.02
Mid-descending thoracic	803.00 ± 211.12	664.20 ± 187.22	0.02
Supra-celiac	690.25 ± 106.49	592.61 ± 246.30	0.01
Infra-renal	449.80 ± 188.54	334.32 ± 137.78	0.02

Continuous data expressed as mean standard deviation. Comparisons calculate using the Wilcoxon rank-sum test.

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Table 2–True lumen area as fraction of total lumen area in RTAD *versus* ATAD.

Aortic segment	RTAD (n = 12)	ATAD (n = 96)	P value
Root	0.88 ± 0.13	0.76 ± 0.22	0.11
Ascending aorta	0.50 ± 0.21	0.38 ± 0.28	0.03
Proximal arch	0.54 ± 0.13	0.46 ± 0.22	0.04
Distal arch	0.50 ± 0.19	0.62 ± 0.28	0.13
Mid-descending thoracic	0.45 ± 0.21	0.62 ± 0.32	0.09
Supra-celiac	0.49 ± 0.29	0.63 ± 0.34	0.28
Infra-renal	0.65 ± 0.32	0.71 ± 0.38	0.53

Continuous data expressed as mean standard deviation. Comparisons calculate using the Wilcoxon rank-sum test.

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Table 3–Patient demographics in RTAD *versus* ATAD.

Variable	RTAD (n = 12)	ATAD (n = 96)	P value
Age (y)	59.0 ± 12.9	60.3 ± 15.5	0.68
Sex (male)	8 (66.7%)	67 (69.8%)	0.82
Weight (kg)	94.6 ± 14.7	88.5 ± 24.3	0.12
Height (cm)	174.6 ± 11.2	174.1 ± 10.6	0.97
Preoperative creatinine (mg/dL)	1.21 ± 0.45	1.12 ± 0.43	0.42
Ejection fraction (%)	61.5 ± 3.4	57.6 ± 9.8	0.21
Hypertension	9 (75.0%)	65 (67.7%)	0.61
Cerebrovascular disease	1 (8.3%)	5 (5.2%)	0.66
Peripheral vascular disease	2 (16.7%)	17 (17.7%)	0.93
Lung disease	2 (16.7%)	15 (15.6%)	0.92
Liver disease	0 (0%)	2 (2.1%)	0.61
Diabetes	1 (8.3%)	6 (6.2%)	0.78
Hyperlipidemia	7 (58.3%)	43 (44.8%)	0.38
Cancer within 5 y of surgery	0 (0%)	3 (3.1%)	0.53

All nominal data expressed as n and percentage of total population and compared with Pearson χ^2 or Fisher's exact test. Continuous data expressed as mean ± standard deviation with comparisons calculated using the Wilcoxon rank-sum test.

Table 4--

Presentation and operative parameters.

Variable	RTAD (n = 12)	ATAD (n = 96)	P value
Cardiogenic shock on presentation	1 (8.3%)	8 (8.3%)	1.00
Malperfusion on presentation	2 (16.7%)	14 (14.6%)	0.85
Operation year*	2014 (4, 2011–2015)	2012 (5, 2009–2014)	0.13
Redo surgery	0 (0%)	8 (8.3%)	0.30
AV resuspension	12 (100%)	71 (74.0%)	0.04
CVG	0 (0%)	19 (19.8%)	0.09
AVR	0 (0%)	5 (5.2%)	0.42
VSR	0 (0%)	1 (1.0%)	0.72
CABG	1 (8.3%)	2 (2.1%)	0.21
Mitral valve surgery	0 (0%)	0 (0%)	0
Tricuspid valve surgery	0 (0%)	0 (0%)	0
Ascending aortic replacement only	5 (41.7%)	54 (56.2%)	0.34
Ascending and hemiarch replacement	7 (58.3%)	36 (37.5%)	0.16
Ascending and total arch replacement	0 (0%)	6 (6.2%)	0.37
Cardiopulmonary bypass time (min)	270.67 ± 84.25	267.26 ± 96.75	0.83
Cross-clamp time (min)	82.50 ± 22.40	108.41 ± 47.59	0.07
Circulatory arrest (min)	40.92 ± 8.02	33.66 ± 15.02	0.01

AV = aortic valve; AVR = aortic valve replacement; CABG = coronary artery bypass grafting; CVG = composite valve graft; VSR = valve sparing root replacement.

* Median, first quartile-third quartile. All nominal data expressed as *n* and percentage of total population and compared with Pearson χ^2 or Fisher's exact test. Continuous data expressed as mean standard deviation with comparisons calculated using the Wilcoxon rank-sum test. Year of operation is expressed as median with interquartile range and analyzed with median test.

Table 5–

Postoperative outcomes.

Variable	RTAD (<i>n</i> = 12)	ATAD (<i>n</i> = 96)	<i>P</i> value
Neurologic events	1 (8.3%)	11 (11.4%)	0.74
Pneumonia	1 (8.3%)	10 (10.4%)	0.82
Prolonged ventilation	6 (50.0%)	58 (60.4%)	0.49
Gastrointestinal	0 (0%)	6 (6.2%)	0.37
Acute renal failure	2 (16.7%)	10 (10.4%)	0.52
Surgery to discharge (d)	11.42 ± 7.39	10.52 ± 17.27	0.31

All nominal data expressed as *n* and percentage of total population and compared with Pearson χ^2 or Fisher's exact test. Continuous data expressed as mean ± standard deviation with comparisons calculated with the Wilcoxon rank-sum test.

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