Growth Rate of Aortic Diameter in Patients With Type B Aortic Dissection During the Chronic Phase

Eijun Sueyoshi, MD; Ichiro Sakamoto, MD; Kuniaki Hayashi, MD; Tetsuji Yamaguchi, MD; Tatuya Imada, MD

Background—The purpose of this study was to evaluate the growth rate of type B double-barrel aortic dissection with computed tomography (CT) and the factors influencing its enlargement.

Methods and Results—Sixty-two patients were entered into this study, and regular follow-up CT studies (mean; 49.1 months) were performed. The affected aortas and iliac arteries were divided into 5 segments (aortic arch, descending thoracic, suprarenal abdominal, infrarenal abdominal aorta, and iliac artery). Fifty-two of 62 patients (83.9%) had 1 or more segments increased in size during follow-up period. In a total of 177 segments, the presence or absence of blood flow in the false lumen and aortic diameter were evaluated on CT during the follow-up period. The factors (gender, diabetes mellitus, atherosclerotic disease, smoking, entry site in arch, initial diameter, chronic obstructive pulmonary disease, blood pressure, and age) influencing increase in the diameter and growth rate were also evaluated. Of 177 segments, 132 segments (74.6%) increased in size during the follow-up period. The presence of blood flow in the false lumen was the only significant risk factor for increase in the diameter in the univariate and multivariate analysis. The group with blood flow in the false lumen had a significantly higher mean growth rate (3.3 mm/year) than the group without blood flow (−1.4 mm/year) (P<0.0001). The growth rate of aortic dissections in thoracic aorta and abdominal aorta were 4.1 and 1.2 mm/year, respectively. There was a significant difference in the growth rate between the 2 groups (P=0.0003).

Conclusion—In type B aortic dissection, the affected aortas have shown a high incidence of enlargement during the follow-up period, and more careful follow-up study is needed for aortic dissections in the thoracic aorta. The presence of blood flow in the false lumen is the most important risk factor for aortic enlargement. (Circulation. 2004;110[suppl II]:II-256–II-261.)

Key Words: aorta ■ aneurysm ■ risk factors ■ follow-up studies ■ tomography

Generally, patients who have Stanford type B double-barrel (classic) aortic dissection (AD) without complications are treated medically, but some of the affected aortas progress to aneurysmal dilatation and rupture during the chronic phase.1–3 Recently, using noninvasive imaging techniques such as computed tomography (CT), magnetic resonance (MR) imaging, and ultrasound, aortic diseases can be followed-up closely.4–8 In comparison with MR, CT may be a better technique for evaluating aortic disease because the use of MR is limited by the restricted availability of the magnets and by considerations of patient safety while undergoing study.9 Moreover, it takes longer to obtain MR images. In comparison with ultrasound, CT can provide superior images in obese patients and is not hindered by the lungs or intestinal gas.4 Ultrasound is not useful in imaging of the thoracic aorta. As a major disadvantage of CT, contrast media cannot be used for patients with severe renal dysfunction. However, CT has a major role in the diagnosis and follow-up evaluation of aortic diseases.

Recently, several articles have reported the growth rate of aortic aneurysms measured with CT.4–8 However, to our knowledge, there have been few reports on the growth rate of AD, and the natural history of the affected aorta in patients with type B AD is not clearly understood.10 The purpose of this study was to evaluate the growth rate of type B double-barrel AD with repeated CT examinations and the factors influencing its enlargement.

Materials and Methods

Patients
In this study, patients who were followed-up nonoperatively for >1 month after the onset with sequential CT scans were studied. Sixty-eight consecutive patients had Stanford type B AD diagnosed between 1986 and 2002 in 2 hospitals (Omura Municipal Hospital and Nagasaki University Hospital). Of the 68 patients, 62 patients were entered into this study. The reasons for the 6 exceptions included 4 cases of urgent surgery (aortic rupture in 3 and organ ischemia in 1) and 2 cases of early death (aortic rupture in 2) within 1 month of the onset. There were 41 men and 21 women between the
ages of 40 and 83 years, with a mean age of 63.3±12.3 years. All patients had sudden back or chest pain. Patients with traumatic AD were excluded from this study. Patients with Marfan syndrome were also excluded. This aim was to avoid underestimating the growth rate, because the growth rate of patients with Marfan syndrome may be higher. Fifty of 62 patients (80.6%) had a history of hypertension.

Chronic obstructive pulmonary disease (COPD) was considered present if the patient reported shortness of breath on even mild exertion and had been reported to have obstructive lung disease on the basis of previous examinations of pulmonary function or other tests.1 Seven patients (11.3%) had a history of COPD. Six of 7 patients were current smokers.

The diagnosis of type B AD was established with CT. In all cases, intimal flap and contrast enhancement within the false lumen were seen on postcontrast CT. Forty-six of 62 patients (74.2%) had arch involvement of AD. The mean CT follow-up period was 49.1±48.7 months (range, 2 to 192 months).

Regular follow-up studies were performed every week during the first month, and 1 to 3 times per year thereafter. In patients with a new episode suggesting complications during follow-up periods, additional studies were performed. The CT follow-up period after surgery had been excluded from this study. All patients with hypertension were managed with beta-blockers. During follow-up period, 20 of 62 patients (32.3%) had surgery 2 to 190 months after the onset (aneurysmal dilatation in aortic rupture in 2 and/or organ ischemia in 20). Eleven of 42 patients without surgery died (from) 1 to 192 months after the onset (malignancy in 3, aortic rupture in 5, organ ischemia in 2, and renal failure in 1). The remaining 31 patients have been followed-up continuously.

CT Examination
In 62 patients, a total of 411 studies were performed with conventional (n=151) or spiral (n=260) CT scanners. The follow-up CT studies were performed with nonenhanced and enhanced CT in all patients. Enhanced CT was performed with a bolus injection of 100 mL of ionic or nonionic contrast material. CT was performed with a 8800, 9800, High Speed Advantage, Light Speed Qx/i scanner (GE Medical Systems) or Somatom Plus 4 scanner (Simens Medical Systems) generating axial images with contiguous 5-mm-thick sections from the top of the aortic arch to the abdominal aorta. In the spiral CT scanner, a power injector was used, and scanning began at 20 to 30 seconds and 120 to 150 seconds (2 phases) after the start of the injection of the contrast material.

Image Analysis
CT images were evaluated by 2 experienced (E.S. and I.S.; >10 years of experience) cardiovascular radiologists. The affected aortas were divided into 5 segments (aortic arch, 48; descending thoracic aorta, 62; suprarenal abdominal aorta, 38; infrarenal abdominal aorta, 19; and iliac artery, 10). Eight of 62 patients had AD limited to 1 segment (descending thoracic aorta). The remaining 54 patients had 2 or more affected segments. A total of 177 segments were evaluated. In this study, the aortic arch was defined as the segment between the brachiocephalic artery and the ligamentum arteriosus. The descending aorta was defined as the segment between the ligamentum arteriosus and the aortic hiatus of the diaphragm.

All 177 segments had blood flow in the false lumen on the initial CT. The presence or absence of blood flow in the false lumen and aortic diameter of each segment were evaluated during the follow-up period. The absence of blood flow in false lumen was defined as no contrast enhancement in the false lumen on postcontrast CT. Final decisions regarding the findings were reached by consensus. Comparing the growth rate of AD in the thoracic aorta with that in the abdominal aorta, we chose the higher value if AD involved 2 or more affected segments. A total of 177 segments were calculated independently if AD involved both segments of thoracic and abdominal aorta.

The initial and final CT measurements were used to calculate changes in aortic size at the same level in each segment. The largest short-axial diameter of the outer contour of the affected segment of aorta was measured (Figure a). In the aortic arch, the largest diameter perpendicular to the curvature was measured (Figure b). The diameters were measured with direct-reading calipers from hardcopy images and corrected for the appropriate scale.

The growth rate of each segment was obtained at the portion of the largest diameter on the final CT. The growth rate was calculated in the following manner: the difference in the diameter between initial (D1) and final (D2) measurements was divided by the time interval (T) between the 2 measurements, ie, growth rate=(D2−D1)/T.

Statistical Analysis
All values are expressed as mean±SD. Statistical analysis was performed on clinical and morphological variables, with the χ² or Fisher exact test used for categorical variables, and the paired t test and the Mann–Whitney U test for continuous variables.

If the expected number of cells was <5, Fisher exact test was used for categorical variables. Variables with statistical significance set at P<0.05 (2-side) were included in a multivariate logistic regression model. Estimates of risk (odd ratios) were calculated based on coefficients from the logistic models. In all tests, a value of P<0.05 was considered significant. Data analysis was performed using Stat-View J-5.0 for Windows (Abacus Concepts).

Results
Fifty-two of 62 patients (83.9%) had 1 or more segments increased in size during follow-up period. Table 1 shows the
mean initial diameter, final diameter, and growth rate of the 177 segments. In all 177 segments, the mean initial diameter and final diameter were 33.9 ± 0.7 and 39.2 ± 0.1 mm, respectively. There was a significant difference between them (P<0.0001). Of the 177 segments, 132 segments (74.6%) increased in size during the follow-up period. Forty-five segments (25.4%) remained unchanged or decreased in size. In 54 segments (30.5%), the false lumens were completely thrombosed (no blood flow) from 1 day to 72 months after the onset. However, 3 segments recanalized during the follow-up period. In remaining 51 segments (28.8%), the false lumens had been completely thrombosed until the final CT (aortic arch, 16; descending thoracic aorta, 22; suprarenal abdominal aorta, 7; infrarenal abdominal aorta, 2; and iliac artery, 3). Of the 51 segments without blood flow in the false lumen, 19 (37.3%) increased in size. In contrast, of 126 segments with blood flow in the false lumen, 113 (89.7%) increased in size during the follow-up period. Forty-five of 177 (25.4%) did not increase in size or unchanged during the follow-up period (aortic arch, 16; descending thoracic aorta, 13; suprarenal abdominal aorta, 8; infrarenal abdominal aorta, 6; and iliac artery, 2).

Of 62 patients, 28 (45.2%) had the segment with the largest diameter in the aortic arch, 33 (53.2%) in the descending aorta, and 1 (1.6%) in the suprarenal abdominal aorta. The mean largest diameter on the final CT was 49.0 ± 5.3 mm. However, 19 patients (30.6%) had the segment with the fastest growth rate in the aortic arch, 38 (61.3%) in the descending aorta, and 5 (8.1%) in the suprarenal abdominal aorta. The mean fastest growth rate was 5.2 ± 6.4 mm/year. Forty-nine of 62 patients (79.0%) had same segments with the largest diameter and the fastest growth rate. In 13 patients (21.0%), the segments with the largest diameter did not show the fastest growth rate.

We divided all 177 segments into 2 groups, with an increase or no increase in the diameter. Table 2 shows that patient characteristics (risk factors) such as gender, diabetes mellitus, atherosclerotic disease (including atherosclerotic aneurysm, ischemic heart disease, and cerebrovascular disease), smoking >20 years, entry site in arch, initial diameter >40 mm, COPD, and blood pressure >140 mm Hg during follow-up period were not significant risk factors for increase in the diameter in univariate or multivariate analysis. Age older than 60 was a significant risk factor for increase in the diameter in the univariate analysis. The presence of blood flow in the false lumen during the follow-up period was the only significant risk factor for increase in the diameter in the univariate and multivariate analysis.

Table 3 shows that the mean growth rates in the 2 groups of the segments divided by gender or by the presence or absence of characteristics such as age older than 60, diabetes mellitus, atherosclerotic diseases, smoking >20 years, no blood flow in the false lumen, entry site in the arch, initial diameter >40 mm, COPD, and blood pressure >140 mm Hg during follow-up period.

Gender, no blood flow in false lumen, and COPD were significantly different between the 2 groups; the probability values were 0.0147, <0.0001, and 0.008, respectively. Fe-

<p>| TABLE 1. Mean Initial Diameter, Final Diameter, and Growth Rate of the 177 Segments |
|-----------------------------------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Segment</th>
<th>Initial Diameter (mm)</th>
<th>Final Diameter (mm)</th>
<th>Growth Rate (mm/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic arch (n=48)</td>
<td>39.4±7.7</td>
<td>45.6±11.0</td>
<td>2.2±6.9</td>
</tr>
<tr>
<td>Descending aorta (n=62)</td>
<td>37.7±9.3</td>
<td>45.0±13.0</td>
<td>2.2±10.1</td>
</tr>
<tr>
<td>Suprarenal abdominal aorta (n=38)</td>
<td>29.6±5.3</td>
<td>33.3±5.9</td>
<td>1.0±5.8</td>
</tr>
<tr>
<td>Infrarenal abdominal aorta (n=19)</td>
<td>29.7±4.7</td>
<td>26.8±5.8</td>
<td>1.0±2.2</td>
</tr>
<tr>
<td>Iliac artery (n=10)</td>
<td>17.5±4.8</td>
<td>18.8±3.9</td>
<td>-0.4±4.7</td>
</tr>
</tbody>
</table>

<p>| TABLE 2. Risk Factors for Increase in Diameter of the 177 Segments |
|---------------------------------------------------------------|-----------------|-----------------|------------------|</p>
<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Increase (n=132)</th>
<th>No Increase (n=45)</th>
<th>Univariate</th>
<th>Multivariate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, male, n (%)</td>
<td>79 (59.8)</td>
<td>33 (73.3)</td>
<td>2.08</td>
<td>0.1492</td>
</tr>
<tr>
<td>Age &lt;60 y, n (%)</td>
<td>79 (59.8)</td>
<td>35 (77.8)</td>
<td>4.71</td>
<td>0.0301</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>8 (6.1)</td>
<td>6 (13.3)</td>
<td>1.55</td>
<td>0.2138</td>
</tr>
<tr>
<td>Atherosclerotic disease, n (%)</td>
<td>27 (20.5)</td>
<td>9 (20.0)</td>
<td>0</td>
<td>&gt;0.9999</td>
</tr>
<tr>
<td>Smoking &lt;20 y, n (%)</td>
<td>23 (17.4)</td>
<td>13 (28.9)</td>
<td>2.06</td>
<td>0.1508</td>
</tr>
<tr>
<td>Presence of blood flow in false lumen, n (%)</td>
<td>112 (85.6)</td>
<td>13 (28.9)</td>
<td>49.92</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Entry site in arch, n (%)</td>
<td>117 (84.8)</td>
<td>37 (82.2)</td>
<td>1.22</td>
<td>0.2891</td>
</tr>
<tr>
<td>Initial diameter &lt;40 mm, n (%)</td>
<td>22 (16.7)</td>
<td>7 (15.6)</td>
<td>0</td>
<td>&gt;0.9999</td>
</tr>
<tr>
<td>COPD, n (%)</td>
<td>20 (15.2)</td>
<td>2 (4.4)</td>
<td>3.35</td>
<td>0.0654</td>
</tr>
<tr>
<td>BP &lt;140 mm Hg during follow-up period, n (%)</td>
<td>9 (6.8)</td>
<td>1 (2.2)</td>
<td>0.61</td>
<td>0.4192</td>
</tr>
</tbody>
</table>

Atherosclerotic disease including atherosclerotic aneurysm, ischemic heart disease, and cerebrovascular disease.
COPD indicates chronic obstructive pulmonary disease; BP, blood pressure; OR, odds ratios.
There was a significant difference in the growth rate between the 2 groups \((P=0.0003)\), and the AD in the thoracic aorta grew at a faster rate than those in the abdominal aorta. Of 62 AD in the thoracic aorta, 48 AD involved 2 segments (arch and descending aorta). In 24 of 48, distal (descending aorta) segments grew at a faster rate than proximal (arch) segment. Of 38 AD in the abdominal aorta, 19 AD involved 2 segments (suprarenal and infrarenal abdominal aorta). In 7 of 19, distal (infrarenal abdominal aorta) grew at a faster rate than proximal (suprarenal abdominal aorta) segments.

## Discussion

The optimal treatment for patients with type B AD remains a matter of debate.\(^1\),\(^2\),\(^3\) Medical and surgical treatment strategies are used in different populations. Usually, medical treatment is selected for patients with uncomplicated type B AD, and surgical treatment is selected for patients with complicated type B AD. Those complications typically include intractable pain, rapid expansion and/or rupture, and organ ischemia. Patients without complications are usually treated with hypotensive drugs during the acute phase because the mortality rate with this treatment was reported to be equal to or slightly better than that for surgical treatment during the acute phase.\(^1\),\(^2\) Surgical treatment should be selected if the aortic diameter enlarges or if a new complication appears during the chronic phase.\(^1\),\(^2\),\(^3\) Therefore, follow-up imaging studies for patient with type B AD is essential.

In the present study, 52 of 62 patients (83.9%) had 1 or more segments increased in size, and affected aortas of type B AD had a high incidence of enlargement (74.6%) during the follow-up period. However, in 13 patients (21.0%), the segments with the largest diameter did not show the fastest growth rate. These results suggest that the segment with the largest diameter does not always show the fastest growth rate, and the proximal portion of the aorta does not always show the fastest growth rate. Recognition of these results is useful for following-up patients with type B AD.

In this study, the presence of blood flow in the false lumen was the only significant risk factor for increase in the diameter in both univariate and multivariate analysis (Table 2).

In addition, segments with blood flow in the false lumen had a significantly higher mean growth rate (Table 3). Therefore, the presence of blood flow in the false lumen is considered to be the most important risk factor for aortic enlargement. In a segment with false lumen thrombosis, the aorta frequently becomes a single-barrel chamber, with dissected layers becoming a single layer. This may restore normal vessel wall strength. In addition, these results suggest that both structural weakness of the aortic wall and direct mechanical stress from blood flow cause progressive enlargement of the segment with blood flow in the false lumen.

Previously, a few articles reported the risk factors for rupture or aortic enlargement (diameter ≥60 mm) in chronic type B AD.\(^10\),\(^11\),\(^12\) One article revealed that the presence of blood flow in the false lumen was not a risk factor for rupture of type B AD.\(^10\) However, another article revealed that the presence of blood flow in the false lumen was a significant risk factor for aortic enlargement (diameter ≥60 mm) of

### Table 3. Patient Characteristics for the Mean Growth Rate of the 177 Segments

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Mean Growth Rate (mm/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male (n=112)</td>
<td>1.2±6.6*</td>
</tr>
<tr>
<td>Female (n=65)</td>
<td>3.3±5.2*</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Age &gt;60 (n=114)</td>
<td>1.9±5.8</td>
</tr>
<tr>
<td>Age ≤60 (n=63)</td>
<td>2.1±6.8</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>Present (n=14)</td>
<td>-1.1±8.3</td>
</tr>
<tr>
<td>Absent (n=163)</td>
<td>2.3±5.9</td>
</tr>
<tr>
<td>Atherosclerotic disease</td>
<td></td>
</tr>
<tr>
<td>Present (n=36)</td>
<td>3.0±7.2</td>
</tr>
<tr>
<td>Absent (n=141)</td>
<td>1.7±5.9</td>
</tr>
<tr>
<td>Smoking &lt;20 y</td>
<td></td>
</tr>
<tr>
<td>Present (n=36)</td>
<td>1.0±9.0</td>
</tr>
<tr>
<td>Absent (n=141)</td>
<td>2.2±5.2</td>
</tr>
<tr>
<td>Thrombosed false lumen</td>
<td></td>
</tr>
<tr>
<td>Present (n=51)</td>
<td>-1.4±8.6*</td>
</tr>
<tr>
<td>Absent (n=126)</td>
<td>3.3±4.2*</td>
</tr>
<tr>
<td>Entry site in arch</td>
<td></td>
</tr>
<tr>
<td>Present (n=154)</td>
<td>1.6±7.9</td>
</tr>
<tr>
<td>Absent (n=23)</td>
<td>1.9±3.5</td>
</tr>
<tr>
<td>Initial diameter &lt;40 mm</td>
<td></td>
</tr>
<tr>
<td>Diameter &gt;40 mm (n=29)</td>
<td>1.8±6.2</td>
</tr>
<tr>
<td>Diameter ≤40 mm (n=148)</td>
<td>2.9±6.2</td>
</tr>
<tr>
<td>COPD</td>
<td></td>
</tr>
<tr>
<td>Present (n=22)</td>
<td>5.9±7.0*</td>
</tr>
<tr>
<td>Absent (n=154)</td>
<td>1.4±5.8*</td>
</tr>
<tr>
<td>BP during follow-up period</td>
<td></td>
</tr>
<tr>
<td>BP &gt;140 mm Hg (n=10)</td>
<td>3.0±3.7</td>
</tr>
<tr>
<td>BP ≤140 mm Hg (n=167)</td>
<td>1.9±6.3</td>
</tr>
</tbody>
</table>

Atherosclerotic disease including atherosclerotic aneurysm, ischemic heart disease, and cerebrovascular disease.

*Significant difference between the 2 groups.

COPD indicates chronic obstructive pulmonary disease; BP, blood pressure.

### Table 4. Mean Initial Diameter, Final Diameter, and Growth Rate of 100 Lesions of the Thoracic and Abdominal Aorta

<table>
<thead>
<tr>
<th></th>
<th>Initial Diameter (mm)</th>
<th>Final Diameter (mm)</th>
<th>Growth Rate (mm/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic aorta, n=62</td>
<td>39.6±9.9</td>
<td>48.7±13.7</td>
<td>4.1±6.5</td>
</tr>
<tr>
<td>(Aortic arch, n=48)</td>
<td>(39.4±7.7)</td>
<td>(45.6±11.0)</td>
<td>(2.2±9.3)</td>
</tr>
<tr>
<td>Abdominal aorta, n=38</td>
<td>28.7±5.4</td>
<td>32.5±6.1</td>
<td>1.2±5.9</td>
</tr>
</tbody>
</table>
chronic type B AD. Although the results in these 2 articles were different, our results support the latter.

In this study, the presence of COPD was not a significant risk factor for increase in the diameter in the univariate or multivariate analysis (Table 2). However, segments in patients with COPD had a significantly higher mean growth rate than those without COPD (Table 3). Previous reports revealed that a history of COPD was a powerful predictor for rupture of aortic aneurysms. In this study, it is not clear that the presence of COPD is a sensitive indicator of intolerance in connective tissue in patients with AD. Further studies are needed.

Our results showed that age was a significant risk factor for increase in the diameter in the univariate analysis. According to previous studies, the risk that an aneurysm or AD will rupture increases with age. However, in this study, the incidence of increased diameter of the segments in patients 60 or younger was higher than in patients older than 60 (Table 2). In addition, segments in female patients had a significantly higher mean growth rate (Table 3). The reasons for these results are unknown. Anatomically, the elasticity and distensibility of the aorta decline with age. Such changes occur even in normal healthy adults and, for some reasons, these changes appear earlier and are more progressive in males than in females. We hypothesize that the loss of elasticity may limit enlargement of the aorta in patients with AD. However, further studies are needed to clarify this issue.

Previous studies revealed that aortic diameter was a predictor of rupture and increase in the diameter of aortic aneurysm, AD, and aortic intramural hematoma. These results can be explained by the La Place Law. It states that the perpendicular stress on a cylinder is directly proportional to the pressure exerted by the fluid contents and its radius and is inversely proportional to wall thickness. This means that the larger the diameter, the faster the growth rate if the pressure is constant. However, in our results, initial aortic diameter was not a significant risk factor for increase in the diameter. As a hypothesis, the perpendicular stress may be weaker in AD than aortic aneurysms and intramural hematomas, because the aortic lumen of AD is separated and the diameter of each true and false lumen is small. However, patients with AD usually undergo a more aggressive blood control than patients with aortic aneurysm. Further study is needed to clarify why initial aortic diameter was not a significant risk factor for increase in the diameter.

In the present study, there was a significant difference in the growth rate between the AD in the thoracic aorta and the AD in the abdominal aorta. The AD in the thoracic aorta grew at a faster rate than those in the abdominal aorta (Table 4). Regarding growth rate of AD, we found only 1 previous study that demonstrated the growth rate in no rupture or operation, rupture, and operation groups, respectively. However, this article does not show the mean growth rate of AD in all patients and does not include aortic arch AD. It may not be appropriate to compare these results with our results.

In contrast, there are several articles on the growth rate of aortic aneurysms, but most of them are related to abdominal aortic aneurysm (AAA). According to these results, the growth rate of AAA had a range of 2.2 to 5.7 mm/year. Although AD and aortic aneurysm ultimately have different natural histories, the growth rate of the abdominal aorta in our results was 1.2 mm/year, which was slower than previous reports. For this reason, the aortic diameter of AD may usually be smaller than that of AAA. However, there were only a few reports on the growth rate of thoracic aortic aneurysm (TAA) that demonstrated that the growth rate had a range of 1.3 to 4.3 mm/year. In the present study, the growth rate of AD in the thoracic aorta was 4.1 mm/year, and the ADs in the thoracic aorta grew at a faster rate than those in the abdominal aorta. These results were similar to some results in previous studies. One study showed that TAA significantly grew at a faster rate than AAA. In contrast, another study showed that the mean growth rate of TAA was significantly lower than that of AAA. In fact, it is still unclear whether the mean growth rate of TAA is significantly lower than that of AAA. Some articles suggested that the mechanism of slow growth rate of TAA could be explained by the aortic structure itself. The lamellar unit and vasa vasorum are normally absent in the human abdominal aortic media. Therefore, elevated tension per lamellar unit and excessive avascular media may be factors in the increased growth rate of AAA. However, in AD, the aortic wall is separated and directly damaged more than an aortic aneurysm. Our results suggest that the damage to the aortic wall by AD and mechanical stress are also great contributing factors for growth rate because the lesions in the thoracic aorta grow at a faster rate than those in the abdominal aorta.

A limitation of this study was the difficulty in obtaining accurate measurements of aortic diameter on axial CT images. We measured the largest short-axial diameter of the outer contour of the aorta to avoid any errors caused by tortuosity or the curvature of some aortas so that the measurement would be accurate on axial CT images. Although previous studies used a similar measurement method to ours, multiplanar reconstructed images or other modalities such as MR imaging and transesophageal echocardiography may be needed to obtain more accurate sizing of the aorta. Moreover, aortic growth in patients with AD is not always circular. The measurement of this study may underestimate growth rate in some cases. As another limitation, other anatomic factors such as the size of the primary tear, circumferential extent of dissection, and presence or absence of adequate re-entry tears that may contribute to late complication were not addressed in this study. As other limitations, the sample size was small, and the follow-up periods of patients varied. Further studies addressing other anatomic factors and involving larger numbers of patients with a longer follow-up period are needed.

**Conclusion**

In type B AD, the affected aortas have a high incidence of enlargement during follow-up period. The presence of blood flow in the false lumen is the most important risk factor for enlargement during the follow-up period. The ADs in the thoracic aorta can grow at a faster rate than those in the abdominal aorta. Therefore, more careful follow-up study is needed for ADs in the thoracic aorta. Additionally, we should keep in mind that the largest diameter or the proximal portion of the aorta does not always show the fastest growth rate.
References
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