Circulation Journal
Circ J 2023; **87:** 440–447
doi:10.1253/circj.CJ-22-0359

ORIGINAL ARTICLE

Location of the False Lumen Within the Medial Layer in Acute Intramural Hematoma

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Background: We compared the location of the false lumen within the medial layer between acute intramural hematoma (AIH) and acute aortic dissection (AAD) using microscopic images of aortic specimens and examined the associations with patient characteristics, CT findings, and late outcomes.

Methods and Results: Among 293 patients undergoing surgery for Stanford type A acute aortic syndrome between 2008 and 2018, 45 patients had neither an identifiable intimal tear, flow to the false lumen on preoperative CT or intimal tear by intraoperative observation (AIH group), and 98 patients with patent false lumen were enrolled (AAD group). The AIH group had a significantly thinner outer media thickness (OMT) than the AAD group. The AIH group showed more pericardial effusion, but distal progression of dissection and branch vessel involvement were limited. The change in aortic diameter after surgery was insignificant in the AIH group, whereas in the AAD group it continued to increase. Cumulative incidence of aortic adverse events was significantly higher among AAD patients, but no significant difference was observed in survival between groups.

Conclusions: The AIH group had a significantly thinner OMT than the AAD group, which was significantly associated with a large amount of pericardial effusion, greater false lumen diameter, and limited progression of aortic dissection.

Key Words: Acute aortic syndrome; Aneurysm; Imaging; Intramural hematoma; Pathology

Acute aortic dissection (AAD) and acute intramural hematoma (AIH) share similar clinical features, and diagnostic and therapeutic challenges, but each has a distinct etiology.[1–3] AAD is characterized by intimal-medial tearing and longitudinal cleavage of the aorta within the medial layer, creating a false lumen, whereas AIH is characterized by the rupture of the vasa vasorum within the medial layer without intimal-medial tearing. Regarding the histological features of AAD, Uchida and his colleagues provided some thought-provoking data, showing that the false lumen in AIH was created at a more superficial location, closer to the adventitia than in AAD.[4] There is room for further research on this topic, as the generalizability was not confirmed. In fact, no additional data have been published since that report. Therefore, the present study compared the location of the false lumen within the medial layer between AIH and AAD using microscopic images of aortic specimens, and examined the association of outer medial thickness (OMT) with patients' characteristics, computed tomography (CT) findings, and late outcomes.

Methods

The institutional review board approved the study (R2018-185; February 28th, 2019), and written informed consent was waived given the retrospective nature of the study, which was performed in accordance with the principles of the Declaration of Helsinki. Of 293 patients who underwent aortic surgery with a diagnosis of Stanford type A acute aortic syndrome between 2008 and 2018, 77 were excluded due to a lack of aortic specimens (n=56) or preoperative CT data (n=25). Of the remaining 216 patients, 45 (21%) who met the following criteria were included in the AIH group: a thrombosed false lumen with neither identifiable intimal tear nor flow to the false lumen anywhere in the aorta and no intimal tear by intraoperative observation. Patients who had a completely thrombosed false lumen but in whom intimal tear was clearly identified during surgery were not included in the AIH group and excluded from the analysis (n=25). Patients with a partially thrombosed false lumen were also excluded (n=48). Therefore, 98 patients with a patent false lumen were enrolled in the AAD group. The patient flow diagram is shown in

Received June 19, 2022; revised manuscript received September 13, 2022; accepted September 27, 2022; J-STAGE Advance Publication released online November 3, 2022 Time for primary review: 17 days

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ISSN-1346-9843

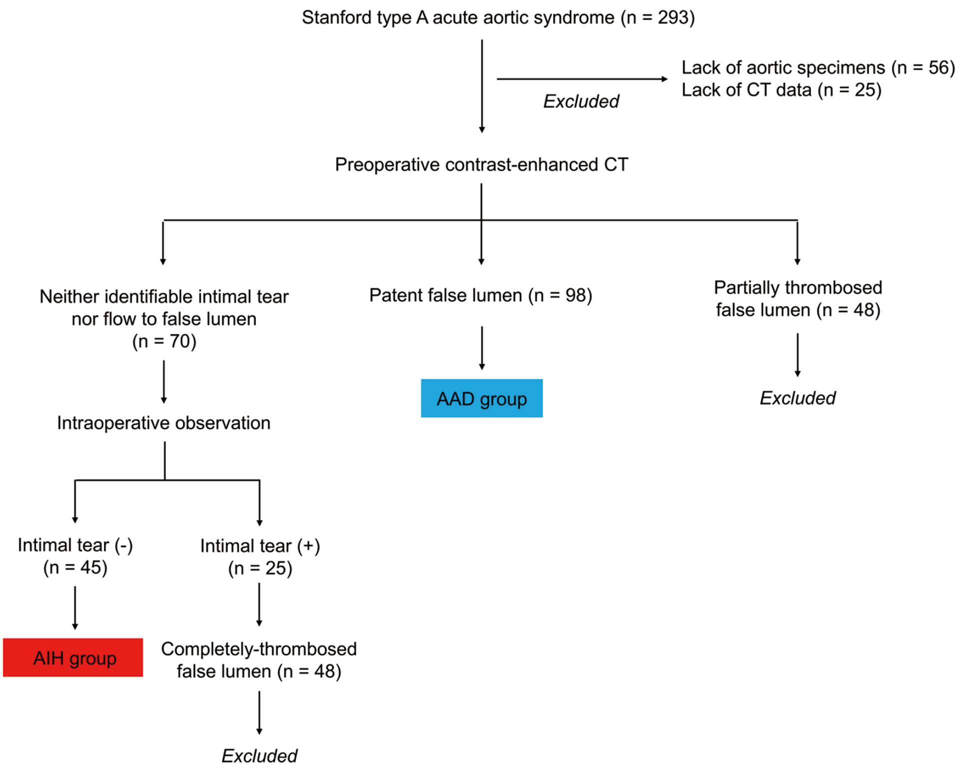

Figure 1. Patient flow chart. CT, computed tomography.

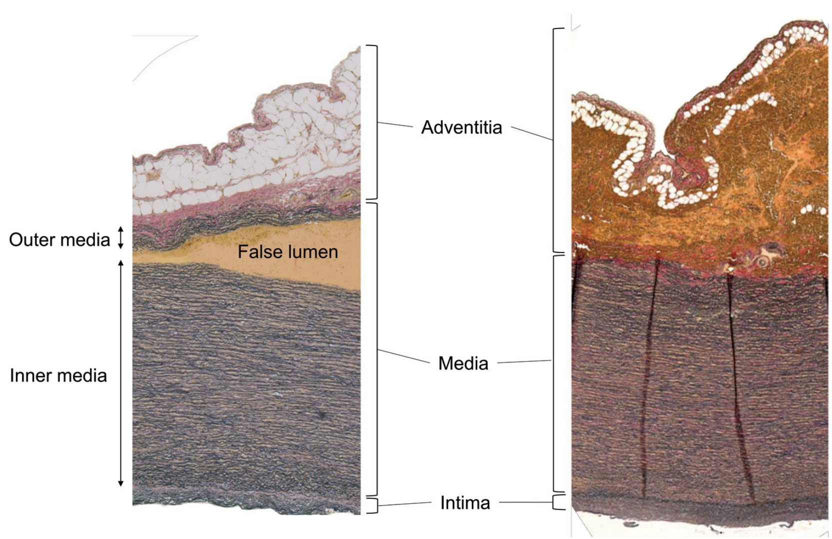

Figure 2. Microscopic measurement of wall thickness. (**Left**) Typical microscopic image from the AIH group (elastica van Gieson stain, ×40). There was no clear evidence of a false lumen in the media in some patients, and hematoma was observed outside the media (**Right**). In these cases, we calculated the outer media thickness as 0 mm. Measurements of inner and outer media were made at 8 circumferentially evenly spaced places.

Figure 1. In all cases, patients underwent emergency surgery unless contraindicated for neurologic devastation, metastatic cancer, or patient refusal. Detailed surgical procedures have been described[5–7] and are summarized in the supplementary text. All data were obtained directly from the electronic medical records.

Tissue Measurements

The aortic wall specimens were stained with elastica van Gieson stain (**Figure 2**). The thickness of the media of the inner and outer layers at the false lumen and overall wall thickness of the dissected aorta were measured at 8 circumferentially evenly spaced places using image analysis soft-

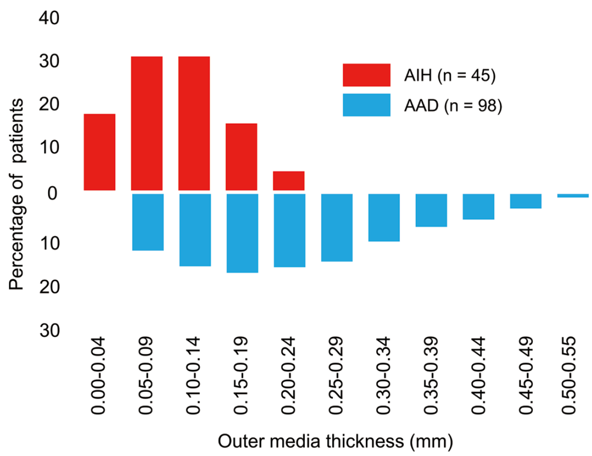

Figure 3. Mirrored histogram of the distribution of outer media thickness for both groups. AAD, acute aortic dissection; AIH, acute intramural hematoma.

ware (Image-Pro Plus, Media Cybernetics, Bethesda, MD, USA), and the average values were calculated.

Image Analysis

All postoperative CT was performed using a 64-multidetector row system (Aquilion 64, Toshiba Medical Systems, Otawara, Japan) with 1- to 5-mm collimation. The majority of follow-up CT images were unenhanced and used only for aortic diameter measurements. All CT images were retrieved from the local archive and analyzed with an independent 3D server (Aquarius WorkStation, TeraRecon Inc., San Mateo, CA, USA). Aortic diameters were measured using 3-dimensional multiplanar reconstruction images in an axial plane perpendicular to the median aortic centerline by the external-to-external edge method.[8] The median centerline was manually adjusted to measure the largest overall aortic diameter. Measurements were performed at 5 levels: (1) the ascending aorta (at the level of pulmonary trunk bifurcation); (2) the aortic arch (between the left common carotid artery and the left subclavian artery); (3) the proximal descending aorta (2 cm distal to the left subclavian artery); (4) the middle descending aorta (at the level of pulmonary trunk bifurcation); (5) the aorta at the diaphragm (2 cm above the celiac trunk). False lumen diameters were measured perpendicular to the contour of the intimal flap.

Follow-up

Patients were discharged on β-blocker treatment, unless contraindicated, combined in some cases with other antihypertensive drugs aimed at achieving systolic blood pressure ≤120 mmHg. Patients were regularly followed in the outpatient department. Physicians checked for adequacy of blood pressure control and any symptoms or complications on CT scans obtained at 3–6 months and annually thereafter. Surgical or endovascular intervention was considered if any of the following were observed: (a) dissection progression or aortic dilatation with signs of impending rupture, (b) progressive severe aortic valve regurgitation with symptoms or severe left ventricular dilatation, (c) prosthesis infection, and (d) aortic diameter >55 mm. Aortic events were defined as a need for aortic surgery, aortic dissection/rupture occurrence, dilatation of the distal aorta >55 mm, and aortic-related death (death from aortic rupture or dissection, or sudden unexplained death). Survival was ascertained by reviewing medical reports from follow-up visits at the outpatient clinic or phone contact with the patient, family, or physician. Specification of the cause of death was based on information obtained from family members, death certificates, and hospital records. Follow-up information was available for 91.4% of the study group.

Statistical Analysis

Bivariate differences were compared using the chi-square test or Fisher's exact test for categorical data as appropriate, and unpaired t-test or Mann-Whitney U-test for continuous variables after evaluation of normality by Kolmogorov-Smirnov tests. Logistic regression analysis was used to estimate the association of independent variables with 1 dichotomous dependent variable. To clarify the association between OMT and baseline characteristics and CT findings, multivariable logistic regression was conducted with OMT of 0.00–0.10 mm as the binary dependent variable and the following variables as independent variables: age, female sex, body mass index, hypertension, smoking, estimated glomerular filtration rate, pericardial effusion of 10–20 mm, false lumen diameter >15 mm, and progression of dissection beyond the left subclavian artery. To eliminate multicollinearity, variables that essentially measured the same thing were not entered into the model. Multicollinearity of the regression model was examined by calculating the variance inflation factor for each variable. None of them exceeded 2 (greatest, 1.23), which indicated that multicollinearity was not a significant issue. The linear mixed-effects model assessed changes in aortic diameter with random intercepts. Statistical analysis of time to aortic events considered death as a competing risk. Cumulative incidence function was estimated, and Gray's test was used to determine differences in cumulative incidence function among groups.[9,10] Probabilities of survival were calculated using the Kaplan-Meier method, in which patients still alive were censored at the date of their last follow-up. The log-rank test determined differences in survival function among groups. To determine the interobserver variability in the evaluation of wall thickness and aortic diameters, measurements were repeated in 20 patients by a second observer blinded to the values obtained by the first observer. To assess intraobserver variability, measurements were repeated 2–4 weeks later in 20 patients by an observer blinded to the results of the previous measurements. We calculated Lin's concordance correlation coefficient for each variable and confirmed that none of them was <0.86, which indicated that intraobserver and interobserver variability was not a significant issue.[11] For associations, we used a 2-sided test. A P value of 0.05 was considered statistically significant without controlling type I error for multiple testing. Estimations of 95% confidence intervals were based on 1,000 bootstrap samples.[12] All analyses were performed with SPSS statistical package version 25.0 (SPSS Inc., Chicago, IL, USA) and R software (https://www.r-project.org/).

Results

Distribution of OMT

Considerable variation in the OMT was observed in both

Table 1. Patients' Baseline Characteristics			
	AAD	AIH	P value
No. of patients	98	45	
Characteristics			
Age, years	66±11	75±7	0.01
Female sex	32 (33)	28 (62)	0.01
Body surface area	1.74±0.21	1.51±0.17	0.01
Body mass index	23.3±2.5	22.6±2.9	0.09
Hypertension	74 (75)	26 (58)	0.03
Smoking	41 (42)	10 (22)	0.02
Estimated GFR, mL/min/1.73 m²	72±19	66±17	0.04
CT findings			
Distal extent of the dissection			0.01
Ascending	0	9 (20)	
Arch	2 (2)	14 (31)	
Descending	10 (10)	13 (29)	
Abdominal-femoral	86 (88)	9 (20)	
Pericardial effusion (thickness on CT)			0.01
<5 mm	52 (53)	7 (16)	
5–10 mm	29 (30)	14 (31)	
10–20 mm	16 (16)	24 (53)	
Branch vessel involvement			0.01
None	37 (38)	42 (93)	
Cerebral	25 (26)	2 (4)	
Celiac or mesenteric	12 (12)	0	
Renal	23 (23)	1 (2)	
Femoral	10 (10)	0	
Aortic diameter, mm			
Middle ascending aorta			
Aortic diameter	48.9±7.2	48.3±5.5	0.82
False lumen diameter	27.7±5.4	16.4±4.2	0.01
Aortic arch			
Aortic diameter	41.4±5.6	38.0±4.9	0.12
False lumen diameter	16.7±4.2	8.1±3.1	0.01
Proximal descending aorta			
Aortic diameter	38.0±3.7	32.2±3.5	0.01
False lumen diameter	17.1±3.2	9.5±2.2	0.01
Middle descending aorta			
Aortic diameter	37.7±3.8	31.9±3.4	0.01
False lumen diameter	16.4±3.0	8.8±3.0	0.01
Aorta at diaphragm			
Aortic diameter	36.2±3.8	29.1±3.4	0.01
False lumen diameter	12.9±3.3	8.6±3.9	0.01

Data are mean ± standard deviation or frequency (percentage). AAD, acute aortic dissection; AIH, acute intramural hematoma; GFR, glomerular filtration rate.

groups (**Figure 3**), but the AIH group had a significantly thinner OMT than the AAD group (0.10±0.05 mm and 0.23±0.08 mm; t-test, P<0.01). No significant difference was found in overall wall thickness between groups (1.76±0.27 mm and 1.75±0.20 mm; t-test, P=0.93).

Baseline Characteristics and Preoperative CT

The AIH group was more likely have shorter, older women and less likely to have patients with hypertension and a smoking history than the AAD group (**Table 1**). Preoperative CT findings significantly differed between groups. In most cases, the aortic dissection extended beyond the diaphragm in the AAD group, and the false lumen diameters were significantly larger at all measurement sites. In the AIH group, the progression of aortic dissection and branch vessel involvement was relatively limited: only 20% of patients had dissection beyond the diaphragm, and approximately 90% had patent branch vessels. The amount of pericardial effusion was significantly larger in the AIH group. Multivariate logistic regression revealed that OMT of 0.00–0.10 mm was independently associated with pericardial effusion of 10–20 mm thickness on CT, false lumen diameter >15 mm, and a limited progression of dissection (**Table 2**).

Table 2. Variables Associated With Outer Media Thickness of 0.00–0.10 mm in the AIH Group

	OR	95% CI	P value
Age per 10-year increment	0.79	0.19–3.28	0.51
Female sex	1.21	0.37–3.96	0.65
Body mass index per 1-SD increment	1.02	0.51–2.04	0.85
Hypertension	1.33	0.41–4.31	0.72
Smoking	0.88	0.31–2.49	0.55
Estimated GFR per 1-SD increment	0.92	0.41–2.06	0.79
Pericardial effusion of 10–20 mm	2.61	1.19–5.72	0.02
False lumen diameter >15 mm at the ascending aorta	1.89	1.02–3.51	0.03
Progression of dissection beyond the LSCA	0.44	0.22–0.88	0.01

CI, confidence interval; LSCA, left subclavian artery; OR, odds ratio; SD, standard deviation. Other abbreviations as in Table 1.

Table 3. Intra- and Postoperative Outcomes

	AAD	AIH	P value
No. of patients	98	45	
Shock on arrival (systolic BP <80 mmHg)	24 (24)	20 (44)	0.02
Ongoing cardiopulmonary resuscitation	3 (3)	5 (11)	0.05
Operative outcome			
Operative procedure			0.43
Ascending/hemiarch replacement	93 (95)	44 (98)	
Total arch replacement	5 (5)	1 (2)	
Intimal tear in the ascending aorta	72 (73)	0	0.01
Operation time (min)	186±22	183±27	0.82
Cardiopulmonary bypass time (min)	105±14	96±16	0.29
Circulatory arrest time (min)	23±3	23±4	0.89
Prosthetic size (mm)	27.4±1.8	26.1±1.6	0.23
Lowest core temperature	24±2	23±2	0.39
Intraoperative bleeding (mL)	844±412	1,513±604	0.01
Postoperative outcomes			
LVEF, %	56±5	54±7	0.51
Re-exploration for bleeding	1 (2)	1 (2)	0.57
New stroke	9 (9)	3 (7)	0.61
AKI requiring hemodialysis	9 (9)	4 (9)	0.95
Heart block requiring pacemaker	0	1 (2)	0.57
30-day death	9 (9)	3 (7)	0.61
Postoperative hospital stay (days)	17±9	15±8	0.69
Follow-up, days	1,598±441	1,451±495	0.52
Medication at discharge			
β-blocker	72 (73)	34 (76)	0.79
ARB or ACE inhibitor	59 (60)	31 (69)	0.32
Calcium-channel blocker	56 (57)	27 (60)	0.75

Data are mean±standard deviation or frequency (percentage). AKI, acute kidney injury; ARB, angiotensin II receptor blocker; ACE, angiotensin-converting enzyme; BP, blood pressure; LVEF, left ventricular ejection fraction. Other abbreviations as in Table 1.

Intra- and Postoperative Outcomes

Patients in the AIH group were more frequently in cardiogenic shock than those in the AAD group (**Table 3**). An intimal tear was identified in the ascending aorta in 73% of patients in the AAD group. Intraoperative blood loss volume was significantly greater in the AIH group. There were no intraoperative deaths, but 3 patients died within 30 days after surgery in the AIH group and 9 in the AAD group. Of the 3 patients with 30-day mortality in the AIH group, 2 were transported to the operating room with ongoing cardiopulmonary resuscitation for severe hypotension due to cardiac tamponade and died within 1 week due to extensive brain injury.

Change in Aortic Diameter After Surgery

On average, 4.7 scans were obtained per patient (range, 2–7 scans), and the median follow-up was 4.1 years (25th–75th quartile, 2.3–6.4 years). **Figure 4** shows the change in

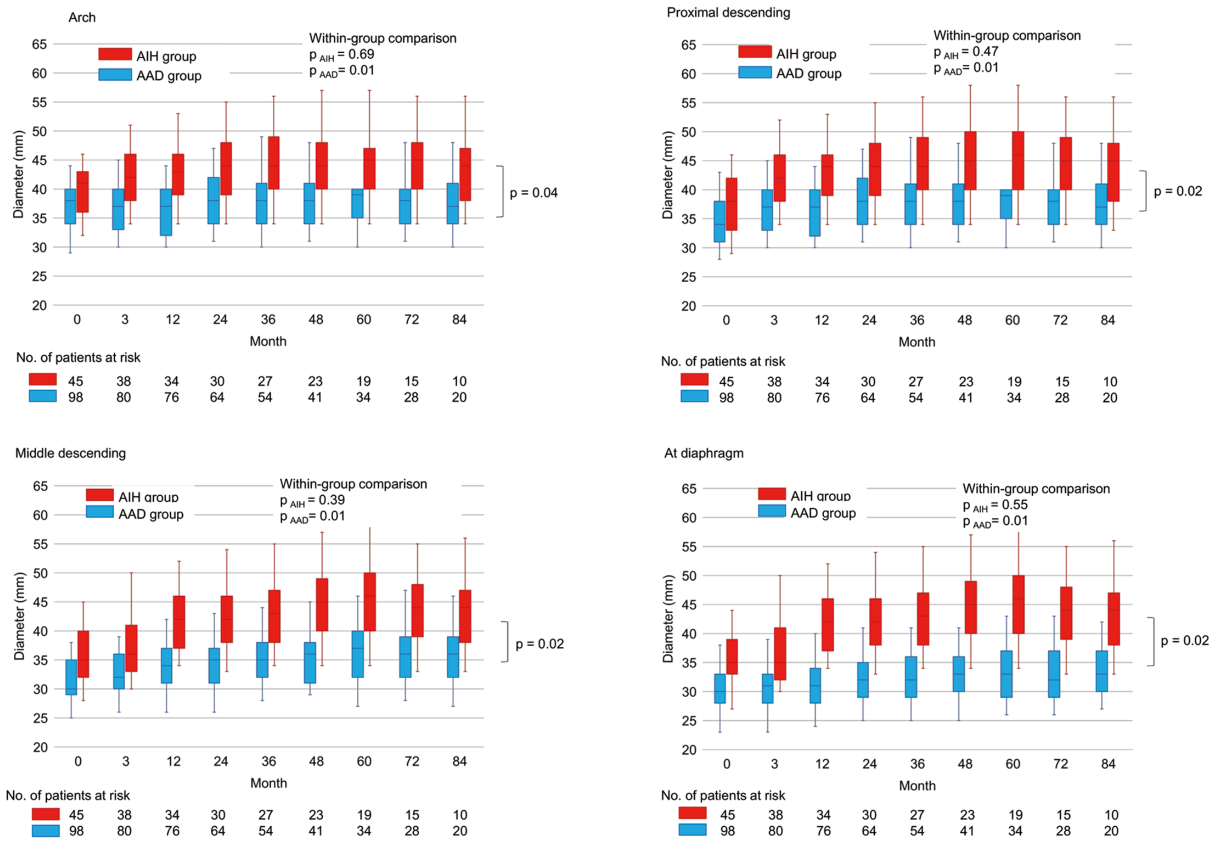

Figure 4. Box and whisker plots showing changes in aortic diameter. Boxes denote the lower and upper quartiles, and whiskers represent the entire range. The horizontal bar indicates the median. Between-group and within-group comparisons were based on the linear mixed model analysis. AAD, acute aortic dissection; AIH, acute intramural hematoma.

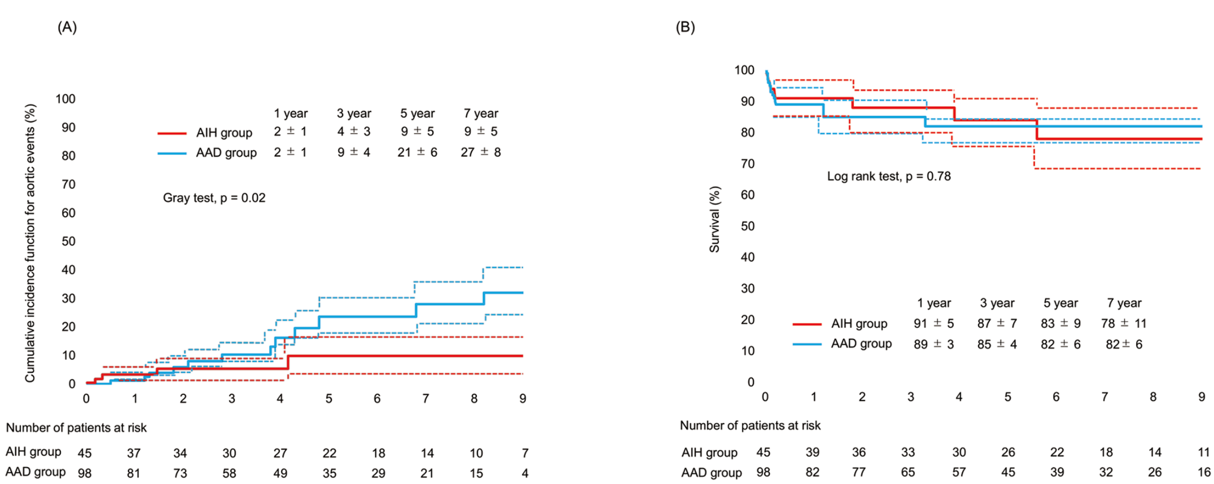

Figure 5. (**A**) Cumulative incidence function for aortic events and (**B**) Kaplan-Meier survival estimates. AAD, acute aortic dissection; AIH, acute intramural hematoma.

maximal aortic diameter after surgery at 4 different sites of the aorta. Based on the linear mixed-effect models, no significant within-group changes occurred in the AIH group, in contrast to the AAD group in which aortic diameter continued to increase at all 4 sites (within-group comparisons). Between-group comparison of the AIH and AAD groups was statistically significant at all locations.

Long-Term Clinical Outcomes

In the AIH group 1 patient underwent aortic surgery during the follow-up period: ascending aorta replacement for pseudoaneurysm formation of the proximal anastomosis. No aortic-related deaths were reported in the AIH group. On the other hand, 5 patients required aortic surgery in the AAD group: arch replacement in 2 and descending aortic replacement in 3. Five patients refused surgery despite meeting the surgical indication (enlargement of the arch or descending aorta >55 mm in diameter). Aortic-related death occurred in 3 AAD patients (rupture of the descending aorta in 2; sudden unexplained death in 1). The incidence of aortic events was significantly higher in the AAD group than in the AIH group (**Figure 5**). No significant difference was observed in the Kaplan-Meier estimated survival between groups.

Discussion

The main finding of the present study was that the AIH group had a significantly thinner OMT than the AAD group, which was significantly associated with a large amount of pericardial effusion, greater false lumen diameter, and limited progression of aortic dissection.

AIH has been classically defined by the absence of intimal tears,[1–3] but it is often difficult to confirm the lack of intimal tears on CT. In fact, as much as 36% of patients with neither identifiable intimal tears nor flow to a false lumen on preoperative CT are identified with intimal tears during surgery. In the current study, such patients were excluded to avoid mixing patients with two different pathogenic origins into 1 group. The AIH group had a significantly thinner OMT than the AAD group. No patient in the AIH group had an OMT >0.25 mm in contrast to ≈40% of patients in the AAD group who had OMT >0.25 mm. Furthermore, in some patients in the AIH group, the OMT was extremely thin (almost zero), and at first glance, it looked like an intra-adventitial hemorrhage of the vasa vasorum. The adventitia was filled with blood and thickened, as shown in **Figure 2**. Because the tissue pressure in the adventitia is lower than in the media, intra-adventitial hemorrhage seems to spread easily within this layer, which may explain why the ascending aorta had a greater false lumen diameter than the arch or descending aorta. The tendency for greater intraoperative blood loss volume in the AIH group may reflect the thinness and weakness of the vascular wall at the anastomotic sites increasing the risk of bleeding from needle holes.

The AIH group showed limited progression of aortic dissection with a lower incidence of organ malperfusion than the AAD group, a finding that concurs with previous studies.[13–15] The false lumen formed through an intimal tear is considered as directly receiving the aortic pressure that propagates between the elastic fibrous layers within the media in the long-axis direction. On the other hand, AIH may have a high risk of rupture due to the thinner and weaker aortic wall,[16–19] although the force with which the bleeding spreads longitudinally is weaker. The limited longitudinal extension of dissection in the AIH group is considered a factor in the relative absence of enlargement of the distal downstream aorta in the chronic phase.

Pericardial effusion induced by aortic dissection is a sign of aortic rupture or a result of blood extravasation through an irritated adventitia. Our finding was consistent with previous studies showing that pericardial effusion is more prevalent in AIH than in AAD.[16–18] Knowing that cardiac tamponade caused by massive pericardial effusion is one of the most common causes of death in the acute phase,[18] urgent treatment is required, especially for AIH patients with a thin OMT. Although it cannot be proved by the data presented in this study, the hypothesis that a thin outer membrane is simply prone to rupture should be examined in the future.

Study Limitations

This was a retrospective, single-center study, so a center-specific bias cannot be excluded. No data were available on thrombosis of the false lumen during the late phase. More than 90% of the patients were followed up after discharge, but the clinical course of some patients was not fully recorded. There were no data on the interval from the onset of aortic dissection to CT, which can affect the amount of pericardial effusion. Most patients underwent ascending/hemiarch replacement, which limited the range in which the intimal tear could be observed during surgery.

Conclusions

In AIH a false lumen is created at a more superficial location of the ascending aorta, closer to the adventitia, than in AAD.

Funding Sources

This work was supported, in part, by a grant-in-aid for scientific research from the Ministry of Education, Culture, Sport, Science and Technology of Japan (20K09146).

Disclosures

T.A. is a member of *Circulation Journal*'s Editorial Team.

IRB Information

The institutional review board approved the study (R2018-185. February 28th, 2019).

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