False Lumen Patency as a Predictor of Late Outcome in Aortic Dissection

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Aortic dissection (AD) is a disease with a high-risk of mortality. Late deaths are often related to complications in nonoperated aortic segments. Between 1984 and 1996, we retrospectively analyzed the data of 109 patients with acute AD (81 men and 28 women; average age 61 ± 14 years). All imaging examinations were reviewed, and a magnetic resonance imaging examination was performed at the time of the study. Aortic diameters were measured on each aortic segment. Predictive factors of mortality were determined by Cox's proportional hazard model, in univariate and multivariate analyses, using BMDP statistical software. Follow-up was an average of 44 ± 46 months (range 24 to 164). Actuarial survival rates were 52%, 46%, and 37% at 1, 5, and 10 years, respectively, for type A AD versus 76%, 72%, and 46% for type B AD. Predictors of late mortality were age >70 years and postoperative false lumen patency of the thoracic descending aorta (RR 3.4, 95% confidence intervals 1.20 to 9.8). Descending aorta diameter was larger when false lumen was patent (31 vs 44 mm; p = 0.02) in type A AD. Furthermore, patency was less frequent in operated type A AD when surgery had been extended to the aortic arch. Thus, patency of descending aorta false lumen is responsible for progressive aortic dilation. In type A AD, open distal repair makes it possible to check the aortic arch and replace it when necessary, decreases the false lumen patency rate, and improves late survival.

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Despite major improvements in diagnosis and surgical treatment over the past 10 years, acute aortic dissection (AD) remains a disease with a high-risk of mortality. In Stanford type A AD, 30-day mortality is as high as 25%.[1] Late deaths are mainly due to chronic dissection rupture, end-stage heart failure, stroke, and reoperations.[2] In Stanford type B AD, the 30-day mortality rate is lower and death usually occurs later, due to aortic rupture, extension of dissection, or development of aneurysms in the descending aorta.[3] At 10 years, the survival rate was an average of 42% to 44% in type A AD in the most recent series[4,5] and 32% in nonoperated type B.[6] This retrospective study examines factors of long-term survival after spontaneous AD. We focused on the evolution of nonoperated aorta segments, especially the evolution of false lumen patency, after serial magnetic resonance imaging (MRI) examinations. We also examined the role of surgical techniques on the false lumen patency rate.

METHODS

Patient selection: We analyzed the data for all patients referred to Besançon University Hospital for suspicion of AD between January 1984 and August 1996. Patients whose diagnosis was confirmed by at least 1 of the following examinations were included in the study: transthoracic echocardiography, transesophageal echocardiography (TEE), aortography, helicoidal computed tomographic scanning (CT scan), and MRI. A few patients, whose diagnosis had been modified after surgical or autopsy findings, were excluded. Depending on the period considered, the examinations performed were different; transthoracic echocardiography and aortography have been available since 1984, TEE since 1989, and MRI since 1992.

Follow-up: INITIAL EVALUATION: Demographic and clinical data at admission were collected from medical and surgical files. Examinations that led to diagnosis were reviewed, as well as surgical description of lesions and technical repair, if any.

Follow-up was performed either by surgeons or hospital cardiologists, or more often by the general practitioner and/or referring cardiologist. When applicable, patients' doctors received a questionnaire to update data. Clinical status, complications, reoperations (if any), and therapy were described. The most recent results of imaging technique examinations (transthoracic echocardiography, TEE, aortography, CT scan, or MRI) were obtained from the patients' doctors and were reviewed by cardiologists or radiologists, depending on the investigation. Patients who had not undergone an imaging examination for >1 year underwent a MRI examination.

For deceased patients, time and cause of death were obtained from general practitioner and/or cardiologists, and when possible, the last imaging examination was obtained. We distinguished between 30-day and late mortality.

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Magnetic resonance imaging examination: A Siemens Magnetom 1.5-T (Erlangen, Germany) device was used, with a standardized protocol, including electrocardiographic-gated spin echo sequences and cine MRI sequences, with a repetition time of 22 to 40 ms. The presence of an intimal tear was mandatory to confirm diagnosis. Extension of the intimal tear, as well as false lumen patency, and complications were sought in each patient. Aortic diameters were measured on each aortic segment, that is, ascending aorta or segment I (transverse axial plane), aortic arch or segment II (frontal plane), and descending aorta or segment III (transverse axial plane at the level of the left atrium). Measurements included the whole aortic lumen and 1 aortic wall thickness. All measurements were assessed by 2 independent radiologists, who were blinded to the data.

Statistical analysis: Predictive factors of 30-day mortality were determined by logistic regression, in univariate analysis. Multivariate analysis included prognostic factors with a p value ≤ 0.20 at univariate analysis. Predictive factors of late mortality were determined by Cox's proportional hazard model, by univariate analysis first. Multivariate analysis included prognostic factors with p value ≤ 0.20 at univariate analysis. Statistical analysis was performed with BMDP statistical software (BMDP Corp, Los Angeles, California). Comparison between groups was performed by a chi-square test for qualitative variables and by a Mann-Whitney nonparametric test of quantitative variables. A p value < 0.05 was considered statistically significant.

RESULTS

Patient population: Between January 1984 and August 1996, 109 patients with AD were included in the study. There were 81 men (74%) and 28 women (26%); average age was 61 $\pm$ 14 years (range 21 to 83). Sixty patients (55%) had hypertension, 32 of whom were on therapy. Nine patients had Marfan's syndrome (average age 33 $\pm$ 9 years), which had been previously undiagnosed, and was diagnosed at clinical examination and by the systematic examination of the removed aorta.

Acute AD was diagnosed by ≥ 1 of the following: transthoracic echocardiography, TEE, CT scan, or MRI. Use of these different modalities varied according to the period considered; transthoracic echocardiography was performed in nearly all cases. Until 1993, diagnosis was made by aortography in most cases. Conversely, beginning in 1993, multiplane TEE was performed alone in most patients. CT scan and/or MRI was performed when the other examinations were inconclusive.

Eighty patients (73%) had Stanford type A AD and 29 (27%) had type B AD. Sixty-seven patients had aortic regurgitation at admission, 37 experienced collapse, 23 patients with cardiac tamponade had type A AD, 38 patients had neurologic complications (cerebral or paraplegia in 10 patients with type B AD), and 19 had renal failure.

Fifty-six patients underwent emergency surgery; whereas 53 patients received only medical treatment. According to anatomic type, 54 patients with type A AD (68%) underwent surgery (16 died before operation and 10 were excused due to age and/or neurologic complications) versus 2 patients (7%) who had type B AD. The types of operations are listed in Table 1. Deep hypothermia with circulatory arrest and cerebral protection using retrograde venous perfusion, allowing an open distal repair,[6–10] has been systematically used since 1993 by our referring surgical team. Neither anterograde aortic perfusion during cardiopulmonary bypass nor recannulation of the aortic prosthesis after completion of aortic repair were routinely used. Sutures were systematically reinforced by Teflon as well as by GRF glue (Pharmacie Central du CHU, Besançon, France).

TABLE 1 Surgical Technique in Operated Type A Acute Aortic Dissection

Surgical Procedure	No. of Patients (%)
Composite valved conduit	22 (41)
Endoaortic graft	10 (18)
Supracoronary graft	22 (41)
Valvular prosthesis	23 (41)
Aortic valve resuspension	3 (5)
Aortic arch surgery	10* (18)
Aortic clamping	35 (65)
Open distal repair	19 (35)

*Among those 10 patients, 9 had type A and 1 had type B AD. No "elephant trunk" arch replacement was performed.

Medical treatment consisted mainly of antihypertensive medications. Of the 71 patients who survived the in-hospital period, 59 received antihypertensive therapy, 35 of whom were on β-blocker treatment.

Survival: Follow-up was an average of 44 $\pm$ 46 months (range 24 to 164). Global mortality was 54% at the end of the study. Survival rates for the whole group were 53% at 5 years and 40% at 12 years; 35% of patients died during the first 30 days.

Among the patients with type A AD, 16 patients (20%) died before operation and another 18 (23%) died during the in-hospital period. Among the patients who underwent surgery, 12 of 54 (23%) died during the in-hospital period. Before 1993, 15 of 55 patients (27%) died before operation compared with 1 of 25 patients (6%) between 1993 and 1996 ($p < 0.05$). Average age of the 15 patients who died before operation before 1993 was 60 $\pm$ 11 years; none had Marfan's syndrome, the average delay between onset of symptoms and admission was 14 $\pm$ 6 hours (range 1 to 48), 6 patients experienced collapse, and 4 had cardiac tamponade at admission; 9 of 15 patients (60%) underwent aortography. Actuarial survival rate of patients with type A AD was 52%, 46%, and 37% at 1, 5, and 10 years, respectively (Figure 1).

Among patients with type B AD, 3 (13%) died during the in-hospital period. Survival rates were 76%, 72%, and 46% at 1, 5, and 10 years, respectively, which was significantly higher than for type A AD (Figure 1).

Conversely, if we consider only patients with type

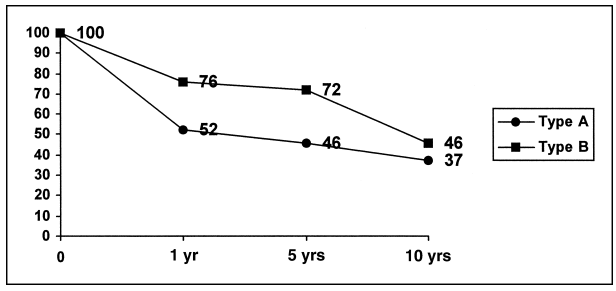

FIGURE 1. Actuarial survival according to dissection type (percent).

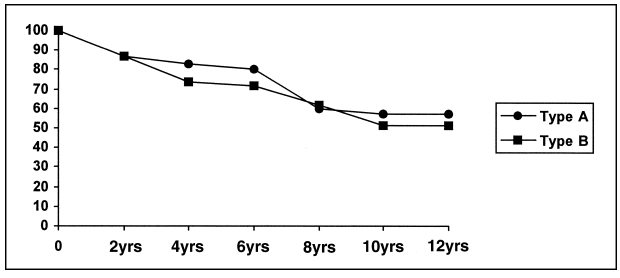

FIGURE 2. Long-term survival of patients who survived the in-hospital period. Abbreviations as in Figure 1.

A who underwent emergency surgery, their survival rate was 65%, 57%, and 46% at 1, 5, and 10 years, respectively; the difference with the type B survival rates were not significant.

In the same way, if we consider only patients who had survived the in-hospital period, actuarial survival rate at 10 years was 57% for type A versus 51% for type B (p = NS) (Figure 2).

CAUSE OF DEATH: The cause of death was identified in 54 of 56 cases (96%). Thirty-seven deaths (66%) were directly linked to AD, whereas 9 (16%) were due to other cardiac causes, 8 (14%) were due to other causes (cancer, sepsis, and so forth), and 2 remain unknown.

REOPERATIONS: Thirteen patients had late surgery, which included reoperation in 9 patients with type A AD, and an initial operation in 4 patients with type B AD, who had only received medical treatment at initial presentation. Average delay for late operation was 47.6 months and overall mortality was 31% (4 of 13). The late operation rate was 11% for type A AD; patients underwent reoperation because of complications on surgical graft or aneurysmal dilation of the nonoperated aortic segments (4 false aneurysms due to graft dehiscence, 3 recurrent dissections, and 2 patients with type A AD who developed aneurysms in the thoraco-abdominal descending aorta, 4 patients with type B who developed aneurysms in the ascending aorta). Two patients who underwent reoperation had Marfan's syndrome.

Predictive factors of 30-day mortality: In univariate analysis, predictive factors of 30-day mortality were age >70 years, type A AD, absence of Marfan's syndrome, collapse, pericardial tamponade, neurologic complications, renal failure, and diagnosis ascertained by aortography instead of echocardiography alone. Multivariate analysis showed older age, collapse, and neurologic complications to be predictive of in-hospital mortality.

TABLE 2 Predictive Factors of Long-term Mortality (univariate analysis) for the Whole Group

Variable	Class	Relative Risk	95% CI
Sex	M	1	
	F	0.82	0.26–2.53
Age (yrs)	<70	1	
	>70	5.9	2.08–16.6*
Preop hypertension	No	1	
	Yes	1.3	0.5–3.46
Preop treated hypertension	No	1	
	Yes	0.74	0.16–3.32
β-blocker treated hypertension	No	1	
	Yes	1.24	0.40–2.46
Stabilized hypertension	No	1	
	Yes	0.94	0.26–3.3
Stanford type	A	1	
	B	1.3	0.48–3.5
False lumen patency (preop)	No	1	
	Yes	1.7	0.60–4.8
False lumen patency (postop)	No	1	
	Yes	3.2	1.17–8.8*

*Variables with a p <0.20 included in multivariate analysis.
CI = confidence interval; Preop = preoperative; postop = postoperative.

Factors influencing late mortality: Of the 71 patients who survived the first 30 days, long-term data were available for 61. Duration of follow-up was an average of 57 ± 43 months. The following variables were tested at univariate analysis: age, gender, hypertension, treated hypertension, hypertension treated with β blockers, correctly stabilized hypertension, Stanford type, and descending aorta false lumen patency preoperatively and during follow-up. Whatever the analysis, univariate or multivariate, and whatever the dissection type, only 2 factors were found predictive of late mortality: age >70 years and postoperative false lumen patency of the thoracic descending aorta (Table 2). Obviously, false lumen patency, as assessed by MRI, was the strongest predictor of late outcome. Postoperative treatment with β blockers reduced late mortality only in type B AD (RR 0.23, 95% confidence intervals 0.05 to 0.93).

Evolution of false lumen patency and of aortic diameters: Initial investigations (transthoracic echocardiography, TEE, angiography, or others, according to the period considered) showed a false lumen patency of the descending aorta in 37% of patients with type A AD and in 100% of patients with type B. In patients who survived the in-hospital period, iterative MRI examinations showed a tendency toward an occlusion of the false lumen, which remained patent in 17% of patients with type A AD (Table 3) and in 48% of patients with type B AD at 5 years.

Increasing thoracic descending aorta diameters is the main concern for long-term evolution, because the ascending aorta is either prosthetic in most type A AD or normal in type B AD, and because the aortic arch is very difficult to measure precisely for technical reasons. In type A AD, the average descending aorta

TABLE 3 Descending Aorta False Lumen Evolution in Type A Aortic Dissection in Patients Who Survived the In-hospital Period

	False Lumen Patency				
	Whole Group (n = 42)	p Value	Aortic Clamping (n = 30)	Open Distal Repair (n = 12)	p Value
Preoperative	15 (37%)				
6 mo	14 (33%)	NS	11 (36%)	3 (25%)	NS
2 yrs	14 (33%)	NS	12 (40%)	2 (16%)	NS
4 yrs	13 (31%)	NS	12 (40%)	2 (8%)	<0.05
5 yrs	7 (17%)	<0.05	7 (23%)	1 (8%)	<0.05

TABLE 4 Evolution of Descending Aorta Diameter in Type A Aortic Dissection, According to False Lumen Patency

	False Lumen Diameter (mm)		
Year	Not Patent	Patent	p Value
2	26 (n = 19)	38 (n = 11)	<0.05
2.5	26 (n = 11)	36 (n = 10)	<0.05
4	26 (n = 13)	43 (n = 6)	<0.05

diameter did not increase significantly over a 5-year period (from 30 ± 6 to 31 ± 10 mm, p = NS). In type B AD, all patients who had a descending aorta diameter that increased to >55 mm during follow-up died (3 patients), or were reoperated on for replacement of the descending aorta (4 patients).

Moreover, for the whole group, descending aorta diameter was an average of 31 mm when the false lumen was not patent versus 43 mm when the false lumen was patent (p = 0.02), confirming that aneurysmal evolution of the descending aorta is linked to false lumen patency. The link between patent false lumen and aortic enlargement was clearly demonstrated for type A AD (Table 4), but not for type B AD because of the small number of patients in this subgroup.

Last, in operated type A AD, surgical techniques had direct influences on descending aorta false lumen patency at long-term follow-up (Table 3). Extension of surgical reparation to the aortic arch under deep hypothermia allowed for closure of intimal tear entry site more often and reduced late false lumen patency.

DISCUSSION

AD remains a high mortality disease, with a survival rate of about 40% at 10 years, whatever the type of AD. Factors of in-hospital mortality are well identified, including older age (>70 years) and severe complications at initial presentation. Similar survival rates and factors of mortality have been reported by others.[1,3,4,6] It is interesting that early and late mortality did not differ significantly between patients who had survived after type A AD surgery and patients with type B AD. We should consider that patients in both groups have the same disease, with comparable life expectancy, and may have similar risks of complications involving nonoperated aortic segments.

A dramatic decrease in very early mortality of type A AD has been observed since 1993: in our series, 27% of patients (15 of 55) died preoperatively before 1993 versus 4% of patients (1 of 25) after 1993, although the characteristics of these patients were not different from those of the whole population. Since 1993, diagnosis has been established by multiplane TEE alone, instead of angiography, which is a less invasive approach and makes it possible to shorten preoperative delay.[11–16] Rizzo et al[15] showed that angiography was responsible for an increased mortality rate. In contrast, it has been found that TEE has a sensitivity as high as 97%[11,12] and a specificity of 77%[12] for the diagnosis of AD; moreover, it permits determination of anatomic type, and shows entry site and complications (e.g., aortic insufficiency or pericardial effusion). Noninvasive management of patients suspected of AD has been recommended since the early 1990s.[11–16] Nienaber et al,[12] in their review of noninvasive procedures allowing diagnosis of AD, proposed MRI in hemodynamically stable patients and TEE in others, but MRI is seldom available in an emergency. At the same time, our surgical team changed their technique and all patients are currently undergoing surgery under deep hypothermia with circulatory arrest and retrograde cerebral perfusion through the superior vena cava.[7,10,17,18] This technical improvement has made it possible to avoid clamp damage to the aorta beyond the distal graft anastomosis and to perform an open distal repair, allowing extension of aortic exploration and repair of the aortic arch when necessary. Svensson et al[19] reported a decrease of in-hospital mortality due to surgery under deep hypothermia. The combination of these 2 factors, diagnosis by TEE alone and open distal repair, has been responsible for improvement of early survival since 1993.

A surprising fact was a better short-term prognosis in patients with Marfan's syndrome, which is probably due to the much younger age in this subgroup (33 ± 9 vs 61 ± 14 years for the whole group). The same observations have been made by Gott et al[20] in a series of 270 patients. Patients with Marfan's syndrome have, however, a higher rate of reoperation than other patients, 33% in our series, 39% in others' series.[2,21]

Factors of late mortality, either by univariate or by multivariate analysis, were older age and descending aorta false lumen patency, demonstrated by MRI, which was the strongest predictor of late mortality. It is then of vital importance to follow patients indefinitely for evolution of false lumen and aortic diameters by iterative imaging examinations: by TEE, which is difficult for patients to accept, MRI, or helical CT scan.[22–24] Similar observations have been made by other investigators in larger series.[14,24–26] In 1993, Erbel et al[14] showed that false lumen patency signif-

TABLE 5 Predictive Factors of Long-term Mortality (multivariate analysis) for the Whole Group

Variable	Class	Relative Risk	95% CI
Age (years)	<70	1	
	>70	1.08	1.03–1.13
False lumen patency (postop)	No	1	
	Yes	3.4	1.20–9.8

Abbreviations as in Table 2.

icantly increased the reoperation rate and late mortality.

In our experience, evolution of dissected descending aorta diameters was directly linked to false lumen patency or thrombosis in operated type A AD. A correlation between both facts could not be demonstrated in type B AD, because of the small number of patients. Patency of false lumen is clearly the cause of aortic enlargement, leading to reoperation or death. In our series, all patients with type B AD who had a descending aorta diameter >55 mm were reoperated on or died during follow-up, which confirms literature data. Until now, most investigators have hypothesized that operation should be performed when the aorta diameter reaches 65 mm.[27,28] A new trend is emerging, taking into account not only aorta diameter, but also false lumen patency. The trend is to operate on smaller aortas when the false lumen remains patent. Treatment is also changing with the development of endoluminal stent-graft placement by which thrombosis of the false lumen can be achieved.[29]

Another important fact to be noted is the role of surgical technique on evolution of descending aorta false lumen patency and aortic diameters in type A AD. In our series, the rate of false lumen patency was significantly lower after 2 years in patients who underwent open distal repair, compared with patients operated on under aortic clamping. A more complete reparation of lesions, especially closure of entry site, when located on or extending to the transverse arch, was responsible for improvement of the rate of late false lumen closure.[30] Bachet et al[2] showed that closure of the intimal tear entry site at initial emergency operation reduced the rate of reoperation. It is then expected that this technical change could also improve long-term mortality of patients suffering from type A AD.

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