Impact of the entry site on late outcome in acute Stanford type B aortic dissection†

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Abstract

OBJECTIVES: This study aimed to investigate whether the entry site of acute type B aortic dissection affects late outcomes.

METHODS: Inpatient and outpatient records were retrospectively reviewed.

RESULTS: We identified 224 cases of acute type B aortic dissection between 1998 and 2013. Of these 224 patients, 168 were men and the age was 64.2 ± 12.6 (range 23–94) years, from which 130 presented with the entry at a location downstream of the distal aortic arch, 67 with the entry at the outer curvature of the distal aortic arch and 27 with the entry at the inner curvature. At the initial presentation, 127 patients had descending false lumen thrombosis. The 30-day mortality rate was 2%, and 8% of patients had malperfusion. The entry at the outer curvature was associated with a higher risk of 30-day mortality. Patients with the entry at a location downstream were significantly older, and had a higher chance for primarily thrombosed descending false lumen and a lower risk of malperfusion. At follow-up (6.0 ± 4.1 years), the actuarial survival rates were 97, 83 and 60%, freedoms from open aortic surgery were 96, 91 and 86%, aortic intervention were 73, 66 and 63% and aortic events were 71, 60 and 52% at 1, 5 and 10 years, respectively. Multivariate logistic regression analysis revealed that the outer curvature entry and maximum aortic diameter were correlated with open aortic surgery, aortic intervention and aortic events. Of the 127 patients with primarily thrombosed false lumen, the outer curvature entry was significantly correlated with aortic events.

CONCLUSIONS: The primary entry at the outer curvature of the distal aortic arch, as well as the large aortic diameter, is associated with a higher risk of late open aortic surgery, aortic intervention and aortic events in acute type B aortic dissection. Thus, the entry site should be taken into consideration in the establishment of an appropriate treatment indication of type B aortic dissection.

Keywords: Acute type B aortic dissection • Aortic event • Aortic intervention • Entry • Outcome

INTRODUCTION

Uncomplicated acute Stanford type B aortic dissection does not require intervention and is normally managed with medical treatment for blood pressure control [1]. However, in cases with complications such as rupture or malperfusion, an emergency procedure is required. Acceptable early outcomes are reportedly achieved by emergency thoracic endovascular aortic repair (TEVAR) in patients with complicated acute type B aortic dissection [2]. A certain percentage of patients with acute type B aortic dissection develop dilatation during late follow-up [3]. In such patients, open aortic surgery is required, which is associated with a considerable risk of early mortality [4]. Therefore, there has been a drive to expand the indication of TEVAR to uncomplicated type B aortic dissection, so that it can be treated with stent grafting before aortic dilatation. In the INSTEAD trial conducted to compare TEVAR with medical treatments for stable type B aortic dissection at least 2 weeks after onset, no significant differences were observed in mid-term outcomes [5], although it was shown that TEVAR had a positive effect on the remodelling of the dissected descending aorta [6]. However, we previously showed that complete obliteration of the false lumen in the entire aorta is difficult with TEVAR, particularly in cases with a patent false lumen, thus increasing the risk of thoracoabdominal aortic enlargement and warranting late high-risk open aortic surgery. Moreover, we observed that the primary entry at the outer curvature of the distal aortic arch is associated with less chance for thrombosis of the descending false lumen, which is the first step for aortic

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remodelling [7]. Open aortic surgery for chronic type B aortic dissection after TEVAR may be complicated, occasionally involving a non-negligible risk of flap injury during explanting and a risk of spinal cord ischaemia, which is particularly observed in emergency settings [8]. Not all patients with acute type B aortic dissection develop dilatation; therefore, TEVAR in patients with uncomplicated type B aortic dissection may not benefit and instead possibly harm these patients during open aortic surgery at later stage. Thus, an appropriate indication of TEVAR for acute type B aortic dissection is required. To establish the indication for intervention, it is essential to recognize the natural course of acute type B aortic dissection, that is, which type tends to become complicated and which type does not. In this study, we investigated whether the entry site of patients with acute type B aortic dissection affects the late outcomes.

MATERIALS AND METHODS

Patients and methods

From 1998 to 2013, a total of 237 patients with a diagnosis of acute Stanford type B aortic dissection were treated at Kitasato University Hospital. Among these, patients with traumatic dissections, inconclusive computed tomography (CT) angiography findings, acute type A aortic dissection with thrombosed ascending false lumen at the initial presentation and equivocal onset were excluded. The remaining 224 cases were selected for detailed analysis, and were included in this study.

Patient medical records were retrospectively reviewed for demographics, diagnosis and complications at the initial presentation. All patients underwent CT angiography at presentation, within 10 days after the onset and in the late period (3.7 ± 4.0 years). CT images were used for measuring the aortic diameter and locating the site of the initial entry. If there was no dissection at the distal arch and if the descending false lumen was thrombosed at the initial presentation, the entry was considered to be located downstream of the distal aortic arch.

The outcomes, including open surgery and intervention for aortic dissection, were investigated. An event was considered as an aortic event if malperfusion, retrograde extension of the aortic dissection or aortic rupture developed, or if TEVAR or open aortic surgery was necessary.

Statistical analysis

The JMP 11 software (SAS Institute, Cary, NC, USA) was used to perform statistical analysis. Continuous variables were presented as means ± standard deviations. Student’s t-test was used to analyse differences in continuous variables. Fisher’s exact test was used to compare categorical variables. Analysis of variance (ANOVA) was used to test the equality of continuous variables between the three groups with different entry sites. Kaplan–Meier analysis was used to assess the survival, freedoms from open aortic surgery, aortic intervention and aortic event, and the log-rank test was used for comparison between groups. To analyse the factors affecting survival, freedoms from open aortic surgery, aortic intervention and aortic event, a Cox proportional hazard model was used, with age, site of the entry and maximum aortic diameter entered as explanatory variables. All P-values were two-sided, and a P-value of <0.05 was considered statistically significant.

RESULTS

Two hundred and twenty-four patients with definite diagnosis of acute type B aortic dissection were treated during the study period. Table 1 summarizes the profile of all 224 patients. The mean age was 64.2 ± 12.6 (range 23–94) years. Six patients had Marfan syndrome and 8 had collagen diseases, including rheumatoid arthritis, systemic lupus erythematosus, polymyalgia rheumatica and dermatomyositis. Sixty-seven (30%) patients presented with the primary entry at the outer curvature of the distal aortic arch, 27 (12%) with the primary entry at the inner curvature and 130 (58%) with the primary entry at a location downstream of the distal aortic arch. Patients with the entry at a location downstream were significantly older (versus outer curvature, P = 0.0003; versus inner curvature, P = 0.039). The aortic diameter at onset was 35.8 ± 5.9 mm, and there was no significant difference in the diameter between the three entry groups. At the initial presentation, 127 (57%) patients had descending false lumen thrombosis with or without an ulcer-like projection or localized flow communication between the true and false lumen at the primary entry; 15 (12%) with the primary entry at the outer curvature of the distal aortic arch, 8 (6%) at the inner curvature and 104 (82%) at a location downstream, and the entry at a location downstream was associated with a higher chance for false lumen thrombosis (versus outer curvature, P < 0.0001; versus inner curvature, P < 0.0001). The 30-day mortality rate was 2% (4/224), all of which had the primary entry at the outer curvature. Malperfusion occurred in 17 (8%) patients 0–41 days after the onset. Primary entry at a location downstream was associated with a lower risk of malperfusion (versus outer curvature, P < 0.0001; versus inner curvature, P = 0.032).

The 224 patients were followed up for 6.0 ± 4.1 years, and the follow-up was 100% complete. Of the 7 patients who developed retrograde extension of the dissection to the ascending aorta (1 day–22 months after the onset), 6 had the primary entry at the outer curvature and 1 had the primary entry at the inner curvature. Nineteen patients had aortic rupture 0–3629 days after the onset. The maximum aortic diameter (P < 0.0001) and haemodilysys (P = 0.015) were significantly correlated. Eight cases ruptured within 2 weeks after onset. Of these, 3 underwent TEVAR, 3 underwent open surgery and 2 died before entering the operation room. All 6 patients who received either TEVAR or open surgery survived the operation. Of 11 cases with late aortic rupture, 5 were taken to other hospitals and died. Table 2 reports the details of the remaining 6 patients who were ambulanced to our hospital. One patient had refused surgical treatment before rupture of a distal aortic arch true aneurysm. One patient had rupture of retrograde type A dissection. Two patients had aortic rupture during preoperative evaluation for surgical treatment for aortic expansion. Two patients with severely calcified aorta had rupture at a maximum aortic diameter of less than 60 mm. All these 6 patients died of aortic rupture.

The actuarial survival rates were 97, 83 and 60%, freedoms from open aortic surgery were 96, 91 and 86%, aortic intervention were 73, 66 and 63% and aortic events were 71, 60 and 52% at 1, 5 and 10 years, respectively, in the entire cohort. In the group with the primary entry at the outer curvature of the distal aortic arch, the actuarial survival rates were 94, 88 and 63%, freedoms from open aortic surgery were 92, 83 and 76%, aortic intervention were 51, 43 and 38% and aortic events were 49, 37 and 33% at 1, 5 and 10 years, respectively. In the group with the primary entry at the inner curvature, the actuarial survival rates were 96, 73 and
59%, freedoms from open aortic surgery were 96, 96 and 89%, aortic intervention were 67, 59 and 59%, and aortic events were 59, 49 and 49% at 1, 5 and 10 years, respectively. In the group with the primary entry downstream, the actuarial survival rates were 98, 83 and 57%, freedoms from open aortic surgery were 98, 94 and 91%, aortic intervention were 86, 79 and 77% and aortic event rates were 85, 74 and 63% at 1, 5 and 10 years, respectively (Figs 1–4).

A total of 100 interventions were performed on 75 patients for rupture, malperfusion, retrograde extension of the dissection or aortic dilatation during the study period. The proportion of open surgery to transcatheter procedure was 0.2 (12/60) within 2 years after onset and 3 (21/7) after the second year of onset (P < 0.0001).

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The bivariate analysis showed that younger age, the primary entry at the outer curvature of the distal aortic arch, maximum aortic diameter and Marfan syndrome were significantly correlated with open aortic surgery, aortic intervention and aortic events (Table 3). Collagen disease or haemodialysis showed no significant correlation with any parameters.

Analysis using a Cox proportional hazard model indicated that, after adjusting for age, the primary entry site and maximum aortic diameter, the entry site and survival were not significantly correlated, but that the entry site was significantly correlated with freedoms from open aortic surgery, aortic intervention and aortic events. Patients with the primary entry at the outer curvature of the distal aortic arch had a significantly shorter time free from aortic intervention, compared with those with the primary entry at the inner curvature (Table 4).

Of the 127 patients who had a primarily thrombosed descending false lumen, 29 developed aortic events, and the primary entry at the outer curvature was significantly correlated with aortic events (P = 0.043). Of the 97 patients with a patent false lumen at the initial presentation, 53 underwent aortic intervention and the primary entry at the outer curvature was significantly correlated with aortic intervention (P = 0.026).

<table>
<thead>
<tr>
<th>Case</th>
<th>Entry</th>
<th>Aortic diameter at onset of dissection (mm)</th>
<th>Aortic diameter at onset of rupture (mm)</th>
<th>Time to rupture</th>
<th>Expansion rate (mm/year)</th>
<th>Rupture site</th>
<th>CT findings</th>
<th>Patient condition before rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>D</td>
<td>35</td>
<td>82</td>
<td>9 y</td>
<td>5.2</td>
<td>Distal arch</td>
<td>Rupture of a distal arch true aneurysm with cured aortic dissection</td>
<td>Refused surgery for distal arch true aneurysm</td>
</tr>
<tr>
<td>2</td>
<td>I</td>
<td>40</td>
<td>60</td>
<td>1 y 10 m</td>
<td>10.9</td>
<td>Ascending</td>
<td>Type A dissection Haemopericardium Retroperitoneal haemorrhage</td>
<td>Under preoperative evaluation for surgery</td>
</tr>
<tr>
<td>3</td>
<td>D</td>
<td>33</td>
<td>65</td>
<td>4 y 7 m</td>
<td>7.0</td>
<td>Thoracoabdominal</td>
<td>Haemorrhage</td>
<td>Under preoperative evaluation for surgery</td>
</tr>
<tr>
<td>4</td>
<td>D</td>
<td>38</td>
<td>56</td>
<td>8 y 2 m</td>
<td>2.2</td>
<td>Descending</td>
<td>Mediastinal haemorrhage Severely calcified aorta Left haemothorax</td>
<td>Periodical follow-up</td>
</tr>
<tr>
<td>5</td>
<td>I</td>
<td>34</td>
<td>62</td>
<td>4 y 8 m</td>
<td>6.0</td>
<td>Distal arch</td>
<td></td>
<td>Under preoperative evaluation for surgery</td>
</tr>
<tr>
<td>6</td>
<td>O</td>
<td>39</td>
<td>53</td>
<td>4 y 10 m</td>
<td>2.9</td>
<td>Distal arch</td>
<td>Mediastinal haemorrhage Severely calcified aorta</td>
<td>Periodical follow-up</td>
</tr>
</tbody>
</table>

O: outer curvature; I: inner curvature; D: downstream; y: years; m: months.

Table 1: Description of the subjects (N = 224)

<table>
<thead>
<tr>
<th>Entry Overall</th>
<th>Outer curvature</th>
<th>Inner curvature</th>
<th>Downstream</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(N = 224)</td>
<td>(N = 67)</td>
<td>(N = 27)</td>
<td>(N = 130)</td>
<td></td>
</tr>
<tr>
<td>Male/female</td>
<td>168/56</td>
<td>55/12</td>
<td>20/7</td>
<td>93/37</td>
</tr>
<tr>
<td>Age (years ± SD)</td>
<td>64.2 ± 12.6</td>
<td>60.2 ± 12.8</td>
<td>61.5 ± 14.0</td>
<td>66.8 ± 11.5</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>6</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Collagen disease</td>
<td>8</td>
<td>2</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Haemodialysis</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>DeBakey IIIB/IVB</td>
<td>69/155</td>
<td>10/57</td>
<td>5/22</td>
<td>54/76</td>
</tr>
<tr>
<td>Descending false lumen</td>
<td>97/127</td>
<td>52/15</td>
<td>19/8</td>
<td>26/104</td>
</tr>
<tr>
<td>Patent/thrombosed</td>
<td>4 (2%)</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Thirty-day mortality</td>
<td>17 (8%)</td>
<td>12</td>
<td>4</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2: Description of the cases with late aortic rupture who were ambulanced to Kitasato University Hospital (N = 6)
Not all patients with acute type B aortic dissection show late aortic expansion. The International Registry of Acute Aortic Dissection (IRAD) Investigators reported that approximately 59% of patients with acute type B aortic dissection developed late aortic expansion during a 2.0-year median follow-up [9]. The reported expansion rates varied from a mean of 1.7–3.1 mm/year [9, 10]. An acute type B aortic dissection without aortic enlargement at the onset may take more than 10 years to become large enough to warrant

**DISCUSSION**

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intervention. In our study, among the 24 patients with late open aortic surgery, 8 underwent surgery after more than 10 years of the onset. Thus, a long-term follow-up is required to investigate the nature of the acute type B aortic dissection. However, there have been no large-scale studies on the management of acute type B aortic dissection with a mean follow-up of more than 10 years.

The effect of the primary entry site on complications of acute type B aortic dissection is not well studied. Loewe et al. [11] reported that the primary entry at the inner curvature is associated with a greater risk of complications, including retrograde dissection in the acute phase. Our results, however, were not consistent with their findings; among the 7 patients who developed retrograde extension of the dissection, 6 who developed it within 10 days, had the primary entry at the outer curvature and 1 who developed it at 22 months after the onset, had the primary entry at the inner curvature. Furthermore, the primary entry at the outer curvature was significantly correlated with late open aortic surgery, aortic intervention and aortic events. In the future, progress in computational fluid dynamics using electrocardiographic-gated CT angiography or 4D phase-contrast magnetic resonance [12] would elucidate the mechanism underlying the influence of the wall shear stress created by different locations of the primary entry on aortic expansion. The primary entry at the outer curvature is also associated with failure in TEVAR in patients with chronic aortic dissection [7]. In such patients, if the aortic dissection is uncomplicated, aggressive TEVAR in the acute phase is not recommended.

Table 3: Factors for open aortic surgery, aortic intervention and aortic event (N = 224)

<table>
<thead>
<tr>
<th></th>
<th>Open aortic surgery</th>
<th>Aortic intervention</th>
<th>Aortic event</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
<td>P-value</td>
</tr>
<tr>
<td>Female/male</td>
<td>50/150</td>
<td>6/18</td>
<td>1.000</td>
</tr>
<tr>
<td>Age (years)</td>
<td>64.9 ± 12.1</td>
<td>58.1 ± 14.6</td>
<td>0.012*</td>
</tr>
<tr>
<td>Entry (inner curvature and descending/out outer curvature)</td>
<td>147/53</td>
<td>10/14</td>
<td>0.0035*</td>
</tr>
<tr>
<td>Maximum aortic diameter (mm)</td>
<td>35.3 ± 5.3</td>
<td>40.1 ± 8.5</td>
<td>0.0003*</td>
</tr>
<tr>
<td>DeBakey IIIa/IIIB</td>
<td>66/134</td>
<td>3/21</td>
<td>0.059</td>
</tr>
<tr>
<td>Marfan/non-Marfan</td>
<td>3/196</td>
<td>3/21</td>
<td>0.018*</td>
</tr>
</tbody>
</table>

*P < 0.05.

A large aortic diameter at the onset of acute type B dissection is a risk factor of poor prognosis [13], as was suggested in the present study, although a recent report indicated that a small aortic diameter was associated with increased aortic expansion [9]. In addition, in TEVAR in patients with chronic type B aortic dissection, a large aortic diameter is associated with a lower chance for late aortic remodelling [7].

Conventional open surgery in patients with chronic type B aortic dissection, especially for those with thoracoabdominal aortic expansion, is still associated with a significant risk of mortality and morbidity, with a reported in-hospital mortality of 7.0-13.4% [4, 14]. Therefore, there has been a drive to expand the indication of TEVAR to patients with uncomplicated aortic dissection in order to prevent late aortic dilatation. To date, there are only two ongoing prospective, randomized studies that evaluated the effect of prophylactic stent grafting on patients with uncomplicated type B aortic dissection with favourable early and mid-term outcomes [5, 15–17]. However, the INSTEAD trial in which stent grafting was performed in patients with subacute and chronic type B aortic dissection only showed a 5-year outcome [6], and the ADSORB trial in which stent grafting was performed in patients with acute type B aortic dissection only showed a 1-year follow-up so far [17], indicating that both time spans were not sufficiently long.

A higher proportion of open surgery to transcatheter procedure in the later phase shown in our study may have been attributed to a certain number of cases that developed persistent aortic...
than 10% [22]. However, the early outcomes improved when com-
treated medically used to have high early mortality rates of greater
late open aortic surgery.

do that when inside the aorta, it can be divided during the possible
clamping, and the stent should be segmental, rather than braided,
patients with aortic dissection need to be self-expandable to allow

features, some of which are reported to have favourable outcomes
are a number of commercially available stent grafts with different
stent graft-induced re-dissection of the aorta [19]. Currently, there

Risks of late open aortic surgery should be considered when a
stent graft is used in patients with type B aortic dissection because
complete obliteration of the false lumen in the entire aorta is diffi-
cult to achieve with TEVAR [7]. Therefore, stent grafts used in
patients with aortic dissection need to be self-expandable to allow
clamping, and the stent should be segmental, rather than braided,
so that when inside the aorta, it can be divided during the possible
late open aortic surgery.

Patients with acute type B aortic dissection who had been
treated medically used to have high early mortality rates of greater
than 10% [22]. However, the early outcomes improved when comp-
licated patients were treated with TEVAR [23]. Recently, most
patients with type B aortic dissection survive the acute phase; in our
cohort, the 30-day mortality rate was 1.8%. It is reported that 59–
84% of patients develop aortic enlargement after developing acute
type B aortic dissection [9, 24], especially when the primary entry is
at the outer curvature of the distal aortic arch, but it often takes
more than 10 years to develop a risk of rupture. The question
whether an invasive procedure, i.e. TEVAR, instead of conservative

treatment should be indicated in patients with uncomplicated
acute type B aortic dissection remains unclear. The risks in TEVAR,
including stroke, paraplegia, retrograde extension of dissection and
renal failure, are not negligible [20, 21], and a stent graft inside the
aorta may complicate the late open aortic surgery [8]. The risk of
conventional open aortic surgery in patients with chronic dissection
of the descending aorta has recently improved, with a reported
mortality of 4.5–7.0% [4, 25]. Because TEVAR in patients with acute
type B aortic dissection is not reinforced by long-term clear evi-
dence, at present, considering the risks and benefits of this pro-
cedure, patients with acute uncomplicated type B aortic dissection
basically should still be managed medically, and those with the
primary entry at the outer curvature of the distal aortic arch should be
intensively followed up for aortic enlargement or other complications.

This study has some limitations. This was a single-centre retro-
spective study, and the mean follow-up period of 6.0 years was
not sufficiently long to evaluate the late outcomes of patients with
type B aortic dissection.

In conclusion, a primary entry at the outer curvature of the
distal aortic arch, as well as a large maximum aortic diameter, was

| Table 4: Results of Cox proportional hazard models for times to death, open aortic surgery, aortic intervention and aortic event |
|---|---|---|---|---|---|
| **Entry** |  | **Parameter** |  | **OR** |  | **95% CI** |  | **P-value** |
| Death | 0.0002* | Age (years) 1.06 | 1.03–1.09 | <0.0001* |  |  |  |  |
| O versus D |  | Outer curvature entry 1.25 | 0.63–2.39 | 0.51 |  |  |  |  |
| O versus I | 0.0013* | Age (years) 1.07 | 1.03–1.11 | 0.0002* |  |  |  |  |
| O versus I |  | Outer curvature entry 0.89 | 0.38–2.23 | 0.79 |  |  |  |  |
| O versus I |  | Maximum aortic diameter (mm) 1.03 | 0.96–1.11 | 0.38 |  |  |  |  |
| Open aortic surgery | 0.0004* | Age (years) 0.98 | 0.95–1.03 | 0.51 |  |  |  |  |
| O versus D |  | Outer curvature entry 2.81 | 1.11–7.68 | 0.030* |  |  |  |  |
| O versus I | 0.31 | Maximum aortic diameter (mm) 1.13 | 1.05–1.20 | 0.0010* |  |  |  |  |
| O versus I |  | Age (years) 0.99 | 0.95–1.04 | 0.76 |  |  |  |  |
| O versus I |  | Outer curvature entry 2.04 | 0.65–8.98 | 0.24 |  |  |  |  |
| O versus I |  | Maximum aortic diameter (mm) 1.08 | 0.97–1.19 | 0.14 |  |  |  |  |
| Aortic intervention |  | Age (years) 0.96 | 0.94–0.98 | <0.0001* |  |  |  |  |
| O versus D | <0.0001* | Outer curvature entry 3.18 | 1.91–5.41 | <0.0001* |  |  |  |  |
| O versus I | 0.0003* | Maximum aortic diameter (mm) 1.06 | 1.02–1.10 | 0.0078* |  |  |  |  |
| O versus I |  | Age (years) 0.95 | 0.93–0.98 | <0.0001* |  |  |  |  |
| O versus I |  | Outer curvature entry 2.1 | 1.07–4.55 | 0.031* |  |  |  |  |
| O versus I |  | Maximum aortic diameter (mm) 1.01 | 0.95–1.07 | 0.67 |  |  |  |  |
| Aortic event |  | Age (years) 0.97 | 0.95–0.99 | 0.0019* |  |  |  |  |
| O versus D | <0.0001* | Outer curvature entry 2.83 | 1.77–4.57 | <0.0001* |  |  |  |  |
| O versus I | 0.012* | Maximum aortic diameter (mm) 1.06 | 1.02–1.10 | 0.0029* |  |  |  |  |
| O versus I |  | Age (years) 0.97 | 0.95–0.99 | 0.0043* |  |  |  |  |
| O versus I |  | Outer curvature entry 1.68 | 0.92–3.30 | 0.094 |  |  |  |  |
| O versus I |  | Maximum aortic diameter (mm) 1.02 | 0.97–1.08 | 0.32 |  |  |  |  |

O: outer curvature; I: inner curvature; D: downstream.
*P < 0.05.
associated with a higher risk of late open aortic surgery, aortic
intervention and aortic events in patients with acute type B aortic
dissection. Considering the poor success rate of TEVAR in patients
with chronic type B aortic dissection with the primary entry at the
outer curvature, medical management for these patients should
be provided with care and conventional surgery over TEVAR
should be opted in patients with late aortic dilatation. Further
long-term studies may lead to the establishment of an appropriate
indication of treatment, and further improve the prognosis of
patients with acute type B aortic dissection.

Conflict of interest: none declared.

REFERENCES


[11] Loewe C, Czerny M, Sodeck GH, Ta J, Schoder M, Funovics M et al. A new mechanism by which an acute type B aortic dissection is primarily compli-


[16] Brunkwall J, Lammers J, Verhoeven E, Taylor P. ADSORB: a study on the effi-


[19] Feng J, Lu Q, Zhao Z, Bao J, Feng X, Qu L et al. Restrictive bare stent for prevention of stent graft-induced distal redissection after thoracic endo-


[21] Lombardi JV, Cambria RP, Nienaber CA, Chiesa R, Tebbken O, Lee A et al. Prospective multicenter clinical trial (STABLE) on the endovascular treat-


APPENDIX. CONFERENCE DISCUSSION

Dr S. Trimarchi (San Donato Milanese, Italy): That’s an interesting and also
tough issue, because we have had several publications over the last three years
about predictors of aortic growth after type B dissection. So, today you
mention the presence of tearing on the outer curvature, which is something
that we often have the opportunity to observe. On the other hand, we heard in
the early afternoon today that Martin Czerny’s group just published the oppo-
site observation. Do you have any idea, in terms of the haemodynamic situation,
what these two different conditions can be ascribed to?

Dr Tadashi: I was also wondering why we had different results. It might be
not only where the primary entry exists but also where the energy goes that
matters. Even if the patient has a primary entry at the inner curvature or con-
cavity, sometimes the false lumen goes around the true lumen towards the
apex of the aortic arch. We are going to start a study looking into the fluid dy-
namics using the vector flow mapping technique. In the near future, this kind of
fluid dynamics analysis may answer the question.

Dr Trimarchi: Yes, I’m not convinced of this because the observations are
very spread out in terms of giving us an indication. It’s not up to your paper or
Martin’s one, but that’s the fact.

Martin, you want to give your feedback?

Dr M. Czerny (Zurich, Switzerland): Yes, this was interesting, of course. What
we did not look for in what we published was the prediction of late diameter
increase. But what was clear was the association within the curvature entry
tears and malperfusion. So what was that in your observation? Was malperfu-
sion also associated?

Dr Kitamura: Well, for malperfusion there was no significant difference
between outer curvature and inner curvature. The primary entry at the distal
aortic arch was significantly correlated with malperfusion compared with the
entry downstream. But we could not find any difference between the outer
curvature and the inner curvature.
Acute type B aortic dissection is regarded as the ‘nice’ dissection as it is usually not a cardiac surgical emergency and the acute phase responds well to medical therapy in a large number of patients. Therefore, the term ‘uncomplicated’ was attributed to this group when compared with the ‘complicated’ ones presenting with malperfusion, rupture or retrograde type A aortic dissection. Primary TEVAR for patients presenting with complications has already been established and remains the treatment of choice in the vast majority of patients with complicated type B aortic dissection. However, ‘uncomplicated’ should not be confused with ‘harmless’ as many patients will develop the need for any kind of treatment—early or late. ‘Uncomplicated’ should be used as a synonym to ‘currently not in need of intervention’ and this may change sooner or later as shown by the present study [1].

Recently, a better understanding of the natural course of the disease has been gained and thereby a much better anticipation of the need for treatment is available. Every physician treating patients with acute type B aortic dissection knows the clinical scenario of an initially very stable patient being found dead soon afterwards. Recent research has identified a new sub-group at risk for early adverse events and thereby the location of the primary entry tear comes into the focus of attention. It was demonstrated that patients with a primary entry tear located at the concavity of the distal aortic arch have a substantially higher incidence of primary present or secondary developing complications such as malperfusion or retrograde type A aortic dissection/type A intramural haematoma [2]. Also the distance of the primary entry tear to the left subclavian artery is decisive; the closer the distance, the higher is the likelihood of already having or developing complications [3]. Consequently, these patients qualify for ‘early’ therapy to close the primary entry tear, aimed at prevention of these complications.

Kitamura and his group now share with us their experience, identifying a primary entry tear at the convexity of the aortic arch (which is new) as well as aortic diameter (which is known) as independent predictors of ‘late’ adverse outcome. What is not reported in their study is the time from the onset of symptoms to the time of referral and diagnosis as patients with a primary entry tear at the concavity of the distal aortic arch may either never have reached a hospital as limiting complications such as severe malperfusion or retrograde type A aortic dissection might have already occurred and thereby a natural selection process or a triage process (retrograde type A directly to cardiac surgery) may have already taken place.