ORIGINAL RESEARCH

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Extent of Preoperative False Lumen Thrombosis Does Not Influence Long-Term Survival in Patients With Acute Type A Aortic Dissection

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***Background*—**Partial thrombosis of the false lumen has been related to aortic growth, reoperations, and death in the chronic phase of type B and repaired type A aortic dissections. The impact of preoperative false lumen thrombosis has not been studied previously. We used data from a contemporary, multinational database on aortic dissections to evaluate whether different degrees of preoperative false lumen thrombosis influenced long-term prognosis.

***Methods and Results*—**We examined the records of 522 patients with surgically treated acute type A aortic dissections who survived to discharge between 1996 and 2011. At the preoperative imaging, 414 (79.3%) patients had patent false lumens, 84 (16.1%) had partial thrombosis of the false lumen, and 24 (4.6%) had complete thrombosis of the false lumen. The annual median (interquartile range) aortic growth rates were 0.5 (−0.3 to 2.0) mm in the aortic arch, 2.0 (0.2 to 4.0) mm in the descending thoracic aorta, and similar regardless of the degree of false lumen thrombosis. The overall 5-year survival rate was 84.7%, and it was not influenced by false lumen thrombosis (P=0.86 by the log-rank test). Independent predictors of long-term mortality were age >70 years (hazard ratio [HR], 2.34; 95% confidence interval [CI], 1.20 to 4.56, P=0.012) and postoperative cerebrovascular accident, coma, and/or renal failure (HR, 2.62; 95% CI, 1.40 to 4.92, P=0.003).

***Conclusions*—**Patients with acute type A aortic dissection who survive to discharge have a favorable prognosis. Preoperative false lumen thrombosis does not influence long-term mortality, reintervention rates, or aortic growth. **(*J Am Heart Assoc.* 2013;2: e000112 doi: 10.1161/JAHA.113.000112)**

Key Words: aortic dissection • prognosis • surgery • thrombosis

Acute type A aortic dissection (AAAD) is a challenging clinical emergency. Despite continuous improvements in diagnosis, surgical treatment, and perioperative management, the latest report from the International Registry of Acute Aortic Dissection (IRAD) revealed an in-hospital surgical mortality of 23.9%.[1] This is similar to that found in a more recent study from another large registry,[2] while several single-center series report mortality well below 10%.[3–5] Patients who survive to discharge have reasonable intermediate and long-term survival rates.[6,7]

Several reports[8–12] have indicated that patients with a residual patent false lumen following an AAAD repair have an increased risk of distal aortic enlargement and death. In addition, the IRAD data have shown that in patients with acute type B aortic dissections, partial thrombosis, more than a completely patent false lumen, predicts a higher follow-up mortality.[13] As an extension of this observation, Song et al[14] found that partial thrombosis of the false lumen after an extensive Stanford type A (DeBakey type I) aortic dissection repair is a predictor of aortic enlargement, aorta-related procedures, and poor long-term survival. Different factors have been proposed to account for this increased risk, and one possible mechanism could be that the thrombus itself is a risk factor by enhancing the coagulation system.[15]

In this context, we anticipated that a partial thrombosis of the false lumen observed at the first hospitalization would negatively affect the remodeling of the distal aorta, increase

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the need for reintervention, and negatively influence the long-term survival of AAAD patients. To assess this hypothesis, in the IRAD database, we analyzed the patients with surgically treated AAAD who were discharged alive from their primary hospitalization.

Methods

IRAD Registry

IRAD is a multinational registry that collects consecutive and unselected cases of acute aortic dissection at 30 aortic centers in 10 countries. Participation in the registry does not per se imply treatment standardization. Further details about the IRAD structure and data collection have been previously published.[16] The study was approved by the institutional review board or ethics committee at each participating center.

Study Population

We screened the data records for all patients who were enrolled in IRAD between January 1, 1996 and January 31, 2011. We identified 2380 patients with an AAAD, which was defined as any nontraumatic dissection involving the ascending aorta and presenting within 14 days of symptom onset. Iatrogenic dissections were included. The patients were identified prospectively at presentation or retrospectively from discharge diagnoses. The diagnosis was based on imaging, intraoperative findings, and/or autopsy.

The patients who were exclusively managed medically, had intramural hematomas, died during the index hospitalization, or for whom follow-up or information on false lumen status was lacking were excluded from the analysis (Figure 1). Our final study population included 522 patients. Of these patients, 414 (79.3%) had a patent, 84 (16.1%) a partially thrombosed, and 24 (4.6%) a completely thrombosed false lumen.

Data Collection

A standardized form with 290 variables was used at all IRAD centers to describe the index hospital stay. This form included patient demographics, past medical histories, presenting symptoms, physical findings, imaging results, treatments, and outcomes (including complications and mortality).

All of the patients underwent computed tomography, magnetic resonance imaging, angiography, and/or echocardiography. The false lumen was classified as patent when flow was present without evidence of thrombus at any level of the aorta, as partially thrombosed when both flow and thrombus were present, and completely thrombosed in the absence of flow in the false lumen at any aortic level. Intramural

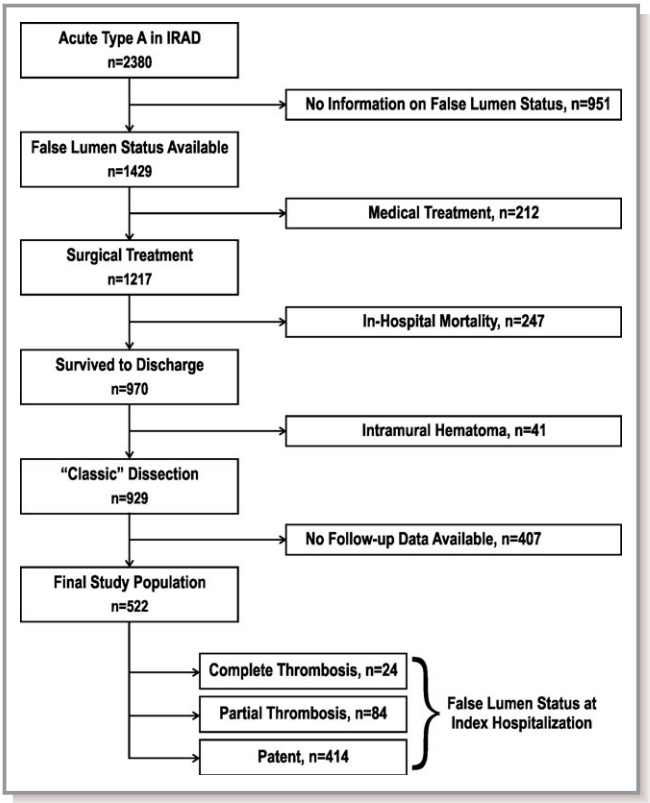

Figure 1. Study inclusion flow-chart. IRAD indicates International Registry of Acute Aortic Dissection.

hematoma was defined as an aortic wall hematoma without an intimal flap or tear on any imaging study. The distinction between an intramural hematoma and a classic double-barrel dissection with complete thrombosis of the false lumen was determined by experts at each IRAD center.

Follow-up data were obtained annually for up to 5 years using a standardized form to record clinical variables, imaging data, reoperations (including endovascular and/or open repair), and mortality.

Aortic Growth Analysis

The growth rate was calculated for the aortic arch and the descending thoracic aorta. Aortic size was measured as the largest transversal diameter perpendicular to the flow axis for each segment. Patients who underwent arch replacement at the index hospitalization were only included in the growth calculations for the descending thoracic aorta. For patients who underwent reintervention during follow-up, the latest diameter measure before reintervention was used. The growth rate was calculated as the difference between the most recent diameter measure and the diameter at the index hospitalization at the same aortic level, divided by the time interval between the 2 measurements and expressed as millimeters per year.

Statistical Analysis

Based on preoperative imaging at the index hospitalization, the patients were stratified into 3 groups according to the status of the false lumen: patent, partial thrombosis, and complete thrombosis. The clinical characteristics of each group are presented as frequencies and percentages for the categorical variables and as mean±standard deviation (SD) for the continuous variables. In cases of skewed distributions, the continuous variables are presented as the median and interquartile range (Q1 to Q3, the range between the 25th and the 75th percentile). For categorical variables, between-group differences were analyzed using the chi-squared or 2-sided Fisher's exact test, as appropriate. Continuous variables were compared with an analysis of variance or a Kruskal–Wallis test for data with skewed distributions. Missing data were not defaulted to negative and denominators reflect only those cases reported.

Univariate associations between clinical variables and mortality were calculated using a Cox regression analysis. Independent predictors of mortality were determined using a stepwise Cox proportional-hazards analysis. The initial model used variables with a P value of <0.15 in univariate testing and also included false lumen status. A backward stepwise selection of variables (after adjusting for sex and age) was performed sequentially with a default value for inclusion set at $P<0.05$.

Kaplan–Meier curves were created for the overall patient cohort and stratified according to the false lumen status. Curves were created for survival and for freedom from major adverse events (all-cause mortality, aortic rupture, and reoperation [including endovascular repair]). Between-group differences in survival and freedom from major adverse events were analyzed using the log-rank test.

All data analyses were performed using SPSS version 20 for Windows (SPSS Inc).

Results

Baseline Characteristics, Imaging, Treatment, and Complications

The mean age (±SD) of the 522 patients was 57.9±13.6 years, and 19.6% were aged >70 years (Table 1). The majority of patients (74.9%) were male and had a history of hypertension (70.9%). Other common comorbidities were atherosclerosis (19.1%), previous aortic aneurysm (11.7%), previous open-heart surgery (8.8%), and cardiac catheterization or percutaneous coronary intervention (7.8%). Marfan syndrome was present in 5.8% of the patients, and 30.0% were current smokers. Diabetes mellitus was rare in this patient cohort (4.9%), as were chronic renal insufficiency (3.6%) and chronic obstructive pulmonary disease (4.3%).

Almost a quarter of patients presented with hypotension, shock, or cardiac tamponade. Chest pain was common (85.3%); 15.0% of the patients presented with neurologic deficits, and 28.9% had a pulse deficit.

The mean number (±SD) of imaging studies per patient was 1.8±0.7, with a median (Q1 to Q3) number of studies of 2.0 (1.0 to 3.0). The most frequent procedure was computed tomography, which was performed in 81.0% of the patients, and 78.6% underwent a trans-esophageal echocardiography. Arch vessel involvement occurred in 40.3% of the patients. About one-third of the dissections were confined to the ascending aorta and aortic arch.

Complete arch replacement was performed in 12.3% of cases, and the aortic valve was replaced in 29.9% of patients (Table 2). Deliberate interruption of systemic perfusion was used in 86.0%, with a median (Q1 to Q3) circulatory arrest time of 44 (28 to 97) minutes.

Neurologic complications occurred in one quarter of the patients, acute renal failure in 19.3%, and limb ischemia in 13.4%. Mesenteric ischemia was rare, occurring in only 3.2% of patients.

Clinical Differences According to Preoperative False Lumen Status

Patients with a patent false lumen were on average 3 years younger than those with a partially thrombosed false lumen and 6 years younger than those with complete false lumen thrombosis. There were no differences with regard to previous medical history, presenting symptoms, or clinical findings. Patients with a patent false lumen had fewer imaging studies per patient, with a mean (±SD) of 1.7±0.6, versus 2.0±0.7 for those with partial thrombosis and 2.0±0.8 for those with complete thrombosis ($P=0.006$). Computed tomography was performed more frequently in the patients with partial thrombosis of the false lumen (90.5%) compared to the patients with complete patency or complete thrombosis of the false lumen (79.4% or 75.0%, respectively, $P=0.034$). The distal extension of the dissection was similar between the groups; however, extension merely to the aortic arch occurred more frequently in the partial thrombosis and complete thrombosis groups than in the patent false lumen group (31.8%, 37.5% and 17.1%, respectively, $P=0.005$). The surgical strategy did not differ between the groups.

Aortic Growth and Long-Term Outcome

The median aortic growth rate (Q1 to Q3) was 0.5 (−0.3 to 2.0) mm/year in the aortic arch and 2.0 (0.2 to 4.0) mm/year in the descending thoracic aorta (Table 3). Aortic growth was similar regardless of the degree of preoperative false lumen thrombosis.

Table 1. Patient Characteristics Stratified by False Lumen Status

	All Patients (n=522)	Status of the False Lumen			*P* Value
		Patent (n=414)	Partial Thrombosis (n=84)	Complete Thrombosis (n=24)	
Baseline patient characteristics					
Age, mean±SD	57.9±13.6	57.0±13.6	60.8±13.4	63.3±12.6	0.009*
Age ≥70 years, no./total no. (%)	102/521 (19.6)	73/413 (17.7)	20/84 (23.8)	9/24 (37.5)	0.036
Female gender, no./total no. (%)	131/522 (25.1)	96/414 (23.2)	26/84 (31.0)	9/24 (37.5)	0.12
Marfan syndrome, no./total no. (%)	30/514 (5.8)	25/407 (6.1)	4/83 (4.8)	1/24 (4.2)	0.93
Hypertension, no./total no. (%)	365/515 (70.9)	289/410 (70.5)	60/82 (73.2)	16/23 (69.6)	0.88
Atherosclerosis, no./total no. (%)†	98/513 (19.1)	82/406 (20.2)	10/83 (12.0)	6/24 (25.0)	0.16
Previous aortic dissection, no./total no. (%)	17/512 (3.3)	11/406 (2.7)	4/83 (4.8)	2/23 (8.7)	0.15
Previous aortic aneurysm, no./total no. (%)	60/513 (11.7)	52/407 (12.8)	5/82 (6.1)	3/24 (12.5)	0.21
Current smoking, no./total no. (%)‡	36/120 (30.0)	28/89 (31.5)	7/27 (25.9)	1/4 (25.0)	0.85
Diabetes mellitus, no./total no. (%)	25/513 (4.9)	19/407 (4.7)	5/82 (6.1)	1/24 (4.2)	0.84
COPD, no./total no. (%)	6/140 (4.3)	3/106 (2.8)	3/30 (10.0)	0/4 (0.0)	0.26
Chronic renal insufficiency, no./total no. (%)	5/140 (3.6)	4/106 (3.8)	1/30 (3.3)	0/4 (0.0)	1.00
Previous invasive cardiac procedures					
Open heart surgery, no./total no. (%)	44/499 (8.8)	33/396 (8.3)	8/81 (9.9)	3/22 (13.6)	0.53
Catheterization and/or PCI, no./total no. (%)	32/409 (7.8)	25/326 (7.7)	6/65 (9.2)	1/18 (5.6)	0.86
Clinical presentation					
Abrupt onset of pain, no./total no. (%)	439/498 (88.2)	350/395 (88.6)	67/79 (84.8)	22/24 (91.7)	0.60
Chest pain, no./total no. (%)	434/509 (85.3)	338/402 (84.1)	73/83 (88.0)	23/24 (95.8)	0.25
Migrating pain, no./total no. (%)§	72/478 (15.1)	57/380 (15.0)	12/78 (15.4)	3/20 (15.0)	1.00
Hypotension/shock/tamponade, no./total no. (%)¶	118/509 (23.2)	94/404 (23.3)	18/81 (22.2)	6/24 (25.0)	0.96
First systolic blood pressure (mm Hg), mean±SD	131.8±37.6	132.1±38.2	130.0±36.3	133.4±33.5	0.89
First diastolic blood pressure (mm Hg), mean±SD	73.0±22.0	72.9±21.8	72.7±23.9	74.7±19.3	0.93
Any pulse deficit, no./total no. (%)‖	122/422 (28.9)	102/338 (30.2)	18/68 (26.5)	2/16 (12.5)	0.30
Any neurologic deficit, no./total no. (%)**	77/513 (15.0)	65/405 (16.0)	10/84 (11.9)	2/24 (8.3)	0.49
Abnormal ECG, no./total no. (%)††	311/495 (62.8)	238/388 (61.3)	56/83 (67.5)	17/24 (70.8)	0.41
Diagnostic imaging					
Number of studies per patient, mean±SD	1.8±0.7	1.7±0.6	2.0±0.7	2.0±0.8	0.006
Computed tomography, no./total no. (%)	421/520 (81.0)	327/412 (79.4)	76/84 (90.5)	18/24 (75.0)	0.034
Magnetic resonance imaging, no./total no. (%)	25/492 (5.1)	15/391 (3.8)	8/77 (10.4)	2/24 (8.3)	0.037
Trans-esophageal echocardiography, no./total no. (%)	408/519 (78.6)	324/411 (78.8)	66/84 (78.6)	18/24 (75.0)	0.91
Arch vessel involvement, no./total no. (%)‡‡	183/454 (40.3)	147/357 (41.2)	30/78 (38.5)	6/19 (31.6)	0.66
Widest diameter of ascending aorta (cm), median (Q1 to Q3)§§	5.0 (4.4 to 5.8)	5.0 (4.4 to 5.6)	4.9 (4.1 to 6.0)	5.0 (4.1 to 6.1)	0.94
Widest diameter of descending aorta (cm), median (Q1 to Q3)§§	3.3 (3.0 to 3.7)	3.2 (3.0 to 3.6)	3.5 (3.0 to 4.0)	3.4 (2.9 to 4.0)	0.51

Continued

Table 1. Continued

	All Patients (n=522)	Status of the False Lumen			*P* Value
		Patent (n=414)	Partial Thrombosis (n=84)	Complete Thrombosis (n=24)	
Most distal extension of dissection					
Ascending aorta, no./total no. (%)	59/392 (15.1)	47/310 (15.2)	10/66 (15.2)	2/16 (12.5)	1.00
Aortic arch, no./total no. (%)	80/392 (20.4)	53/310 (17.1)	21/66 (31.8)	6/16 (37.5)	0.005
Left subclavian level, no./total no. (%)	22/392 (5.6)	19/310 (6.1)	3/66 (4.5)	0/16 (0.0)	0.74
Descending thoracic aorta, no./total no. (%)	103/392 (26.3)	86/310 (27.7)	12/66 (18.2)	5/16 (31.2)	0.24
Abdominal aorta, no./total no. (%)	128/392 (32.7)	105/310 (33.9)	20/66 (30.3)	3/16 (18.8)	0.41

SD indicates standard deviation; COPD, chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; ECG, echocardiogram; ANOVA, analysis of variance.
*Analyzed using ANOVA. Independent T-test between groups: Patent vs partial thrombosis, *P*=0.019. Patent vs complete thrombosis, *P*=0.026. Partial vs complete thrombosis, *P*=0.411.
†Any history of PCI, coronary artery bypass graft surgery, or catheterization demonstrating >70% stenosis in coronary, cerebral, or peripheral vasculature.
‡Tobacco use during the last month.
§Pain changed location.
¶Hypotension defined as systolic blood pressure <100 mm Hg.
‖Diminution or absence of pulse in either right or left carotid, brachial or femoral arteries.
**Paraparesis, paraplegia, stroke, or coma.
††Showing signs of old or new infarction, nonspecific ST-T segment changes, left ventricular hypertrophy or low voltage.
‡‡Any imaging modality showing dissection extending into the brachiocephalic trunk, left common carotid artery or left subclavian artery.
§§Q1 to Q3 denotes interquartile range.

On univariate testing, age, preexisting renal failure and aortic aneurysm, presenting with chest or back pain, and the composite of postoperative cerebrovascular accident, coma, or renal failure were significantly associated with death during follow-up (Table 4). In the multiple regression models, only age and the composite of postoperative cerebrovascular accident, coma, or renal failure were statistically significant (Table 5). The preoperative status of the false lumen did not predict death after discharge in the univariate testing or when adjusted for age and gender.

Table 2. Surgical Treatment and In-Hospital Complications

	All Patients (n=522)	Status of the False Lumen			*P* Value
		Patent (n=414)	Partial Thrombosis (n=84)	Complete Thrombosis (n=24)	
Surgical treatment					
Complete arch replacement, no./total no. (%)	60/489 (12.3)	42/385 (10.9)	14/81 (17.3)	4/23 (17.4)	0.20
Descending aortic replacement, no./total no. (%)*	8/490 (1.6)	5/387 (1.3)	2/80 (2.5)	1/23 (4.3)	0.17
Aortic valve replacement, no./total no. (%)	145/485 (29.9)	124/384 (32.3)	16/78 (20.5)	5/23 (21.7)	0.080
Hypothermic circulatory arrest					
HCA used, no./total no. (%)	430/500 (86.0)	342/393 (87.0)	69/84 (82.1)	19/23 (82.6)	0.39
HCA duration (minutes), median (Q1 to Q3)†	44 (28 to 97)	44 (28 to 100)	42 (25 to 80)	46 (33 to 87)	0.52
In-hospital complications (pre- and postoperative)					
Neurologic deficit, no./total no. (%)‡	125/501 (25.0)	98/397 (24.7)	21/81 (25.9)	6/23 (26.1)	0.98
Mesenteric ischemia or infarction, no./total no. (%)	16/496 (3.2)	15/393 (3.8)	1/80 (1.2)	0/23 (0.0)	0.57
Acute renal failure, no./total no. (%)§	97/502 (19.3)	76/397 (19.1)	18/81 (22.2)	3/24 (12.5)	0.60
Limb ischemia, no./total no. (%)	67/499 (13.4)	55/395 (13.9)	11/80 (13.8)	1/24 (4.2)	0.48

HCA indicates hypothermic circulatory arrest.
*Replacement of at least part of the aorta between the left subclavian level and the diaphragm.
†Q1 to Q3 denotes interquartile range.
‡Stroke, coma, or spinal cord ischemia.
§Three-fold increase in serum creatinine, 75% reduction in glomerular filtration rate, serum creatinine ≥354 μmol/L, urine output <0.3 mL/kg per hour over 24 hours or anuria for ≥12 hours.

Table 3. Aortic Growth Rates (mm/year).

	All Patients (n=522)	Status of the False Lumen			*P* Value
		Patent (n=414)	Partial Thrombosis (n=84)	Complete Thrombosis (n=24)	
Aortic arch, median (Q1 to Q3)	0.5 (−0.3 to 2.0)	0.5 (−0.5 to 1.8)	1.7 (0.0 to 4.2)	0.3 (−1.0 to 9.2)	0.24
Descending thoracic aorta, median (Q1 to Q3)	2.0 (0.2 to 4.0)	1.8 (0.4 to 4.0)	2.1 (−0.1 to 4.9)	0.3 (−0.5 to 3.4)	0.29

Q1 to Q3 denotes interquartile range.

Table 4. Univariate Predictors of Long-Term Mortality

Variable	Hazard Ratio	95% Confidence Interval	*P* Value
Age	1.04	1.02 to 1.07	0.001
Age ≥70 years	2.96	1.65 to 5.31	<0.001
Female gender	1.24	0.66 to 2.35	0.51
Atherosclerosis*	1.48	0.78 to 2.82	0.23
Patent false lumen†	1.00		
Partially thrombosed false lumen	0.80	0.34 to 1.88	0.60
Completely thrombosed false lumen	0.87	0.21 to 3.61	0.85
Postoperative CVA, coma and/or acute renal failure‡	2.73	1.46 to 5.11	0.002
Postoperative spinal cord ischemia	0.93	0.13 to 6.80	0.95
Presenting diameter ascending aorta	1.02	0.80 to 1.31	0.86
Presenting diameter descending aorta	1.20	0.77 to 1.87	0.42
Chronic renal insufficiency	7.00	1.40 to 34.87	0.018
Previous aortic aneurysm	2.31	1.15 to 4.66	0.019
Peripheral artery disease	6.23	0.75 to 51.88	0.091
Postoperative mesenteric ischemia/infarction	5.66	0.77 to 41.77	0.089
Presenting syncope	0.50	0.20 to 1.27	0.14
Presenting CVA	1.87	0.67 to 5.23	0.23
Surgery on descending aorta	3.02	0.73 to 12.46	0.13
Presenting chest or back pain	0.44	0.21 to 0.92	0.029

CVA indicates cerebrovascular accident.
*Any history of percutaneous coronary intervention, coronary artery bypass graft surgery or catheterization demonstrating > 70% stenosis in coronary, cerebral or peripheral vasculature.
†Patent false lumen is the reference group.
‡Three-fold increase in serum creatinine, 75% reduction in glomerular filtration rate, serum creatinine ≥354 μmol/L, urine output <0.3 mL/kg per hour over 24 hours or anuria for ≥12 hours.

The Kaplan–Meier curves showed an overall 5-year survival of 84.7% (95% CI; 79.6% to 88.6%; Figure 2). The extent of preoperative false lumen thrombosis did not affect the 5-year survival rates (Figure 3). Nor did thrombosis of the false lumen preoperatively affect freedom of major adverse events (Figure 4).

Table 5. Independent Predictors of Long-Term Mortality After Multivariate Model Adjustments

Variable	Hazard Ratio	95% Confidence Interval	*P* Value
Female gender	0.90	0.43 to 1.87	0.78
Age ≥70 years	2.34	1.20 to 4.56	0.012
Patent false lumen*	1.00		
Partial thrombosis false lumen	0.78	0.30 to 1.99	0.60
Complete thrombosis false lumen	0.81	0.19 to 3.44	0.78
Postoperative CVA, coma and/or renal failure†	2.62	1.40 to 4.92	0.003

CVA indicates cerebrovascular accident.
*Patent false lumen is the reference group.
†Three-fold increase in serum creatinine, 75% reduction in glomerular filtration rate, serum creatinine ≥354 μmol/L, urine output <0.3 mL/kg per hour over 24 hours or anuria for ≥12 hours.

Discussion

The data included in the present analysis reject the hypothesis that a partially thrombosed false lumen on preoperative imaging was a predictor of an ominous clinical course in patients with surgically treated AAAD.

To our knowledge, the relationship between partial thrombosis of the false lumen and long-term outcome in patients with aortic dissection has been examined in 7 studies.[13–15,17–20] All of these studies have included patients with either type B dissections, postoperative AAAD, or a combination of both. The results have been divergent. Partial thrombosis was identified as an independent predictor for aortic enlargement, aortic-related procedures, or death in 2 studies.[13,14] In the remaining 5 studies,[15,17–20] partial thrombosis was not associated with a worse outcome, faster aortic growth, or higher incidence of aneurysm development compared to complete patency of the false lumen. However, in the

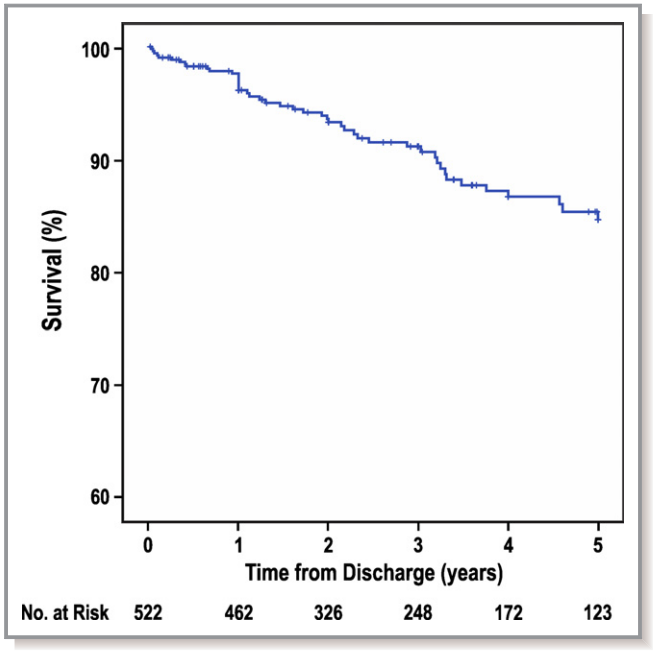

Figure 2. Kaplan–Meier postdischarge survival curve for all patients.

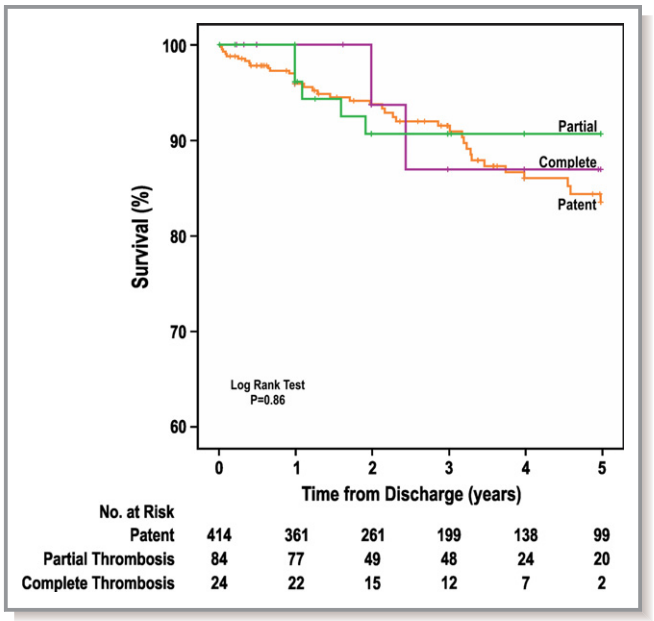

Figure 3. Kaplan–Meier postdischarge survival curves stratified according to the false lumen status.

study by Sueyoshi et al,[15] which compared aortic enlargement across different degrees of false lumen thrombosis in type B dissections, a subset within the partial thrombosis group with a blind pouch in the false lumen (ie, thrombosis covering the potential reentry site) had considerably faster growth rates. This group was small, only accounting for 15% of the patients with partial thrombosis of the false lumen.

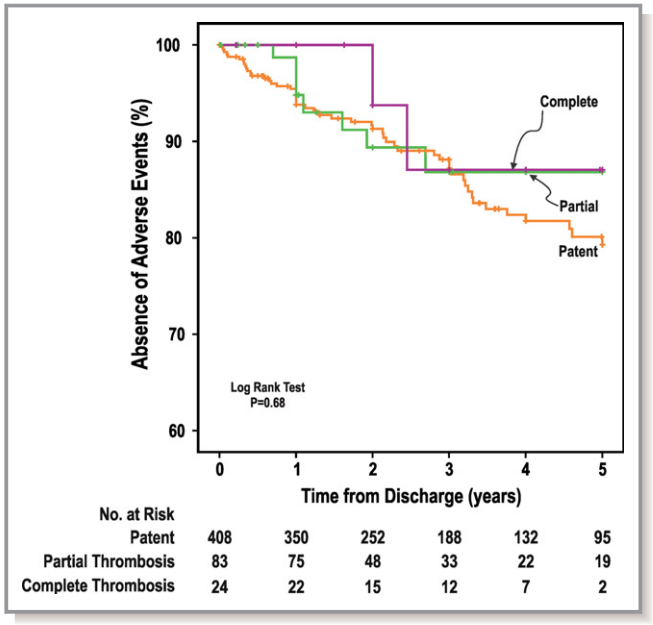

Figure 4. Kaplan–Meier curves of postdischarge freedom from major adverse events (all-cause mortality, aortic rupture and reoperation, including endovascular repair), stratified according to the false lumen status.

Results from previous studies on type B dissections and postoperative AAAD cannot readily be extrapolated to our data, as we examined the influence of preoperative false lumen thrombosis in AAAD. In addition, the fact that partial thrombosis of the false lumen seems to be unfavorable in type B dissections does not necessarily imply that this is the case in postoperative AAAD. The notion that an operated AAAD with a persistent false lumen in the descending aorta mirrors a type B dissection is not intuitive. In AAAD, the primary entry tear is typically located in the ascending aorta, whereas the area just distal to the subclavian artery is the predilection site in type B dissections.[21,22] This region is also most prone to dilation and rupture in the chronic phase.[20] By surgically removing the primary entry tear, the pathological process of an AAAD is fundamentally changed. Residual intimal tears, which maintain flow in the false lumen and remain distal after type A dissection repair, might have a different impact on flow and pressure dynamics compared to that associated with the primary entry in type B dissections. In fact, a residual primary entry tear independently predicts the need for reintervention in patients with operated AAAD only.[17] Moreover, patients with a patent false lumen distally to AAAD repair have better outcomes compared to type B dissections, which is potentially related to the size and location of intimal tears.[23] It is also to be noted, that the one study supporting partial thrombosis as a negative predictor in surgically treated AAAD[14] is based on 27 patients with a particularly high mortality of 60% at 5 years.

We observed that the degree of false lumen thrombosis appeared to increase with age. It is unknown why older

patients are more likely to have spontaneous complete thrombosis of the false lumen, but blood coagulability increases with age.[24] The group with complete thrombosis was small: 24 patients (4.6% of the total patient cohort). Traditionally, complete thrombosis of the false lumen has been thought to be a prerequisite for healing of the aorta postdissection, as flow and pressurization of the false lumen are thought to contribute to late dilation and rupture. Accordingly, one could expect lower mortality during follow-up in this setting. Our data did not show that there was any survival benefit associated with complete false lumen thrombosis. Because of the small number of patients, even in the IRAD database, adjusted comparisons with this group lacked statistical power. Of great practical importance, however, is that there are no definitive clinical epidemiological data supporting the traditional view that a persistent blood-flow in the false lumen is a definitive negative predictor for outome.[25] Thus, an extensive procedure with arch replacement and intraoperative stentgrafting of the descending aorta[3,4] has little support as a routine procedure in AAAD. Such an extensive operation is also at odds with the acceptable overall long-term survival rate of 85% at 5 years in surgically treated AAAD in the IRAD database.

The imaging characteristics in our material warrant further attention. Patients with partial or complete thrombosis of the false lumen were subjected to more imaging studies compared to those with no thrombosis of the false lumen. It could be argued that partial or complete thrombosis of the false lumen is more likely to be diagnosed with an increasing number of different imaging modalities and that a given portion of the patients who were categorized as having patent lumens may have actually had some degree of thrombosis. Conversely, diagnosing aortic dissection itself can be challenging, more so when there is no flow in the false lumen. In the case of a "classic" aortic dissection with flow in the false lumen and a clearly visible intimal flap, the diagnosis is straightforward. When the false lumen is void of flow, establishing the diagnosis can be difficult, particularly by echocardiography. Additional imaging is required in such circumstances.

A key aspect in the interpretation of our data is that the status of the false lumen was established once (at presentation, ie, before surgery). The main goal of surgery in AAAD is to prevent lethal complications, such as rupture, cardiac tamponade, and malperfusion. A secondary goal is to resect the entry tear and redirect the blood flow to the true lumen. Resection of the primary tear and aortic reconstruction will alter flow in the false lumen and might promote thrombosis. Thus, our classification (based on aortic morphology at presentation), may be altered postoperatively. However, a distal false lumen remains patent in as many as 79% of patients following the initial repair.[9,10,26–29]

Strengths and Limitations of the Study

The main strength of the study is that it included >500 patients in an orderly, prespecified manner during a limited time period. However, IRAD is an observational registry and selection bias is possible. Furthermore, follow-up data were not available for more than half of the survivors. As a result, our data might not be representative of the entire IRAD patient population but might represent a selection from the centers with the most systematic patient registration and follow-up.

Information on preoperative false lumen status was lacking for 40% of the AAAD patients in IRAD. In cases of iatrogenic dissections that occur during elective or emergency cardiac surgery for other reasons, preoperative imaging will not be relevant. Also, when a patient with AAAD present with cardiac tamponade, cardiac arrest, or other critical conditions, preoperative imaging will in many cases be limited to a screening echocardiography, and details regarding false lumen thrombosis will not be available.

Imaging techniques may have varied among the centers. Misclassification of false lumen status was possible as the imaging data were collected and reviewed at each participating center before the start of the study and were not reevaluated in a core laboratory. Likewise, patients with a completely thrombosed false lumen might have been excluded and patients with an intramural hematoma might have been included, as this distinction can be challenging. Furthermore, traditional first-pass imaging of the aorta might have overestimated the degree of thrombosis in the false lumen.[30]

We were unable to provide cause-of-death data and the distribution of aortic-related and nonaortic-related death could be different between the groups.

Conclusions

The present study revealed that preoperative partial thrombosis of the false lumen in surgically treated AAAD in the IRAD database was not an independent risk factor for aortic enlargement, intervention, or death in the follow-up period. The AAAD survivors had favorable prognoses, but the factors that influence aortic dilation and rupture following acute aortic dissections are still incompletely understood. New imaging techniques, for example, based on flow-dynamics[31] and/or bioimaging[32,33] can hopefully improve our ability to predict an ominous outcome in patients with aortic dissection.

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Disclosures

None.

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Extent of Preoperative False Lumen Thrombosis Does Not Influence Long-Term Survival in Patients With Acute Type A Aortic Dissection

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