Aortogenic Embolic Stroke: A Transesophageal Echocardiographic Approach

Kazunori Toyoda, MD; Masahiro Yasaka, MD; Seiki Nagata, MD; and Takenori Yamaguchi, MD

Background and Purpose: We studied the frequency and grade of atherosclerotic changes in the thoracic aorta and their significance as embolicogenic lesions in patients with stroke.

Methods: Using transesophageal echocardiography, we evaluated complicated lesions in the thoracic aortas of 62 patients who met our clinical criteria for embolic stroke. A complicated lesion was defined as a raised lesion with an irregular surface or acoustic shadow based on a comparative study of echocardiographic and histopathologic findings. The results were compared with plain radiographic findings and in some cases with computed tomographic or aortographic findings.

Results: Twenty-six patients (42%) showed complicated aortic arch lesions on echocardiogram. Transesophageal echocardiography brought us more abundant information than other techniques. Aortic knob calcification by plain radiography correlated well with the presence of echocardiographically complicated lesions, suggesting its utility for rough screening of atherosclerotic changes. Fifty-two patients had other potential embolic sources in the heart or cervical arteries. Among the remaining 10 patients without extra-aortic embolic sources, three showed complicated lesions and were diagnosed as having aortogenic embolic stroke.

Conclusions: Aortic atherosclerosis should be recognized as an embolic source of stroke and the advantage of transesophageal echocardiography recognized in its evaluation. (Stroke 1992;23:1056-1061)

KEY WORDS • cerebrovascular disorders • echocardiography • embolism

The heart and cervical arteries have drawn considerable attention during investigation of the mechanism of ischemic stroke. Investigation of the thoracic aorta, however, has not been emphasized as a potential source of emboli despite pathological evidence that cholesterol emboli from aortic atheroma can cause stroke.1-6 This lack of investigation may result in part from the absence of suitable diagnostic techniques for evaluating the aorta in vivo.

Transesophageal echocardiography (TEE), a recently developed imaging technique, provides clear information about the thoracic aorta.7 However, only a few investigations emphasizing the importance of aortic lesions in stroke, which can be detected by TEE, have been performed.8-10 This study describes our attempts to clarify the importance of atherosclerotic changes in the aorta as an embolic source of stroke using this new technique.

Subjects and Methods

Forty-two patients with acute embolic stroke were admitted to the Stroke Care Unit of the National Cardiovascular Center from June 1, 1990, to May 31, 1991. Forty of them were examined by TEE. The remaining two patients were too ill to undergo this technique and died within 1 week of admission. We also performed TEE in another 22 patients with chronic embolic stroke. A total of 62 patients, 41 men and 21 women, ranging in age from 24 to 87 (mean±SD, 63.7±13.5) years were evaluated. The diagnosis of embolic stroke was made if the patients met at least two of the following criteria based on our previous reports11-12: 1) sudden onset of clinical symptoms with maximal focal neurological deficit at onset; 2) evidence of embolization in other parts of the body; 3) angiographic features such as the visualization of an embolus and reopening of a previously occluded vessel; and 4) computed tomographic features such as a sharply marginated hypodense area involving the cortex and hemorrhagic infarction. Patients meeting the above four criteria numbered 62, 8, 30, and 50, respectively. Those who had significant atherosclerotic lesions in the affected cervical or intracranial arteries (luminal stenosis of more than 50%) were excluded from this study even if they satisfied the above criteria. Baseline characteristics of the subjects were obtained with regard to the risk factors for atherosclerosis,13-14 such as hypertension, hypercholesterolemia, hypo-HDL-cholesterolemia, diabetes mellitus, and cigarette smoking.

Transesophageal echocardiography was performed within 4 weeks (median, 7 days) after onset of stroke in all 40 patients admitted in the acute stage. In the 22 patients with chronic-stage stroke, examination was...
FIGURE 1. Echocardiographic (top panels) and histopathologic (bottom panels) findings of the thoracic aorta in autopsy specimens. Each echographic finding corresponds to the following histopathologic lesion: A, normal finding; B, diffuse intimal thickening; C, simple atheroma; and D, complicated lesion. Only echographic finding D was considered significant in this study.

carried out within 2 months of stroke onset in 11, 2–3 months in four, and 3 months or more in seven patients.

We used a commercially available real-time, two-dimensional system (model SSD-870, Aloka, Tokyo) with a 5.0-MHz phased array biplane transesophageal transducer (model UST-5228-5, Aloka) which provides both biplane B-mode views and color Doppler flow. After local anesthesia of the hypopharynx with xylocaine, patients were put in the left lateral decubitus position, and the probe was inserted into the esophagus. We observed the thoracic aorta as deep as 40 cm from the incisors with both transverse and sagittal views. The severity of atherosclerotic involvement was judged in each segment of the aorta: the ascending aorta, aortic arch, and descending aorta. The most advanced lesion was used to judge the severity of the disease.

Before the present study, we investigated the echographic findings of the pathologically verified complicated lesion in the aorta (Figure 1). From five autopsied men (28–76 years old) who died from cerebrovascular or cardiovascular diseases, we observed 40 areas of aortas (soaked in physiological saline) using the same TEE system. Echographic findings such as the intimal-medial thickness, presence or absence of surface irregularity, and acoustic shadow were recorded from each area. A localized raised lesion with a maximal intimal-medial thickness of more than 3.0 mm and a markedly irregular surface or broad acoustic shadow was defined as an echographically significant lesion. Eighteen of the 40 areas satisfied the above criteria. Histopathologic evaluation was performed by a qualified pathologist before the designation code was revealed and was compared with the echographic findings. Pathological study showed ulceration of the luminal surface (14 lesions) and/or massive calcification (8 lesions) in 17 of the above 18 lesions, and they were diagnosed as complicated lesions. Two severely ulcerated plaques with the pealed surface showed on echography the slender, stringlike findings swinging in the lumen. In the remaining 22 areas that were not diagnosed as echographically significant, six had ulceration with rather smooth surfaces or small patchy calcification. These six lesions were also diagnosed pathologically as complicated lesions. Therefore, sensitivity of echographic detection of the complicated lesion was 74% and specificity was 94%.

We also evaluated atherosclerotic changes of the thoracic aorta using plain radiography in 62, computed tomography (CT) in 15 (all with contrast enhancement), and aortography in 31 patients. Seven patients were examined by ultrafast CT (UFCT scanner C-100, Imatron, South San Francisco, Calif.). Aortography was performed in the left oblique position (inclined at 45–55°) with iodinated contrast medium.

All patients underwent precordial echocardiography, TEE, and continuous electrocardiography for detection of potential embolicogenic heart diseases or arrhythmia. Cervical and intracranial arteries were evaluated in 52 patients by cervical duplex echography (model SSA-270A with 7.5-MHz transducer, Toshiba Inc., Tokyo) and in 46 by cerebral arteriography. An echographic plaque more than 1.0 mm thick or arteriographic steno-
 sis of more than 25% was considered a potential embolic source. The presence of the raised plaques in the intracranial arteries of the affected side were also carefully reviewed on angiogram.

A $\chi^2$ test and unpaired $t$ test were used for statistical analysis. A value of $p<0.05$ was considered statistically significant.

**Results**

The TEE procedure was well tolerated without any complications. We obtained clear aortic views throughout the thoracic aorta except for the upper ascending aorta hidden behind the trachea and the bronchus (although the view of the aortic wall close to the transducer was not as clear because of its focal length).

An echographically significant lesion in the aortic arch was detected in 26 patients (42%) (Figure 2), 20 of 40 admitted in the acute stage (50%) and 6 of 22 in the chronic stage (27%). There was no significant difference in the frequency of significant lesions between the two groups ($\chi^2$ test). The significant lesion was found in the ascending aorta in one patient (2%) and in the descending aorta in 12 (19%).

In the aortic arch, marked thickening of the intima-media complex was frequently seen in patients with significant lesions: in 21 patients thickness was more

**FIGURE 2.** Transesophageal echocardiographic short-axis (left panels) and long-axis (right panels) views of aortic arch. Raised lesions with irregular surfaces (arrows), compatible with complicated lesions confirmed by histopathologic study, are delineated. A narrow protrusion (small arrow) is attached to surface.

**FIGURE 3.** Transesophageal echocardiographic long-axis view (left panel), short-axis view (middle panel), and its M-mode recording (right panel) of aortic arch. Swinging slender echo (arrow) is shown on raised lesion.
than 5 mm and in five it exceeded 10 mm. Twenty-three lesions had markedly irregular surfaces, including three slender echoes swinging from the surface into the lumen (Figure 3). Thirteen showed trailing acoustic shadows. Although we could not clearly identify the ostia of cervical arteries by TEE, a plaque lying on the ostia of the left subclavian artery was detected in one case (Figure 4). Only one patient had accompanying aneurysmal changes of the aorta, one who had aortic dissection with continuous protrusions of the surface from the top of the arch into the descending aorta, which occupied almost half the lumen.

Baseline characteristics of the subjects are described in Table 1. Among the five risk factors, only hypertension showed a significant correlation with the echographic presence of complicated lesions \( (p<0.05, \chi^2 \text{ test}) \). All five patients with more than three risk factors and eight of the 15 patients with two factors had complicated lesions of the arch. Age also correlated significantly with the presence of echographic complicated lesions \( (p<0.0001, \text{unpaired } t \text{ test}) \).

Plain radiography delineated circumferential or partial calcification of the aortic knob in 28 patients: eight (22%) of 36 without echographically significant lesions and 20 (77%) of 26 patients with significant lesions in the aortic arch (Table 2). There was a significant correlation between the presence of radiographic calcification and echographic findings \( (p<0.0001, \chi^2 \text{ test}) \). Wall calcification in the other parts of the aorta was rarely seen in our subjects.

**FIGURE 4.** Transesophageal echocardiographic short-axis view of aortic arch. Small hyper-echoic protrusion (large arrow) lies on the ostia of the left subclavian artery (small arrow).

**TABLE 1.** Clinical Characteristics of 62 Patients With Embolic Stroke

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>Total</th>
<th>Echographically significant lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (mean±SD)</strong></td>
<td>63.7±13.5</td>
<td>71.1±9.8</td>
</tr>
<tr>
<td><strong>Sex (m:f)</strong></td>
<td>41:21</td>
<td>18:8</td>
</tr>
<tr>
<td><strong>Risk factor</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Hypo-HDL-cholesterolemia</td>
<td>23</td>
<td>12</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>Cigarette smoking*</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td><strong>Extra-aortic source of emboli</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart disease</td>
<td>49</td>
<td>23</td>
</tr>
<tr>
<td>Rheumatic disease</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td>Other organic disease</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td>Atrial fibrillation without organic disease</td>
<td>21</td>
<td>11</td>
</tr>
<tr>
<td>Sustained</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Paroxysmal</td>
<td>16</td>
<td>10</td>
</tr>
<tr>
<td>Plaque in cervical artery</td>
<td>14</td>
<td>9</td>
</tr>
</tbody>
</table>

*More than 50 pack-years (cessation more than 5 years ago exclusive).
is thought to be hemodynamically vulnerable to atherosclerosis because of the presence of curvature and orifices to the main branches. The detection rate of atherosclerotic plaques ranged from 7% to 44% in the previous reports. We cannot directly compare our results with them because of their different criteria for the significant lesion or poor description of its location. Our criteria, based on autopsy findings, had fairly high specificity of echographic detection, indicating that overdiagnosis in TEE was minimal.

The TEE gave us more detailed aortic information than plain radiography, CT, and aortography, probably because we could observe the inner surface closely and continuously by TEE. The echographically significant lesions correlated well with calcification of the aortic knob seen on plain radiograms. This easy examination appears to be useful in rough screening for atherosclerotic involvement of the aorta. CT was inferior to TEE regarding the morphological information of the plaque, although the number of the subjects examined by CT was small in this study. We barely found atherosclerotic findings by aortography.

It is difficult to determine the actual origin of emboli in patients with multiple potential sources, such as lesions in the aorta, heart, and cervical arteries. All of the heart diseases detected in this study are well-known causes of emboli. However, severe arch lesions were often seen in patients with paroxysmal atrial fibrillation without organic heart diseases or cervical plaques, seven of whom had significant lesions of the aortic arch by TEE. Of the remaining 10 patients (16%) who had no potential embolic source in the heart and cervical artery, three had echographically significant lesions (Figure 5). These lesions were found in the aortic arch proximal to the orifice of the innominate artery. All of them had irregular surfaces and trailing acoustic shadows without stringlike swinging echoes. The maximal intimal-medial thicknesses were 12, 6, and 5 mm. These three patients developed sudden neurological deficits and had sharply marginated massive infarctions involving the cortex, with hemorrhagic transformation (in two patients in the areas supplied by the right middle cerebral artery and one in the area by the right posterior cerebral artery).

**Discussion**

Transesophageal echographic observation detected complicated lesions frequently in the aortic arch, which

<table>
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<tr>
<th>Significant lesion determined by transesophageal echocardiography (n)</th>
<th>Positive findings</th>
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<tr>
<td></td>
<td>Plain radiography (n=62)</td>
</tr>
<tr>
<td>Positive (26)</td>
<td>20 (77)</td>
</tr>
<tr>
<td>Negative (36)</td>
<td>8 (22)</td>
</tr>
<tr>
<td>Total (62)</td>
<td>28</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate the positive finding ratio.
cannot be neglected as an embolic source because it may cause the turbulent flow to produce thrombi.

The concept of aortogenic embolic stroke may overstep the boundary between traditional embolic stroke and thrombotic stroke. A considerable number of patients with potential aortogenic embolism were excluded from the present study because they clinically showed atherothrombotic features and did not meet our inclusion criteria. Aortogenic embolic stroke may not always have clinical characteristics similar to those of cardioembolic stroke. For example, some autopsy cases with aortogenic cholesterol embolization were reported to have no cortical infarcts but, instead, multiple small subcortical infarcts. We can assume that some cases of aortogenic embolism may have been misdiagnosed as thrombotic stroke.

There are some limitations in TEE examination. The aortic wall close to the transducer was not visualized clearly, but a higher-frequency transducer (7.5–10 MHz) may resolve this disadvantage. The blind space behind the trachea and bronchus is another major limitation, which partly resulted in the poor detection rate of ascending aortic lesions. The ascending aorta had been considered to have fewer atherosclerotic changes, but a recent pathological study revealed that a considerable number (38%) had severe plaques in this portion of the aorta. More intensive investigation of the ascending aorta is required. We feel that TEE is the most useful technique for visualization of the aorta at present, despite these defects.

Improvement in diagnostic technology will allow us to detect unanticipated embolic sources in the future, much as attention has been attracted to embologenic subcortical infarcts. We can assume that some cases of aortogenic embolism may have been misdiagnosed as thrombotic stroke.

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