

Predictors of surgical indications for acute type B aortic dissection based on enlargement of aortic diameter during the chronic phase

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Abstract

Objectives. Patients with Stanford type B dissection who have been treated successfully with medical hypotensive therapy during the acute phase (<14 days) have the risk of surgery during the chronic phase because of enlargement of the dissected aorta. The objective of this retrospective analysis is to determine the predictors of surgical indications for acute type B aortic dissection by studying chronic-phase enlargements of aortic dissection in patients treated successfully with medical hypotensive therapy.

Methods. Altogether, 131 patients with type B aortic dissection were treated medically during the acute phase between 1987 and 2004. Multivariate factor analyses were performed to determine the predictors of chronic-phase enlargement (≥ 55 mm, as defined for our surgical criteria) of the dissected aorta.

Results. Overall dissection-related mortality was 17.6%. Patency of the false lumen was an independent risk factor for dissection-related death ($P = 0.0238$, hazard ratio 2.594, confidence interval 1.009–6.122) and for dissection-related events ($P = 0.0157$, hazard ratio 1.870, confidence interval 1.116–3.133). The incidence of patients treated surgically during the chronic phase was 32.8%. The predictors for aortic enlargement during the chronic phase were the condition of maximum aortic diameter ≥ 45 mm with a patent false lumen during the acute phase. The rates of freedom from aortic enlarge-

ment (≥ 55 mm) for patients with maximum diameter (≥ 45 mm) with a patent false lumen during the acute phase at 1, 5, and 10 years were 72.6%, 66.0%, and 42.8%, respectively, whereas in patients with a maximum aortic diameter of <45 mm with a thrombosed false lumen the values were 100%, 94.7%, and 89.2%, respectively ($P < 0.005$).

Conclusions. These data suggest that patients with acute type B dissection with a patent false lumen or a diameter ≥ 45 mm (or both) during the acute phase are at risk for enlargement of the dissecting aorta. The patients with dissecting aortas ≥ 45 mm or a patent false lumen (or both) therefore require close follow-up to detect enlargement of the dissecting aorta, whereas patients with a maximum aortic diameter of <45 mm with a thrombosed false lumen can stay on conservative therapy.

Key words Aortic dissection · Stanford type B · False lumen · Maximum aortic diameter · Predictors

Introduction

Aortic dissection is the most common catastrophic event that affects the thoracic and abdominal aorta. In 1955, DeBakey et al. reported six patients with dissecting aneurysms treated surgically, four of whom survived. In 1965,¹ Wheat et al. demonstrated that intensive hypotensive medical therapy decreased mortality associated with acute aortic dissection.² For patients with Stanford type A acute aortic dissection, immediate surgical therapy has been provided to reduce mortality more effectively than medical therapy.^{3,4} The preference for conservative management of type B dissection is derived from an apparently favorable prognosis during the acute period.

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However, not a few patients with type B dissection die of aortic rupture during the chronic period despite aggressive medical management.^{5,6}

There is still some controversy concerning the therapeutic strategies for patients with type B aortic dissection.^{7,8} One important question is whether any predictors of poor survival for a patient with type B aortic dissection may exist at the time of initial hospitalization. This study was performed to clarify the prognostic factors in patients with type B aortic dissection.

Methods

Study population

Between August 1987 and April 2004, a total of 131 consecutive patients with acute type B aortic dissection were admitted to the Intensive Care Unit at the National Hospital Organization Sapporo Center. All patients had computed tomography (CT) scans available for review and were diagnosed with an accurate date of onset. Acute aortic dissection was diagnosed on the basis of acute back pain or chest pain (or both) and by CT evidence of an intimal flap. Of the 131 patients, 80 were treated successfully with hypotensive therapy without surgery. The 80 included 51 with a thrombosed false lumen (thrombosed group) and 29 with a patent false lumen (patent group) (Fig. 1).

Definitions

We defined a patent false lumen during a false lumen that was enhanced during either an early or a delayed

phase. A thrombosed false lumen is not enhanced during either the early or delayed phase. A thrombosed false lumen was identified by complete occlusion of the false lumen by thrombus. We measured the maximum aortic diameter anywhere in the descending thoracic aorta. Aortic enlargement during the chronic phase for surgical indications is defined at our hospital as follows: (1) maximum diameter of the dissected aorta ≥ 55 mm; (2) rapid enlargement of dissected aorta >10 mm/year. Rupture of the dissected aorta is of course an indication for surgery.

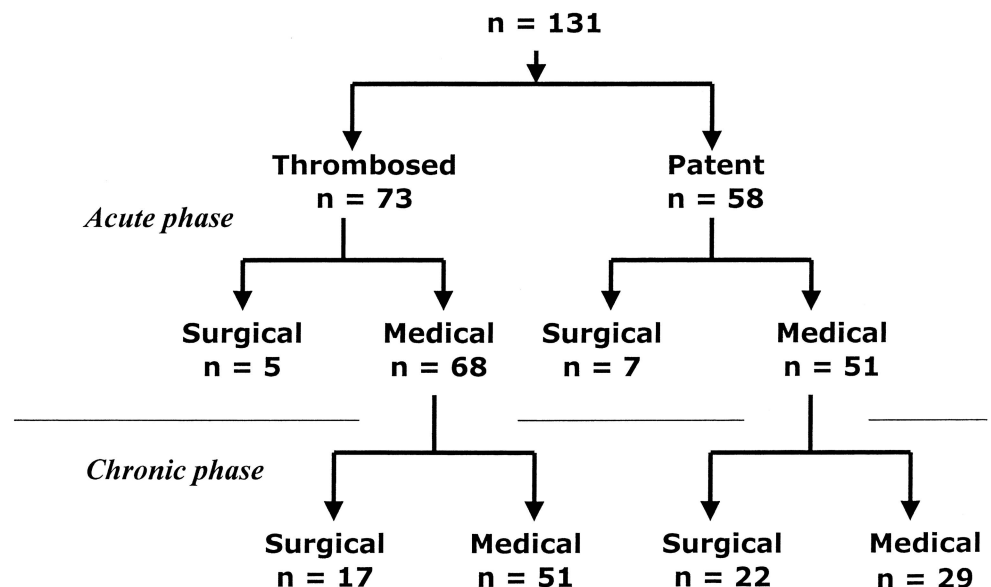
Statistical analysis

The percentages of dissection-related death-free and event-free rates were determined by the Kaplan-Meier method, and the curves of the thrombosed and patent groups were compared by a log-rank test. The Cox proportional hazard model was applied to all 131 patients, to the patent and thrombosed groups, and to the <45 mm or ≥ 45 mm groups to determine the independent effects of freedom from aortic enlargement ≥ 55 mm and surgical treatment for aortic enlargement. Data analysis was performed with the use of StatView J-5.0 for Windows. Significance was assumed at $P < 0.05$.

Results

All 131 patients with type B aortic dissection who had passed the acute phase with medical hypotensive therapy were followed up. Of the 131 patients, 80 continued to receive hypotensive therapy, whereas 51 patients underwent surgery: 12 patients during the acute phase and 39 patients during the chronic phase.

Fig. 1 A total of 131 patients were included in our study. Among them, 119 were treated medically and survived the acute phase. The 119 patients included 68 with a medically treated thrombosed false lumen (thrombosed group) and 51 with a treated patent false lumen (patent group)



Baseline characteristics of the patent and thrombosed groups on admission are shown in Table 1. Patients in the thrombosed group had significantly better outcomes than patients in the patent group in regard to dissection-related death-free (Fig. 2) and dissection-related event-free rates (Fig. 3). The dissection-related deaths and dissection-related events occurred during earlier follow-up periods in both groups. Factors influencing the long-term outcome of 119 type B patients discharged without surgery during the acute phase are shown in Tables 2 and 3. A patent false lumen ($P = 0.029$ and $P = 0.018$) and maximum aortic diameter of ≥ 45 mm ($P < 0.001$ and $P = 0.004$) were shown by multivariate Cox proportional hazard analysis to be significantly predictive of dissection-related death and dissection-related events. The hazard ratio for dissection-related death of an aortic diameter of ≥ 45 mm was 7.042 times higher than that of < 45 mm; and the hazard ratio for a presence of a patent

false lumen was 2.594 times higher than that for the thrombosed false lumen. Similarly, the hazard ratio for the dissection-related events of an aortic diameter ≥ 45 mm was 2.132 times higher than that of one < 45 mm; and the hazard ratio for the presence of the patent false lumen was 1.870 times higher than that of the thrombosed false lumen. To obtain a more accurate estimate of chronic-phase aortic enlargement, the 119 patients were divided into four subgroups according to four combinations of the two independent predictive variables: maximum aortic diameter < 45 mm and a thrombosed false lumen (group A); maximum aortic diameter < 45 mm and a patent false lumen (group B); maximum aortic diameter ≥ 45 mm and a thrombosed false lumen (group C); and maximum aortic diameter ≥ 45 mm and a patent false lumen (group D). The values for freedom from aortic enlargement for group A at 1, 3, 5, and 10 years were 100%, 94.7%, 89.4%, and 89.4%, respectively; the values for group D were 73.9%, 73.9%, 69.6%, and 43.5%, respectively (Fig. 4).

Table 1 Patient characteristics for the entire study group (August 1987 to April 2004)

Basic characteristics	No. (range)
Total patients	131
Age (years)	68.2 (58–81)
Follow-up period (years)	4.3 (0 days to 16.6 years)
Sex (no.)	
Male	98 (74.8%)
Female	33 (25.2%)
False lumen (no.)	
Patent	58 (44.3%)
Thrombosed	73 (55.7%)

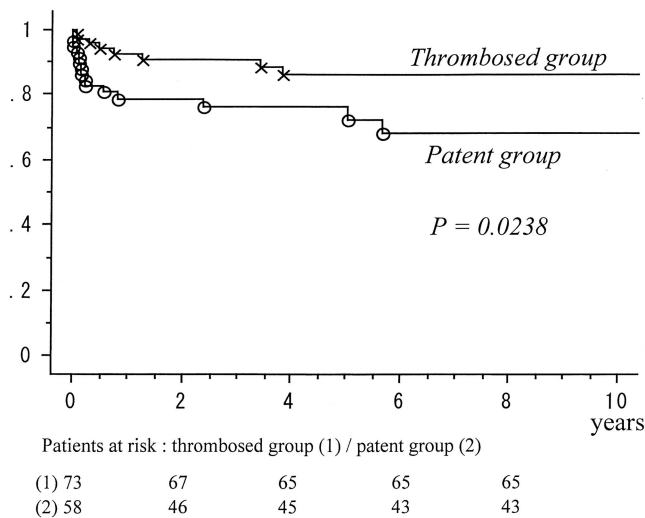


Fig. 2 Dissection-related death-free ratio of patients in the thrombosed group and that of patients in the patent group were compared by the Kaplan-Meier method and log-rank test. The thrombosed group had a significantly better outcome than the patent group

Discussion

Acute aortic dissection is considered a lethal disease unless appropriately treated. The optimal treatment for patients with type B aortic dissection remains a matter of debate. Therefore, it is important to predict chronic-phase aortic enlargement of type B dissection to discuss the indications for surgical treatment of type B dissection. Each physician or surgeon determines the surgical

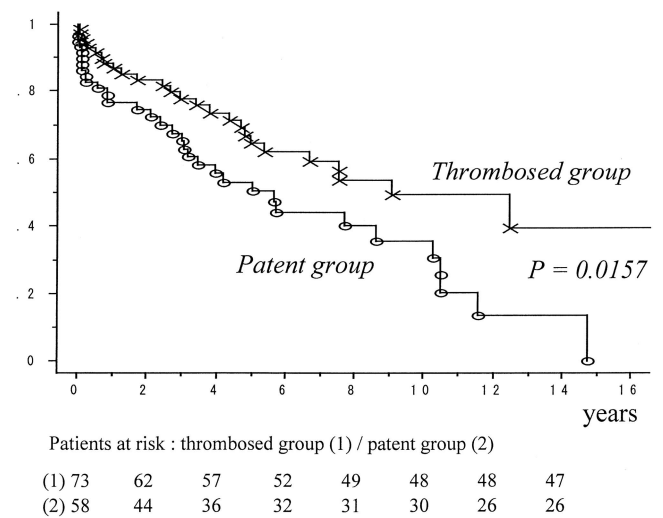


Fig. 3 Dissection-related event-free ratio of patients in the thrombosed group and that of patients in the patent group were compared by the Kaplan-Meier method and log-rank test. The incidence of dissection-related events is higher in the patent group than in the thrombosed group

Table 2 Cox proportional hazard model analysis of dissection-related death

Parameter	Dissection-related death		
	<i>P</i>	Hazard ratio	95% CI
Age	0.242	0.981	0.949–1.013
Male sex	0.679	1.233	0.457–3.322
Hypertension	0.348	2.004	0.469–8.547
Cerebrovascular accident	0.195	1.927	0.715–5.208
Chronic renal failure	0.027	2.865	1.125–7.299
Patency of false lumen on admission	0.029	2.594	1.099–6.122
Maximum dissected aortic segment ≥ 45 mm on admission	<0.001	7.042	2.618–18.868

CI, confidence interval

Table 3 Cox proportional hazard model analysis of dissection-related events

Parameter	Dissection-related event		
	<i>P</i>	Hazard ratio	95% CI
Age	0.359	0.990	0.969–1.011
Male sex	0.231	1.475	0.780–2.793
Hypertension	0.691	1.151	0.574–2.309
Ulcer-like projection in the thrombosed group	0.387	1.406	0.648–3.049
Chronic renal failure	0.217	1.608	0.756–3.413
Patency of false lumen on admission	0.018	1.870	1.116–3.133
Maximum dissected aortic segment ≥ 45 mm on admission	0.004	2.132	1.285–3.584

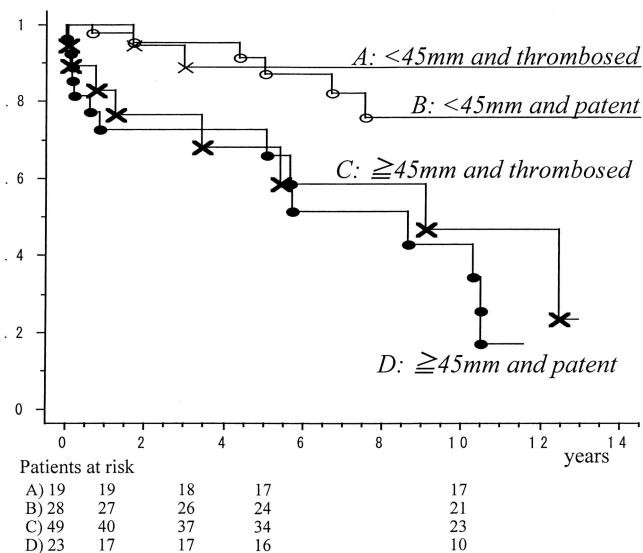


Fig. 4 Freedom from aortic enlargement >55 mm. The 119 patients were divided into four subgroups according to the mode of the therapy as follows (compared by the Kaplan-Meier method and log-rank test): maximum aortic diameter <45 mm and a thrombosed false lumen at admission (group A) were relatively good, however, the values for freedom from aortic enlargement for group, maximum aortic diameter ≥ 45 mm and a patent false lumen (group D) on admission were not good

indication for type B aortic dissection according to his or her experience and the surgical results of the institution. It has been generally advocated that patients who have type B acute aortic dissection without complications,

such as rupture or organ ischemia, should be treated with hypotensive drugs during the acute phase because the mortality rate for medical hypotensive therapy is reported to be equal to or slightly better than that for surgical treatment during the acute phase.^{6,7,9–11} Surgical treatment should be selected if the aortic diameter becomes enlarged during the chronic phase. However, surgical results for these cases of enlarged aorta are definitely not better than the results for acute-phase surgery because more extensive surgery and concomitant reconstruction of visceral arteries are necessary in most of these surgeries during the chronic phase.^{11,12} Unfortunately, some patients who have successfully passed through the acute phase with medical hypotensive therapy suddenly die of aortic rupture during the chronic phase. Ideally, enlargement of the aortic diameter could be predicted and surgery performed appropriately before the aortic diameter becomes critically enlarged.¹³ The predictability of aortic enlargement with type B dissection might help patients avoid the life-threatening risk of the chronic phase. These results on the predictive value of acute-phase variables for chronic-phase aortic enlargement should stimulate consideration of surgical indications.

In our institution, the principal therapy consists of initial medical treatment with antihypertensive and negative inotropic agents unless major complications are recognized during the acute phase. Emergent operations were performed for seven (5.3%) patients in this series.

Our indication for emergent operation is mainly rupture or impending rupture. Ischemic complications of dissection such as visceral and limb ischemia are sometimes indications for emergent surgery. Five of the seven patients showed one of these complications in this series, and there were two cases in which ischemic complications were the main indication for operation. The emergent operative mortality rate of 57.1% (4/7) was much higher than those reported by other authors because our surgical group consisted of ruptured cases in a state of shock before operation.

The recommendations in some institutions are that aggressive surgical treatment should be offered with type B dissection during the acute phase because prevention of aortic rupture and organ ischemia due to acute-phase surgery contributes to a better mortality or morbidity rate. Moreover, a number of patients with medically treated dissection must undergo surgical treatment for aortic enlargement during the chronic phase.^{6,8,10,13}

Our results indicate that chronic-phase aortic enlargement of type B dissection can be predicted with two independent factors: the maximum diameter of the dissected aorta and the patency of the false lumen at the onset of dissection. The dissection-related event-free curve for patients with a patent false lumen was significantly lower than that for patients with a thrombosed false lumen. The patients with a patent false lumen were expected to have a higher incidence of aortic enlargement than the patients with a thrombosed false lumen during the chronic phase. Moreover, the value for freedom from aortic enlargement ≥ 55 mm during the chronic phase in patients with a maximum aortic diameter of ≥ 45 mm and a patent false lumen on admission at 1, 3, 5, and 10 years were 73.9%, 73.9%, 69.6%, and 43.5%, respectively. On the other hand, the values for patients with a maximum aortic diameter of < 45 mm and a thrombosed false lumen at 1, 3, 5, and 10 years were 100%, 94.7%, 89.4%, and 89.4%, respectively.

We believe that the patients with a patent false lumen or a diameter ≥ 45 mm during the acute phase are at risk of enlargement of the dissecting aorta. The patients with a dissecting aorta ≥ 45 mm, a patent false lumen, or both therefore require close follow-up to detect any enlargement of the dissecting aorta. The patients with a maximum aortic diameter of < 45 mm should be treated medically so long as the aortic diameter does not become enlarged, but patients with a patent false lumen, should be carefully observed. Patients with a maximum aortic diameter of < 45 mm and a thrombosed false lumen should continue to receive medical hypotensive therapy. We should also take the favorable effect of beta-blockers into consideration for preventing enlargement of the

diseased aorta, with the result of increasing survival of patients with chronic type B aortic dissection.¹⁴

There were several limitations to the present investigation. It was not a prospective, controlled, randomized study. A potential bias exists because of the other factors influencing the outcome of our patients, such as pulmonary emphysema and the quality of blood pressure control during the follow-up period, making it difficult to determine precisely the critical size for aortic rupture. More detailed study of these issues are needed. Furthermore, the therapeutic strategies for type B aortic dissection have changed during previous years, particularly for malperfusion and the locally enlarged aorta, where new interventional methods with fenestration and endoaortic prostheses are promising.^{15,16} This study, however, provides a direction for the continued discussion of managing patients with acute type B dissection.

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