Transesophageal Echocardiography in the Detection of Intracardiac Embolic Sources in Patients With Transient Ischemic Attacks

Gheorghe Pop, MD, George R. Sutherland, FESC, Peter J. Koudstaal, MD, Tak W. Sit, MD, Gosse de Jong, MD, and Jos R.T.C. Roelandt, FACC

Using both precordial and transesophageal echocardiography, we studied 72 consecutive patients with a recent unequivocal transient ischemic attack or nondisabling stroke to determine the relative value of the two techniques for detecting potential intracardiac sources of cerebral emboli. Group 1 (n=53) patients had no clinical cardiac abnormality, and group 2 (n=19) patients had abnormal cardiac findings upon clinical examination. In group 1, precordial echocardiography detected an abnormality in only one patient (aortic valve thickening) but transesophageal echocardiography defined morphologic abnormalities in five patients (one with a left atrial appendage mass lesion, one with aortic dissection, one with mitral valve prolapse, one with a mitral leaflet mass lesion, and one with aortic valve thickening). In group 2, both precordial and transesophageal echocardiographic studies were normal in 13 patients, while both were abnormal in the remaining six patients. Five of these six patients had pathologic left atrial and/or left ventricular dilatation, but only transesophageal echocardiography defined a left atrial appendage thrombus in two of the six. The sixth patient had mitral chordal rupture, seen on both precordial and transesophageal echocardiography. In addition, in 32 of the 72 patients transesophageal echocardiography identified widespread thoracic aortic atherosclerotic plaques not visualized by precordial echocardiography. We conclude that transesophageal echocardiography significantly increases the yield in visualizing potential intracardiac sources of emboli compared with precordial echocardiography. However, the precise clinical value of the former in the management of such patients requires further study as the number of abnormal transesophageal echocardiographic findings is not high and a causative relation with transient ischemic attacks cannot be proven. (Stroke 1990;21:560-565)
TABLE 1. Cardiac Abnormalities on Clinical Examination

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical history</td>
<td>Angina pectoris</td>
</tr>
<tr>
<td></td>
<td>Previous myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>Heart rhythm disturbances requiring medical treatment</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Signs of heart insufficiency</td>
</tr>
<tr>
<td></td>
<td>Organic heart murmur</td>
</tr>
<tr>
<td></td>
<td>Ventricular extrasystoles (&gt;10/min)</td>
</tr>
<tr>
<td></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>Electrocardiography</td>
<td>Old myocardial infarction</td>
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<tr>
<td></td>
<td>Ischemic repolarization disorders</td>
</tr>
<tr>
<td></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td>Ventricular extrasystoles (&gt;10/min)</td>
</tr>
<tr>
<td>Chest roentgenography</td>
<td>Cardiac enlargement (cardiothoracic ratio &gt;50%)</td>
</tr>
</tbody>
</table>

Subjects and Methods

Seventy-two consecutive patients with recent TIA or minor stroke were admitted to the hospital for neurologic evaluation and subsequently entered into a prospective study carried out between May 1987 and March 1988. Twenty-four patients were women and the other 48 were men; their ages varied between 24 and 73 (mean 60.2) years. Two neurologists independently established a definite clinical diagnosis of TIA or nondisabling stroke defined as the sudden onset of a focal neurologic deficit in a specific vascular distribution. Patients with possible migrainous events, seizures, and vague or nonfocal neurologic symptoms were excluded.

All patients underwent computed tomography and laboratory investigations to exclude cerebral ischemia from causes other than artery-to-artery or cardiac embolism as well as disorders mimicking cerebral ischemia. Laboratory investigations included the erythrocyte sedimentation rate (if >30 mm/hr, then also antinuclear factor), hemoglobin, hematocrit, platelet count, fibrinogen and glucose concentrations, hepatic and renal function, and syphilis serology.

Of the 72 patients, 20 had a TIA and 52 a minor stroke. Sixty-three ischemic events were located in the territory of the carotid artery and the other nine in that of the vertebrobasilar artery. Carotid angiography was performed in 27 patients, who were all <70 years of age and in whom cardiac investigations revealed no abnormality that could have caused the cerebral ischemic event.

The interval from the onset of neurologic symptoms until the cardiologic evaluation varied between 1 day and 3.5 weeks (mean 8 days). The cardiologic evaluation consisted of a clinical history and a physical examination, both carried out by the same cardiologist, a 12-lead electrocardiogram, and a chest roentgenogram.

After this cardiologic evaluation, we divided the patients into two groups. Group 1 patients had no cardiac abnormalities detected, and group 2 patients had cardiac abnormalities. Table 1 shows the criteria used for cardiac abnormalities. Group 1 (n=53) contained 15 patients with TIA and 37 patients with stroke; in group 2 (n=19), there were six patients with TIA and 13 patients with stroke.

All 72 patients underwent precordial and transesophageal echocardiography on the same day, each by a different investigator. After obtaining informed consent, we recorded the precordial echocardiogram

TABLE 2. Cardiac Abnormalities Detected by Precordial and Transesophageal Echocardiography in 72 Patients With Transient Ischemic Attack or Nondisabling Stroke

<table>
<thead>
<tr>
<th>Group</th>
<th>Precordial Abnormality</th>
<th>n</th>
<th>Transesophageal Abnormality</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>1: No cardiac abnormalities on clinical examination (N=53)</td>
<td>Aortic valve sclerosis without stenosis</td>
<td>1</td>
<td>Aortic valve sclerosis without stenosis</td>
<td>1</td>
</tr>
<tr>
<td>2: One or more clinical cardiac abnormalities (N=19)</td>
<td>Left atrial and/or left ventricular dilatation</td>
<td>5</td>
<td>Left atrial and/or left ventricular dilatation</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Mitral valve chordal rupture</td>
<td>1</td>
<td>Mitral valve chordal rupture</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Left atrial appendage mass lesion</td>
<td>1</td>
<td>Left atrial appendage mass lesion</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Aortic dissection (localized in aortic arch)</td>
<td>1</td>
<td>Spontaneous echo contrast effect in left atrium</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Mitral valve prolapse</td>
<td>1</td>
<td>Ascending, arch and descending aortic plaques</td>
<td>10</td>
</tr>
</tbody>
</table>
with a Toshiba SSH 65 A imaging system (Tokyo, Japan) using 2.5- and 3.75-mHz probes and performed transesophageal echocardiography using a 5.6-mHz phased-array transducer developed at the Thoraxcenter, Rotterdam (type HP-TEE) and interfaced with a Hewlett-Packard 7200 imaging system (Palo Alto, California). Introduction of the esophageal probe was performed after local anesthesia with a 5% lidocaine topical spray. No prior sedation was given.

Transesophageal echocardiography was performed systematically and lasted generally for approximately 15 minutes. After introduction, we manipulated the probe until it was located in the stomach, and then we recorded a series of cross-sectional short-axis-of-the-left-ventricle views. The probe was pulled back within the esophagus until a proper four-chamber view was obtained. In this section we focused attention on the mitral valve and its chordae and visualized the aortic valve and the aortic root, orienting the probe superiorly. Further withdrawal of the probe superiorly allowed us to record a series of cross-sections through the left atrium just above the mitral valve. This manipulation allowed complete evaluation of the interatrial septum and left atrial appendage, and with further manipulation good visualization of the main pulmonary artery was obtained.

We then rotated the probe through 180° toward the posteriorly located descending aorta. By pulling the probe slightly upwards, we were able to make sequential cross-sections of the descending aorta and ultimately scanned the aortic arch and ascending aorta.

Results

Transesophageal echocardiography was well tolerated in all 72 patients, with no resulting complications. One patient had bulbar palsy, but introduction of the probe in this patient produced no problems. All results are summarized in Table 2.

All 53 patients in group 1 had a normal precordial two-dimensional echocardiogram except for one patient who had abnormal thickening of the aortic valve. There was no increase in peak blood flow velocity over the aortic valve on Doppler examination in this patient. Transesophageal echocardiography revealed abnormalities that could predispose to TIA in five patients. These abnormalities consisted of a left atrial appendage mass lesion (Figure 1), a very localized dissection within the aortic arch, a mitral valve prolapse (in a patient <45 years old), a mass lesion attached on the left atrial side of the posterior mitral leaflet, and thickening of the aortic valve in one patient detected also by precordial echocardiography. The patient with the left atrial appendage...
mass lesion had no history of atrial fibrillation, and the left atrium was not enlarged.

Of the 19 patients in group 2, both precordial and transesophageal echocardiograms were normal in 13. Of the remaining six patients, five had left atrial and left ventricular dilatation on both precordial and transesophageal echocardiograms and were in atrial fibrillation. Transesophageal echocardiography detected a left atrial appendage mass lesion suggesting thrombus in two of these five patients, and in four of the five a spontaneous echo contrast effect indicating slow blood flow was seen (Figure 2). The sixth patient, known to have an old myocardial infarction, had a mitral chorda tendinea rupture of the anterior leaflet seen on both precordial and transesophageal echocardiography.

In group 1 as well as in group 2, precordial and transesophageal echocardiographic findings did not differ significantly between patients with TIA and those with minor stroke.

In 32 of the 72 patients, transesophageal echocardiography identified diffuse aortic wall irregularities typical of atherosclerotic plaques (Figure 3), confirming the frequent presence of associated vascular disease in TIA patients. In group 2, 10 of the 19 patients showed such aortic wall irregularities.

Discussion

Using transesophageal imaging, Daniel et al recently showed that the heart is a frequent potential source of systemic emboli. However, in patients with TIA or minor stroke, an intracardiac source of emboli is only one of the possible causes. Atherosclerotic lesions in the intracranial or extracranial territories of the carotid or vertebrobasilar arteries can lead to artery-to-artery cerebral embolism and probably represent the major cause of cerebral ischemia.

It has been demonstrated that in TIA patients aged >45 years precordial two-dimensional echocardiography does not offer additional information about possible intracardiac sources of emboli beyond that provided by the clinical cardiac examination. Half of all left atrial thrombi are limited to the appendage. Precordial echocardiography rarely visualizes this structure, and the technique is especially insensitive in detecting appendage thrombi. This may explain its low yield of intracardiac thrombi in TIA patients. Transesophageal echocardiography offers advantages for detecting mass lesions in the left atrial appendage and ascending aorta because the improved signal-to-noise ratio and increased frequency of the transducers lead to high-resolution, high-quality images.
Among group 2 patients, transesophageal echocardiography detected a left atrial appendage mass lesion in two and a left atrial spontaneous echo contrast effect in four. The latter may be an indicator of increased thromboembolic risk. These findings did not change our standard therapeutic management policy, which consisted of anticoagulation therapy in patients with significant dilatation of the left ventricle or left atrium. In TIA patients in group 1 (n=53), additional information potentially relevant for their management was found in five patients (10%) by transesophageal echocardiography, while precordial echocardiography was positive in only one patient.

The finding of mitral valve prolapse is of interest because this entity may have a role in cerebral ischemia, at least in patients <45 years old, as in our study patient. Transesophageal echocardiography is especially valuable in distinguishing a benign mitral valve prolapse from a pathologic, complication-prone one. In the latter, subsequent sequential ultrasound studies of the mitral valve seem useful in predicting valve complications.

Our results suggest that when echocardiography is considered in TIA patients without clinical cardiac abnormalities, they should undergo transesophageal rather than precordial echocardiography. These results agree with the findings of a recently published comparative study of precordial versus transesophageal echocardiography in young patients with TIA. In TIA patients with some clinical cardiac abnormalities, precordial echocardiography often better evaluates the cardiac abnormality because of improved visualization. Transesophageal echocardiography may provide additional, purely morphologic information, but in every case where this occurred, the new information had no therapeutic consequences and gave no additional information compared with clinical examination and precordial echocardiography.

Our study had some definite limitations. Since it was not performed before the TIA, we may have missed the embolic source. Also, because of the delay between onset of the TIA and transesophageal echocardiography, any intracardiac thrombus could have been dissolved by anticoagulation therapy. Another reason for the low yield of intracardiac thrombi may be that transesophageal echocardiography does not adequately visualize the left ventricle apex, where such thrombi can exist. However, small apical thrombi may be just as easily missed by precordial
echocardiography. Recent publications suggest a high prevalence (in some studies up to 20%) of a patent foramen ovale in patients with stroke. We began our study 1 year before these publications, and we did not carry out echo contrast and Doppler investigations of the atrial septum and left atrial appendage. We think that it seems mandatory from now on to include such studies during transesophageal echocardiography to look for a patent foramen ovale and the possibility of paradoxical embolism.

In our study, transesophageal echocardiography showed 32 (44%) of the 72 TIA patients to have widespread atherosclerotic lesions in the aorta, which confirms the frequent presence of associated atherosclerotic vascular disease in TIA patients reported by other authors. Such atherosclerotic lesions in the ascending aorta and beginning of the aortic arch can cause embolism, which gives rise to a TIA.

We conclude that in TIA patients without apparent clinical cardiac abnormalities, transesophageal echocardiography significantly increases the yield of potential intracardiac sources of emboli compared with precordial echocardiography. However, the overall yield of potential intracardiac sources of emboli detected by transesophageal echocardiography in these patients remained relatively small (10%), and it is not clear whether the findings were merely an associated abnormality or a direct cause of the TIA. Transesophageal echocardiography detected a higher percentage yield of potential intracardiac sources of emboli in TIA patients with a clinically apparent cardiac abnormality than in those with clinically normal cardiac findings. However, this extra clinical information was of little relevance in clinical decision-making in this series.

Acknowledgments

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