Coexistent True Aortic Aneurysm as a Cause of Acute Aortic Dissection

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Background: Aortic aneurysms are found in 5–20% of patients with acute dissecting aortic aneurysm (AAD). Coexisting aortic aneurysms might potentially influence the incidence of AAD. The purpose of this study was to elucidate the role of coexistent aortic aneurysms in AAD.

Methods and Results: A total of 140 patients with AAD were enrolled in the present study. Clinical characteristics of the patients were evaluated in relation to the locations of aortic segments affected by the dissection as well as of the coexistent aortic aneurysm. Among the 140 study patients, 34 (24%) had true aortic aneurysms. Patients with coexistent aortic aneurysm were significantly older than those without (72±11 years vs 65±14 years, P=0.012) and had higher incidence of thrombosed false lumen (62% vs 38%, P=0.017), and coronary artery disease (26% vs 8%, P=0.006). Twenty-two of these 34 (65%) patients had a thoracic aortic aneurysm (TAA), and this frequency of TAA was much higher than that observed in the general population. Furthermore, among all patients with AAD, 12 patients (9%) might be associated with development of AAD.

Conclusions: The current study showed that nearly one-quarter of AAD patients had coexisting true aortic aneurysms, and suggests that TAA are likely to be associated with development of AAD. (Circ J 2009; 73: 822–825)

Key Words: Aortic aneurysm; Aortic dissection; Atherosclerosis; Etiology

A acute aortic dissection (AAD) is a potentially catastrophic condition. The etiology of AAD, however, has not been fully elucidated. Cambria et al reported that 18 of 325 (5.5%) patients with AAD had either a thoracic aortic aneurysm (TAA) or an abdominal aortic aneurysm (AAA).† Davies et al also reported that the long-term outcome of patients with TAA is determined by not only aortic rupture but also development of AAD. These findings suggest that coexistent aortic aneurysms might play an important role in the development of AAD. Accordingly, in the present study, we investigated the role of coexistent true aortic aneurysms as a cause of AAD.

Methods

Between January 2004 and December 2006, a total of 150 consecutive patients were admitted to our hospital within 14 days after the development of AAD. After exclusion of Marfan syndrome (n=3), bicuspid aortic valve (n=1), and previous aortic surgery (n=6), 140 patients were enrolled in the study.

The diagnosis of AAD and coexistent aortic aneurysm was made by computed tomography (CT) scan using Aquilion Multislice System or Aquilion 16 (Toshiba, Tokyo, Japan). AAD was classified according to the Stanford classification, and was also classified according to the status of false lumen. Patent type was defined as having a false lumen, which was identified by opacification of at least a portion with contrast media, and included partial thrombosis. Thrombosed type was defined as having complete thrombosis of false lumen with thrombus. Ulcer-like projection (ULP) was defined as a localized blood-filled pouch protruding from the true lumen to the thrombosed false lumen. The definitions of aortic aneurysm were fusiform type dilatation with the maximal diameter ≥45 mm for the ascending aorta, 40 mm for the aortic arch and descending aorta, and 30 mm for the abdominal aorta (thoracoaortic and thoracoabdominal aortic aneurysms were classified as descending TAA); and saccular type dilatation regardless of the size or the location. Distinction between coexistent aortic aneurysm and dissecting aortic aneurysm was made based on the findings of 3-dimensional CT reconstruction and was clear in all cases studied (Figure 1).

To determine the role of aortic aneurysm in the development of AAD, patients were divided into 2 groups; those with coexistent aortic aneurysms (n=34, 24%) and those without (n=106, 76%). Demographics and clinical characteristics, including age, gender, Stanford classification, the status of false lumen, diabetes mellitus, hypertension, dyslipidemia, and smoking history were compared. Coronary artery disease, peripheral artery disease, cerebral vascular disease, and chronic obstructive pulmonary disease were...
also compared. In addition, patients were classified according to the locations of AAD and coexistent aortic aneurysms. One type was aortic aneurysm located at the proximal or distal ends of AAD (type PD: proximal or distally located) (Figure 2A), and the other type was aortic aneurysm located away from the AAD (type non-PD) (Figure 2B). The type PD was divided into 3 types: TAA located at the proximal or distal ends of the AAD; AAA located at the proximal or distal ends of the AAD; and AAD located between the TAA and AAA. Intimal tear of the AAD was suspected because of the presence of the proximal or distal ends of the patent segment of partial thrombosed type AAD3 or ULP of the thrombosed type AAD5,6.

Diabetes mellitus was defined as fasting plasma glucose ≥126 mg/dl, non-fasting plasma glucose ≥200 mg/dl on 2 occasions, or current antidiabetic medications. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or use of antihypertensive medication. Dyslipidemia was defined as a fasting serum total cholesterol concentration of ≥220 mg/dl, low density lipoprotein cholesterol concentration of ≥140 mg/dl, triglyceride concentration of ≥150 mg/dl, high density lipoprotein cholesterol concentration of ≤40 mg/dl, or use of lipid-lowering medications. Smoking history included past or current smoking. Coronary artery disease was defined as history of definite myocardial infarction or coronary artery revascularization, or >50% luminal diameter narrowing by quantitative coronary angiography in at least 1 of the major branches. Peripheral artery disease was defined as history of claudication, amputation for arterial insufficiency, peripheral vascular surgery or angioplasty, or an ankle brachial index <0.9. Cerebral vascular disease was defined as a history of clinically evident neurological symptoms or signs suggestive of cerebral infarction, which were confirmed by imaging studies.
such as CT and magnetic resonance imaging. Chronic obstructive pulmonary disease was defined as a ratio of forced expiratory volume in one second/forced vital capacity ≤70% or use of bronchodilators.

**Statistical Analysis**

Continuous data were expressed as mean±standard deviation (SD), and categorical data were expressed as frequencies and percentages. For categorical variables comparisons between 2 groups were performed with Fisher’s exact test. Student’s t-test was used for comparisons of continuous variables. All statistical analyses were performed using the software package SPSS (version 11.0J). For all analyses, a value of P<0.05 was considered statistically significant.

### Results

The clinical and demographic characteristics of the 140 patients are shown in Table. The mean age was 67±14 years, and 86 (61%) were men. Seventy-eight (56%) patients had Stanford type B dissection, and 61 (44%) was thrombosed type dissection. Among the 140 patients, 34 (24%) patients had coexistent true aortic aneurysm. Comparing patients with coexistent aortic aneurysm and those without, there was significantly higher incidence of older age (72±11 years vs 65±14 years, P=0.012), thrombosed type dissection (62% vs 38%, P=0.017), and coronary artery disease (26% vs 8%, P=0.006), respectively. Among 34 patients with coexistent aortic aneurysms, 16 (47%) had TAA alone, 12 (35%) had AAA alone, and 6 (18%) had both TAA and AAA. The total number of patients with TAA was 22 (65%) and that with AAA was 18 (53%).

All 34 patients with coexisting aortic aneurysm were classified according to the locations of the AAD and coexisting aortic aneurysms: type PD (n=28) and type non-PD (n=6). Furthermore, type PD were divided into 3 categories: TAA located at the proximal or distal ends of AAD (n=8); AAA located at the proximal or distal end of AAD (n=8); and AAD located between the TAA and AAA (n=1). Among 34 AAD patients with coexisting aortic aneurysm, the status of false lumen was 21 thrombosed type and 13 patent type including 10 partial thrombosis of false lumen. Five patients of 21 thrombosed type had ULP locating very close to the site of the TAA, 7 of 10 partial thrombosis of false lumen had patent segments of partial thrombosis AAD that were located very close to the site of TAA, and these sites were considered as intimal tear. In total, 12 patients of all 140 AAD (9%) patients were suspected of an association between the TAA and development of AAD. No retrograde dissections were suspected in these 28 patients.

### Discussion

In 1988, Cambria et al reported that 5.5% out of 325 patients with spontaneous aortic dissections had coexistent TAA or AAA. In 1991, Roberts et al reported similar incidence of coexistent aortic aneurysm in patients with aortic dissection. In a study of 182 autopsies of patients with aortic dissection, 13 (7.1%) had coexistent AAA. However, more recent reports by other investigators, as well as ourselves, have indicated a higher incidences of coexistent aortic aneurysms in patients with AAD. For example, in 2002, Mehta et al reported aortic aneurysms in 12.4% of 550 patients with acute type A dissection. In 2007, Tsai et al reported an incidence as high as 21% of coexistent aortic aneurysms in 195 patients with acute type B dissection. As reported by Tsai et al, we found coexistant true aortic aneurysms in 24% of patients with AAD. These data support the notion that the incidence of coexistent aortic aneurysm in patients with AAD might be increasing, nearly 4-fold during the past 2 decades. One of the probable explanations for this increase is an increase in the average life span. If the percentage of older patients increases, there would be an associated increase in the percentage of patients with atherosclerosis, which would be expected to result in an increase in the percentage of patients with aortic aneurysm especially with...
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An alternative explanation is that detection of AAD, especially thrombosed type AAD with small false lumen, might have increased because of the improvement in the performance of CT scanning technology.

In the present study we found that coexisting aortic aneurysm, especially TAA, is likely to be associated with development of AAD. The most important matter under consideration is the association between atherosclerosis and the development of AAD. Is development of aortic dissection in the patients with coexisting aortic aneurysm a coincidental phenomenon in the elderly? In the current study, we found that patients with coexistent aortic aneurysm were significantly older than those without. In addition, patients with coexistent aortic aneurysm had a higher incidence of coronary artery disease than those without. These findings suggest that patients with coexistent aortic aneurysm might have more advanced atherosclerosis than those without. There could be 2 explanations for these findings. One is the direct effect of atherosclerosis of the aortic wall on the development of AAD and the other is the effect of atherosclerosis in the aortic aneurysm wall on the development of AAD.

A necropsy study of 26,251 patients performed by Brunkwall et al indicated that, among patients with aortic aneurysms, AAA was 3–4 times more frequent than TAA. In contrast, we found more TAA than AAA among patients with coexistent aneurysms in the present study. We previously reported that AAA is more likely to be associated with atherosclerosis than TAA. These facts imply that only atherosclerosis does not contribute to the development of AAD. Furthermore, of all 140 AAD patients, 28 (20%) with coexisting aortic aneurysm were associated with termination or development of AAD and in approximately 10% it was suspected that TAA was associated with the development of AAD. These findings show the effect of atherosclerosis in the aortic aneurysmal wall on the development of AAD, although there might be a direct effect of atherosclerosis of the aortic wall as well.

Roberts et al previously suggested that an atherosclerosis plaque frequently serves to terminate the dissection process. In this regard, aortic aneurysms could serve as the terminator of aortic dissection. The mechanism by which coexisting aortic aneurysm affect the development of aortic dissection could not be clearly elucidated in the present study. However, one possible notion deserves mention. Past pathological studies have shown that the wall of an aneurysmal aortic segment has decreased collagen synthesis, reduced elastin content, and a thinner wall. These biological alterations of the aorta predispose the entire aorta and its branches to dissection.

Some limitations of the present study must be taken into account. First, we did not provide pathological proof of the intimal tear of aortic dissection in the aortic aneurysm. Therefore we could not state emphatically that a coexisting aortic aneurysm affects the development of aortic dissection. However, if we could consider the edge of the dissected segment as the intimal tear and it was located very close to the aortic aneurysm on CT findings, we could consider it a possibility that aortic aneurysm affects the development of aortic dissection. Second, we suspected the patent segment as intimal tear in partial thrombosis AAD based on the report by Tsai et al which have proposed that false lumen where thrombus formation was observed were considered as terminal side of AAD and patent segment in opposite side was considered as intimal tear of AAD. However, the notion was not completely established.

In conclusion, nearly one-quarter of AAD patients had coexistent true aortic aneurysms and TAA is likely to be associated with development of AAD.

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References