

ORIGINAL ARTICLE

Partial Thrombosis of the False Lumen in Patients with Acute Type B Aortic Dissection

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ABSTRACT

BACKGROUND

Patency or thrombosis of the false lumen in type B acute aortic dissection has been found to predict outcomes. The prognostic implications of partial thrombosis of the false lumen have not yet been elucidated.

METHODS

We examined 201 patients with type B acute aortic dissection who were enrolled in the International Registry of Acute Aortic Dissection between 1996 and 2003 and who survived to hospital discharge. Kaplan–Meier mortality curves were stratified according to the status of the false lumen (patent, partial thrombosis, or complete thrombosis) as determined during the index hospitalization. Cox proportional-hazards analysis was performed to identify independent predictors of death.

RESULTS

During the index hospitalization, 114 patients (56.7%) had a patent false lumen, 68 patients (33.8%) had partial thrombosis of the false lumen, and 19 (9.5%) had complete thrombosis of the false lumen. The mean (\pm SD) 3-year mortality rate for patients with a patent false lumen was $13.7\pm 7.1\%$, for those with partial thrombosis was $31.6\pm 12.4\%$, and for those with complete thrombosis was $22.6\pm 22.6\%$ (median follow-up, 2.8 years; $P=0.003$ by the log-rank test). Independent predictors of post-discharge mortality were partial thrombosis of the false lumen (relative risk, 2.69; 95% confidence interval [CI], 1.45 to 4.98; $P=0.002$), a history of aortic aneurysm (relative risk, 2.05; 95% CI, 1.07 to 3.93; $P=0.03$), and a history of atherosclerosis (relative risk, 1.87; 95% CI, 1.01 to 3.47; $P=0.05$).

CONCLUSIONS

Mortality is high after discharge from the hospital among patients with type B acute aortic dissection. Partial thrombosis of the false lumen, as compared with complete patency, is a significant independent predictor of postdischarge mortality in these patients.

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ACUTE AORTIC DISSECTION IS A DANGEROUS condition with high in-hospital and follow-up mortality rates. Dissections confined to the descending aorta (type B) have better in-hospital survival than those involving the ascending aorta (type A). Up to 89% of patients with uncomplicated type B dissections survive to hospital discharge after receiving effective antihypertensive therapy.¹ However, despite a low in-hospital mortality, the short- and long-term prognosis of patients with type B acute aortic dissection after discharge from the hospital is heterogeneous, with reported survival rates ranging from 56 to 92% at 1 year and from 48 to 82% at 5 years.²⁻⁹

Given the variable prognosis of type B acute aortic dissection with current management strategies, predictors of poor outcomes have been sought. In addition to aortic diameter, a reported predictor of outcomes in type B acute aortic dissection has been the patency of the false lumen.^{3,10,11} Studies have suggested that patients with complete thrombosis of the false lumen have improved outcomes, whereas those with a patent false lumen have an increased risk of aortic expansion and death.^{3,10,11} To our knowledge, partial thrombosis of the false lumen, defined as the concurrent presence of both flow and thrombus, has not been studied. The purpose of this analysis was to evaluate the incidence of partial thrombosis of the false lumen on cross-sectional imaging and to assess its effect on mortality in patients presenting with type B acute aortic dissection.

METHODS

The International Registry of Acute Aortic Dissection (IRAD) is a multinational registry of patients with acute aortic dissection evaluated at 22 aortic centers in 11 countries. The registry is supported by grants and receives no commercial funding. Treatment during the index hospitalization is not standardized but is conducted at the discretion of each patient's treating physician. Full details of the IRAD structure and the methods used have been previously published.^{1,12}

The registry was approved by the institutional review board or ethics committee at each participating center, and a waiver of informed consent for retrospective chart review was granted for the registry. Individual written informed consent was obtained for the follow-up study.

STUDY POPULATION

We examined data from all patients with type B acute aortic dissection enrolled in IRAD between January 1, 1996, and December 31, 2003. Type B acute aortic dissection was defined as any non-traumatic dissection not involving the ascending aorta and presenting within 14 days of symptom onset.^{13,14} Patients were identified prospectively at presentation or retrospectively from discharge diagnoses and from imaging and surgical databases. Diagnosis was based on confirmatory imaging, intraoperative visualization, or autopsy.

Of the 532 patients enrolled in IRAD with type B acute aortic dissection, 466 were discharged from the hospital alive. Postdischarge mortality data were available for 342 patients. Of these, 141 were excluded from our study, including 64 with a diagnosis of intramural hematoma, 46 for whom imaging data on the false lumen were lacking, and 31 for whom consensus on the status of the false lumen on imaging was lacking. The distinction between an intramural hematoma and a true dissection with complete thrombosis of the false lumen was made by experts at local IRAD centers. Patients were considered to have an intramural hematoma if the hematoma extended outward from the lumen in a crescent shape and maintained a constant circumferential relationship with the aortic wall without a demonstrable intimal flap and with no radiologically apparent intimal tear. Our final study population included 201 patients (38% of those enrolled in the registry).

DATA COLLECTION

A standardized form was used to record clinical variables, including information on patient demographics and history, clinical presentation, physical findings, imaging results, medical and surgical treatment, and outcomes, including mortality. The data forms were forwarded to the IRAD coordinating center at the University of Michigan, reviewed for internal consistency and face validity, and then scanned electronically into a Microsoft Access database.

The imaging results were interpreted at each patient's respective tertiary care center by experienced radiologists and echocardiographers and were entered on the data form. Each patient underwent spiral computed tomography, transesophageal echocardiography, magnetic resonance imaging, or a combination of these procedures.

The status of the false lumen on imaging was classified as patent if flow was present in the absence of thrombus, as partially thrombosed if both flow and thrombus were present, or as completely thrombosed if no flow was present.

Yearly follow-up data were obtained with the use of standardized forms after the patient was discharged. We obtained clinical and imaging data as well as information about mortality, with the date of death when known. When applicable, missing data on mortality were obtained from the Social Security Death Index.¹⁵

STATISTICAL ANALYSIS

Three comparison groups were created on the basis of the status of the false lumen: patent, partial thrombosis, or complete thrombosis. The clinical characteristics of each of the three groups were presented as frequencies and percentages for categorical variables and as means \pm SD for continuous variables. Univariate differences among the three groups were compared by the chi-square test for categorical variables and by analysis of variance for continuous variables.

Univariate associations between all clinical variables, including false-lumen status, and post-discharge mortality were calculated by Cox regression analysis. No imputation of missing variables was performed. Stepwise Cox proportional-hazards analysis was performed to identify independent predictors of postdischarge mortality. The initial modeling used variables marginally suggestive of an unadjusted association with mortality ($P < 0.20$). Variables were reviewed for clinical significance before testing. Forward-ascending stepwise selection of variables after adjustment for age, sex, and in-hospital treatment (medical, surgical, or endovascular) was performed sequentially, with a default value for inclusion set at $P < 0.05$. SAS software, version 8.2, was used for all analyses.

RESULTS

BASELINE AND IMAGING CHARACTERISTICS

The mean age (\pm SD) of the 201 patients examined in this analysis was 60.8 ± 13.9 years (Table 1). The majority of patients (69.1%) were male, 28.4% were 70 years of age or older, and 77.0% had a history of hypertension. Other coexisting conditions, such as atherosclerosis (29.8%), prior aortic

aneurysm (21.0%), and prior aortic dissection (11.7%), were not uncommon. In comparison with the study population, patients in the IRAD database who were not included in the analysis were older (65.9 ± 12.6 years), more likely to have a history of atherosclerosis (39.7%), and less likely to have had a previous aortic dissection (5.4%).

In more than 90% of patients, the diagnosis of type B acute aortic dissection was confirmed by cross-sectional imaging within 1 day of presentation. The remaining cases were diagnosed within 7 days. On cross-sectional imaging, the false lumen was found to be patent in 114 patients (56.7%), partially thrombosed in 68 (33.8%), and completely thrombosed in 19 (9.5%) (Table 1). The mean number of imaging studies performed per patient was 1.5; the most frequent procedure was computed tomography, which was performed in three quarters of the patients. There were no significant differences in the frequency of performance of different imaging procedures between patients with patent, partially thrombosed, and completely thrombosed false lumens.

CLINICAL FEATURES ASSOCIATED WITH STATUS OF THE FALSE LUMEN

Patients with complete thrombosis of the false lumen were significantly older than those in the other two false-lumen groups, with a mean age of 70.9 ± 12.1 years, versus 57.6 ± 13.5 years in patients with a patent false lumen and 63.3 ± 13.5 years in patients with partial thrombosis of the false lumen ($P < 0.001$). There were no significant differences among the three groups in symptoms or physical findings at presentation (Table 1). With regard to diagnostic testing, patients with complete thrombosis of the false lumen were more likely to have abnormal electrocardiograms and pleural effusions on chest radiography than patients with a patent or partially thrombosed false lumen.

IN-HOSPITAL TREATMENT AND OUTCOMES

All patients initially received medical therapy to regulate systolic blood pressure and the velocity of left ventricular ejection (change in pressure \div change in time [dP/dt]). One hundred forty-six patients (72.6%) received medical therapy only, 36 (17.9%) underwent surgery, and 19 (9.5%) had endovascular treatment, defined as stent placement, fenestration, or both (Table 1). There were

Table 1. Characteristics of Patients Stratified According to the Status of the False Lumen.*

Characteristic	All Patients (N=201)	Status of the False Lumen		P Value
		Patent (N=114)	Complete Thrombosis (N=19)	
Age — yr	60.8±13.9	57.6±13.5	70.9±12.1	<0.001
Age ≥70 yr — no. (%)	57 (28.4)	23 (20.2)	12 (63.2)	<0.001
Female sex — no. (%)	62 (30.8)	30 (26.3)	7 (36.8)	0.28
White race — no./total no. (%)†	159/188 (84.6)	95/109 (87.2)	14/17 (82.4)	0.12
Marfan's syndrome — no./total no. (%)	11/199 (5.5)	8/113 (7.1)	3/68 (4.4)	0.42
Hypertension — no./total no. (%)	154/200 (77.0)	85/113 (75.2)	17/19 (89.5)	0.39
Atherosclerosis — no./total no. (%)‡	59/198 (29.8)	26/112 (23.2)	8/18 (44.4)	0.06
Previous aortic dissection — no./total no. (%)	23/197 (11.7)	17/111 (15.3)	1/18 (5.6)	0.19
Previous aortic aneurysm — no./total no. (%)§	41/195 (21.0)	21/108 (19.4)	4/19 (21.1)	0.81
Diabetes — no./total no. (%)	13/197 (6.6)	6/111 (5.4)	3/18 (16.7)	0.20
Previous cardiovascular surgery — no./total no. (%)	38/191 (19.9)	24/106 (22.6)	4/17 (23.5)	0.41
Clinical presentation				
Chest pain — no./total no. (%)	137/198 (69.2)	79/113 (69.9)	10/17 (58.8)	0.62
Back pain — no./total no. (%)	136/196 (69.4)	75/111 (67.6)	12/18 (66.7)	0.71
Abrupt onset of pain — no./total no. (%)	167/193 (86.5)	100/111 (90.1)	14/17 (82.4)	0.24
Migrating pain — no./total no. (%)	38/188 (20.2)	24/109 (22.0)	2/15 (13.3)	0.69
Any neurologic deficit — no. (%)	19 (9.5)	11 (9.7)	3 (15.8)	0.54
Systolic blood pressure — mm Hg	170.5±36.3	169.5±37.1	162.4±30.9	0.42
Diastolic blood pressure — mm Hg	94.8±20.7	94.7±20.9	87.8±19.3	0.27
Hypotension or shock — no./total no. (%)	4/194 (2.1)	2/111 (1.8)	1/18 (5.6)	0.55
Hypertension — no./total no. (%)	144/196 (73.5)	78/112 (69.6)	14/18 (77.8)	0.37
Any pulse deficit — no./total no. (%)	37/185 (20.0)	21/105 (20.0)	3/17 (17.6)	0.96
Diagnostic imaging				
Chest radiograph — no./total no. (%)				
Normal	33/188 (17.6)	19/106 (17.9)	2/16 (12.5)	0.86
Widened mediastinum	85/184 (46.2)	43/104 (41.3)	9/16 (56.2)	0.31
Abnormal aortic contour	93/182 (51.1)	49/104 (47.1)	10/15 (66.7)	0.31
Pleural effusion	25/181 (13.8)	15/103 (14.6)	5/14 (35.7)	0.02

Electrocardiogram — no./total no. (%)							
Abnormal	135/195 (69.2)	68/111 (61.3)	50/67 (74.6)	17/17 (100.0)	0.003		
Old Q wave	14/179 (7.8)	6/101 (5.9)	6/63 (9.5)	2/15 (13.3)	0.50		
New Q wave, ST elevations, or ischemia	23/185 (12.4)	11/103 (10.7)	10/65 (15.4)	2/17 (11.8)	0.66		
Nonspecific ST or T wave changes	73/186 (39.2)	33/105 (31.4)	29/64 (45.3)	11/17 (64.7)	0.02		
No. of studies performed per patient	1.5±0.7	1.5±0.6	1.5±0.8	1.4±0.7	0.84		
Computed tomography — no. (%)	151 (75.1)	85 (74.6)	49 (72.1)	17 (89.5)	0.29		
Transesophageal echocardiography — no. (%)	78 (38.8)	44 (38.6)	27 (39.7)	7 (36.8)	0.97		
Magnetic resonance imaging — no. (%)	37 (18.4)	18 (15.8)	16 (23.5)	3 (15.8)	0.41		
Widest diameter of descending aorta — cm	4.5±1.2	4.5±1.2	4.7±1.1	4.1±0.8	0.15		
Widest diameter of ascending aorta ≥6 cm — no./total no. (%)	4/115 (3.5)	1/66 (1.5)	2/38 (5.3)	1/11 (9.1)	0.34		
Treatment							
Surgical — no. (%)	36 (17.9)	24 (21.1)	9 (13.2)	3 (15.8)			
Endovascular — no. (%)	19 (9.5)	11 (9.6)	8 (11.8)	0			
Medical only — no. (%)	146 (72.6)	79 (69.3)	51 (75.0)	16 (84.2)			
In-hospital complications							
Neurologic deficit — no./total no. (%)	20/179 (11.2)	15/101 (14.9)	3/63 (4.8)	2/15 (13.3)	0.13		
Hypotension — no./total no. (%)	12/184 (6.5)	5/107 (4.7)	6/61 (9.8)	1/16 (6.2)	0.43		
Malperfusion — no./total no. (%)	45/98 (45.9)	30/58 (51.7)	14/33 (42.4)	1/7 (14.3)	0.15		
Mesenteric ischemia — no./total no. (%)	13/183 (7.1)	9/103 (8.7)	4/59 (6.8)	0/16	0.48		
Acute renal failure — no./total no. (%)	29/184 (15.8)	18/107 (16.8)	10/61 (16.4)	1/16 (6.2)	0.55		
Limb ischemia — no./total no. (%)	17/182 (9.3)	13/108 (12.0)	4/59 (6.8)	0/15	0.23		

* Plus-minus values are means ±SD.

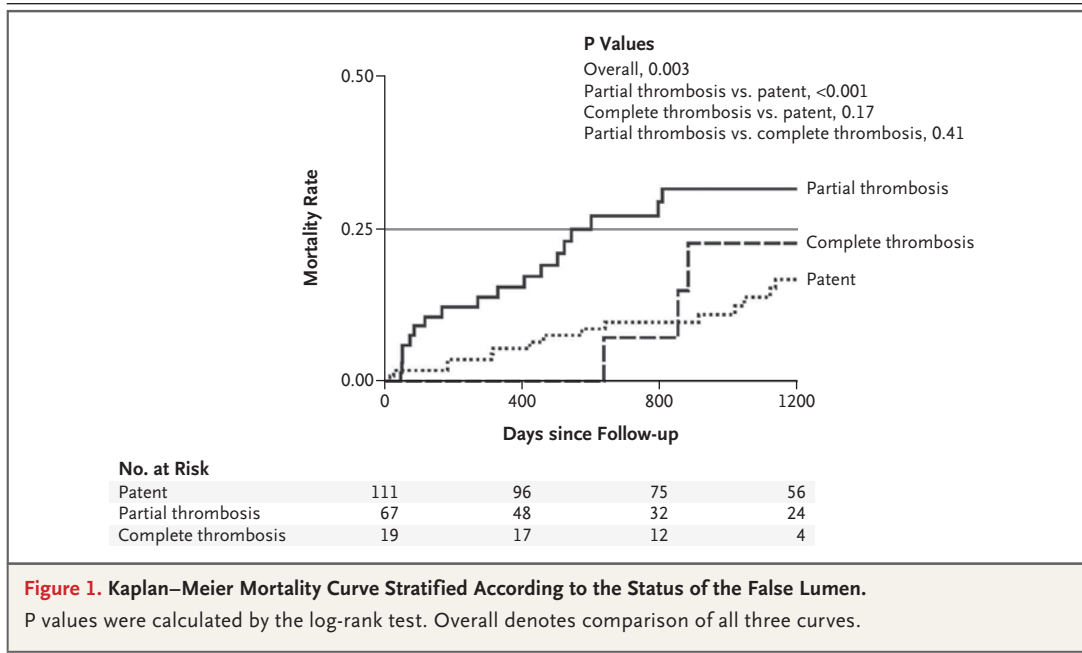
† Race was determined by the investigator.

‡ Atherosclerosis includes coronary, peripheral, and cerebrovascular disease.

§ Aortic aneurysm includes thoracic and abdominal aneurysm.

¶ The P value is for the comparison among surgical, endovascular, and medical management.

|| Endovascular treatment includes fenestration and stent placement.



no significant differences among the three false-lumen groups with regard to choice of therapy or rates of specific in-hospital complications.

The median follow-up for the 201 patients examined in this analysis was 2.8 years. Figure 1 shows the Kaplan–Meier mortality curves stratified according to false-lumen status. The mortality rate was highest in patients with partial thrombosis of the false lumen, with 1- and 3-year mortality rates of $15.4 \pm 8.8\%$ and $31.6 \pm 12.4\%$, respectively, versus $5.4 \pm 4.2\%$ and $13.7 \pm 7.1\%$ in patients with a patent false lumen and 0% and $22.6 \pm 22.6\%$ in patients with complete thrombosis of the false lumen. Separate log-rank testing revealed a significant increase in mortality in patients with partial thrombosis of the false lumen as compared with patients with a completely patent false lumen ($P < 0.001$). Log-rank testing did not reveal significant differences between patients with complete thrombosis of the false lumen and those with a completely patent false lumen ($P = 0.17$) or with partial thrombosis of the false lumen ($P = 0.41$).

In a further analysis, we included patients with intramural hematoma in the group classified as having complete thrombosis of the false lumen. The number of patients in this group was increased to 41, and the 1- and 3-year mortality rates were increased to $5.6 \pm 7.5\%$ and $32.4 \pm 19.2\%$,

respectively. However, the difference between the mortality curves according to the log-rank test did not change significantly.

PREDICTORS OF POSTDISCHARGE MORTALITY

Candidate univariate predictors of postdischarge mortality are shown in Table 2. Patients with type B acute aortic dissection who died during the follow-up period were significantly older and significantly more likely to have a history of aortic aneurysm or atherosclerosis. In addition, they were more likely to have pleural effusions on chest radiography. Independent predictors of mortality are shown in Table 3. After adjustment for age, sex, and in-hospital treatment, the key predictors of mortality were partial thrombosis of the false lumen versus a patent false lumen, a history of aortic aneurysm, and a history of atherosclerosis.

We also assessed the potential effect of surgical or endovascular therapy (both of which tended to be performed independently of false-lumen status) on the relationship between false-lumen status and survival. Thirty-six patients underwent surgery and 19 received endovascular therapy; among the remaining 146 patients, the relative risk of partial thrombosis of the false lumen remained a significant independent predictor of postdischarge mortality (relative risk, 4.01; 95% confidence interval, 1.87 to 8.64; $P < 0.001$).

Table 2. Candidate Univariate Predictors of Mortality.*

Variable	Categorical Variables		
	No. of Deaths/ No. of Patients (%)	Risk Ratio (95% CI)	P Value
Age ≥ 70 yr	20/57 (35.1)	1.77 (0.99–3.13)	0.05
Female sex	17/62 (27.4)	1.11 (0.62–2.00)	0.73
Atherosclerosis†	23/59 (39.0)	2.26 (1.29–3.96)	0.005
Previous aortic aneurysm‡	17/41 (41.5)	2.41 (1.34–4.33)	0.003
Chest pain	29/137 (21.2)	0.65 (0.37–1.14)	0.13
Pleural effusion on chest radiograph	11/25 (44.0)	2.00 (1.02–3.95)	0.04
Status of false lumen			
Patent§	20/114 (17.5)		
Partial thrombosis	25/68 (36.8)	2.67 (1.48–4.82)	0.001
Complete thrombosis	5/19 (26.3)	1.86 (0.70–4.98)	0.22
Treatment			
Medical only§	35/146 (24.0)		
Surgical	10/36 (27.8)	1.17 (0.58–2.36)	0.67
Endovascular¶	5/19 (26.3)	1.07 (0.42–2.75)	0.89
Hypotension after admission	5/12 (41.7)	1.85 (0.73–4.70)	0.19
Continuous Variables			
	Patients Who Died during Follow-up (N = 50)	Risk Ratio (95% CI)	P Value
Age — yr	64.8 \pm 14.2	1.03 (1.00–1.05)	0.02
Diameter of ascending aorta — cm	4.6 \pm 1.0	1.71 (1.22–2.39)	0.004
Diameter of descending aorta — cm	4.8 \pm 1.5	1.29 (0.97–1.73)	0.08

* CI denotes confidence interval.

† Atherosclerosis includes coronary, peripheral, and cerebrovascular disease.

‡ Aortic aneurysm includes thoracic and abdominal aneurysm.

§ Patent false lumen and medical-only treatment are the reference groups.

¶ Endovascular treatment includes fenestration and stent placement.

|| The risk ratio is per additional year or centimeter.

DISCUSSION

In this large cohort of patients with type B acute aortic dissection, mortality was high after discharge from the hospital, with nearly one in four patients (24.9%) dying within 3 years. Partial thrombosis of the false lumen was common (present in a third of patients) and was the strongest independent predictor of postdischarge mortality that we identified: the risk of death in these patients was increased by a factor of 2.7 in comparison with patients with a patent false lumen.

Previous small observational studies have suggested a lower risk of adverse events and better outcomes in patients with complete thrombosis of the false lumen than in those with a patent

false lumen.^{3,10,11,16,17} These studies have linked patency of the false lumen to adverse events resulting from aneurysmal dilatation and rupture during the chronic phase.^{5,18–20} In our study, partial thrombosis of the false lumen was defined as the concurrent presence of both flow and thrombus in the false lumen; this condition was not considered a distinct physiological state in most previous studies and has not been previously associated with increased mortality.

In our study, complete thrombosis of the false lumen occurred in a small number of patients who were, on average, 13 years older than patients with a completely patent false lumen, a finding similar to that of previous studies.^{3,19,21} Since only 19 patients (9.5%) were classified as

Table 3. Independent Predictors of Death after Adjustment with the Use of Multivariate Models.*

Variable	Hazard Ratio (95% CI)	P Value
Age \geq 70 yr	1.42 (0.74–2.74)	0.29
Female sex	1.16 (0.62–2.17)	0.64
Surgical treatment	1.33 (0.49–3.58)	0.57
Endovascular treatment†	1.38 (0.64–3.01)	0.41
Previous aortic aneurysm‡	2.05 (1.07–3.93)	0.03
Atherosclerosis§	1.87 (1.01–3.47)	0.05
Patent false lumen¶	1.00	
Partial thrombosis of the false lumen	2.69 (1.45–4.98)	0.002
Complete thrombosis of the false lumen	1.02 (0.32–3.22)	0.98

* CI denotes confidence interval.

† Endovascular treatment includes fenestration and stent placement.

‡ Aortic aneurysm includes thoracic and abdominal aneurysm.

§ Atherosclerosis includes coronary, peripheral, and cerebrovascular disease.

¶ Patent false lumen is the reference group.

having complete thrombosis of the false lumen, comparisons with this group lack statistical power. We did not find a significant difference in mortality between patients with complete thrombosis of the false lumen and patients with a completely patent false lumen. The small number of cases has also impaired the ability to draw conclusions about this group in other studies of type B acute aortic dissection.^{3,7,22-26} Only one previous study found that complete thrombosis of the false lumen was a predictor of less aortic enlargement; another study found that dissection-related mortality was lower among patients with complete thrombosis of the false lumen.^{10,19}

Determination of the mechanism by which partial thrombosis of the false lumen portends a poor outcome is beyond the scope of this observational study. However, two possible contributing factors deserve mention. One potential explanation relates the pressure within the false lumen to the presence of partial thrombosis. Whereas a patent false lumen may be perfused by a proximal entry tear and decompressed through distal reentry tears (Fig. 2A), formation of a partial thrombus may occlude these distal tears, impeding outflow and, in the most extreme situation (Fig. 2B), resulting in a blind sac. Studies have shown that pulsatile inflow into a lumen with impaired outflow may lead to a significant increase in the mean arterial and diastolic pressure as compared with that in a lumen with

adequate outflow, despite similar systolic pressure.²⁷⁻²⁹

An increase in pressure within the false lumen will increase wall tension, which may elevate the risk of aneurysm expansion, redissection, and rupture and would thus explain the increased mortality seen in these patients. Complete thrombosis of the false lumen (Fig. 2C) excludes the false lumen from the circulation and is thought to be a prerequisite for complete healing. This is the principle on which endovascular stent therapy is based.^{24,30}

Partial thrombosis may also have a role in the rupture of the false lumen similar to its role in the rupture of abdominal aortic aneurysms. Previous studies have suggested a direct relationship between intraluminal thrombosis and the risk of rupture of an abdominal aortic aneurysm as a result of hypoxia of the arterial wall adjacent to the intraluminal thrombus, which leads to increased local inflammation, neovascularization, and localized wall weakening.³¹⁻³⁴ This mechanism may be even more pertinent to the false lumen of a dissected aorta, since in this setting the residual outer layers of the aortic wall already have diminished strength.

In addition to partial thrombosis of the false lumen, independent predictors of mortality in our study included a history of atherosclerosis and a history of aortic aneurysm. Atherosclerosis has been previously linked to mortality in patients recovering from type B acute aortic dissection. Pathological studies have suggested that the wall of an aneurysmal aortic segment has decreased collagen synthesis, reduced elastin content, and a thinner wall as part of a systemic problem throughout the peripheral vasculature.^{33,35,36} These biophysical properties of the aorta predispose the entire aorta and its branches to dissection, further aneurysm formation, or rupture in the future and may contribute to the increased mortality in this group.

As with all observational studies, this investigation has limitations that must be kept in mind when the data are interpreted. First, our cohort consists of patients who were treated at aortic specialty centers and for whom imaging data on false-lumen status as well as follow-up vital statistics were available. As a result, these findings do not represent the entire cohort of the IRAD or patients followed at community hospitals. Second, the mortality data available to us did not include

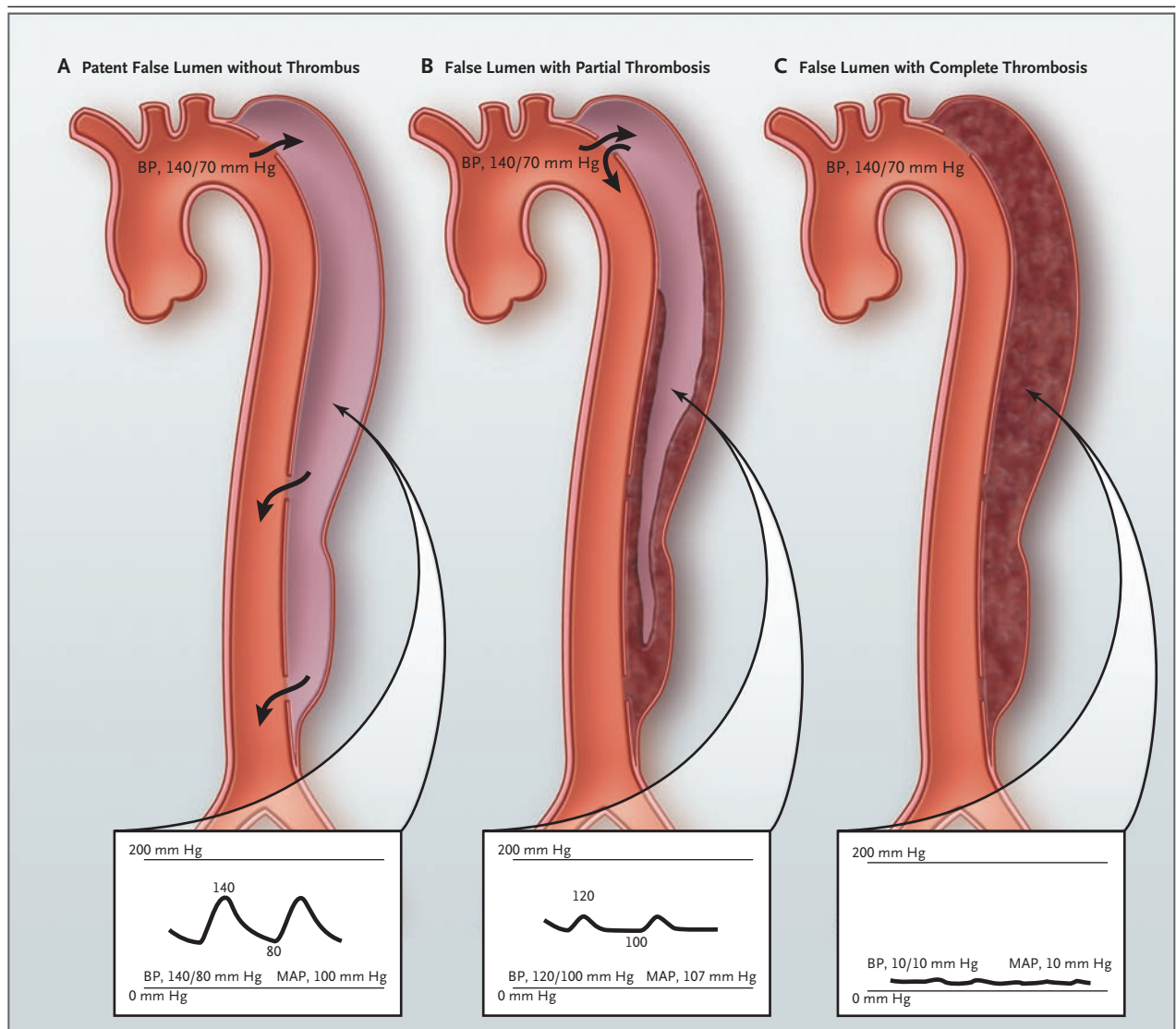


Figure 2. Conceptual Model of Risk According to the Status of the False Lumen.

The figure shows a proposed model of the physiological consequences of false-lumen patency or thrombosis, based on hemodynamic studies in ex vivo models and in patients with aortic dissection.²⁷⁻²⁹ Panel A shows type B aortic dissection with patent proximal and patent distal reentry tears in the absence of thrombus. The blood-pressure tracing shows systolic, diastolic, and mean arterial pressures in the false lumen similar to the pressures in the true lumen. Panel B shows type B aortic dissection with a patent entry tear and partial thrombosis that occupies the inner circumference of the false lumen and obstructs the reentry tears, forming a blind sac. The blood-pressure tracing shows diastolic and mean arterial pressures in the false lumen that exceed the pressures seen in Panel A, with identical pressures in the true lumen. Panel C shows type B aortic dissection with a false lumen filled with thrombus and no longer communicating with the true lumen. The pressure within the false lumen is likely to be low and nonpulsatile. BP denotes blood pressure, and MAP mean arterial pressure.

information on the cause of death. We were therefore unable to evaluate cause-specific mortality or other end points, such as freedom from reoperation, rupture, or redissection, which would be necessary to give more plausibility to our mechanistic hemodynamic theories. However, pre-

vious studies have shown that the majority of deaths in such patients are related to catastrophes of the aorta.^{2-4,9}

Third, imaging techniques were not standardized among centers, and imaging data were collected before our study was designed and were

not subsequently reevaluated. Thus, misclassification of false-lumen status is possible. Follow-up mortality was recorded independently of the designation of false-lumen status on the in-hospital data forms, thereby minimizing any systematic misclassification bias.

Fourth, false-lumen status was determined once during the hospitalization and does not reflect false-lumen status during the follow-up period. In small studies, false-lumen status changed in a minority of patients (18 to 25%) over 10 to 15 months and was not studied in the acute period.^{3,21,22} Therefore, we believe the likelihood of a change in false-lumen status is low in the early window of interest. However, surgery or endovascular therapy used in a complication-specific approach may alter the status of the false lumen before discharge. We therefore performed a separate analysis confined to patients receiving only medical treatment to demonstrate that the effect of false-lumen status on survival remains significant in this subgroup.

Fifth, although intramural hematoma was strictly defined in the IRAD, the true ability of cross-sectional imaging to distinguish between intramural hematoma and a completely thrombosed false lumen in an acute dissection is largely unknown because of the absence of a gold standard. Moreover, only pathoanatomic studies would be capable of determining whether an intimal tear was present. We therefore reanalyzed the data, including patients with intramural hematoma in the group classified as having complete

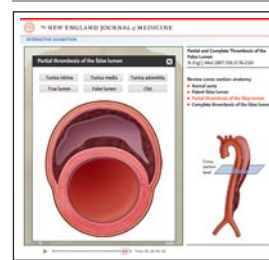
thrombosis of the false lumen. In this analysis, the differences between mortality curves did not change significantly.

In summary, we analyzed data from the IRAD to evaluate the prognosis in patients with type B acute aortic dissection who survive their initial hospitalization. Mortality is high after discharge from the hospital, with nearly one in four patients dying within 3 years. In patients with partial thrombosis of the false lumen, the risk of death is increased by a factor of 2.7 in comparison with patients with a completely patent false lumen.

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An interactive animation showing partial and complete thrombosis of the false lumen can be viewed at www.nejm.org.

APPENDIX

The investigators for the International Registry of Acute Aortic Dissection are as follows: *Coprincipal investigators:* K.A. Eagle, University of Michigan, Ann Arbor; E.M. Isselbacher, Massachusetts General Hospital, Boston; C.A. Nienaber, University of Rostock, Rostock, Germany. *Coinvestigators:* E. Bossone, National Research Council, Lecce, Italy; A. Evangelista, Hospital General Universitari Vall d'Hebron, Barcelona; R. Fattori, University Hospital S. Orsola, Bologna, Italy; J. Froehlich, University of Michigan, Ann Arbor; D. Gilon, Hadassah University Hospital, Jerusalem, Israel; S. Hutchison, St. Michael's Hospital, Toronto; J.L. Januzzi, Jr., Massachusetts General Hospital, Boston; A. Llovet, Hospital Universitario 12 de Octubre, Madrid; D. Mukherjee, University of Kentucky, Lexington; T. Myrnes, Tromsø University Hospital, Tromsø, Norway; P. O'Gara and J. Beckman, Brigham and Women's Hospital, Boston; J.K. Oh, Mayo Clinic, Rochester, MN; L.A. Pape, University of Massachusetts Hospital, Worcester; U. Sechtem and G. Meinhardt, Robert-Bosch Krankenhaus, Stuttgart, Germany; T. Suzuki, University of Tokyo, Tokyo; S. Trimarchi, Policlinico San Donato, San Donato, Italy. *Data management and biostatistical support:* J.V. Cooper and D.E. Smith, University of Michigan, Ann Arbor.

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