Transesophageal Echocardiography for Detection of Cardiac Diseases in Patients with Retinal Artery Occlusion

Yuichiro Inatomi, Hirotake Hino*, Yoichiro Hashimoto**, Naohiko Furuyoshi***, Ikuo Misumi**** and Makoto Uchino*****

Abstract

Objective We analyzed the usefulness of transesophageal echocardiography (TEE) for detection of cardiac diseases in patients with retinal artery occlusion (RAO).

Patients and Methods We retrospectively reviewed the charts of 22 consecutive patients with acute RAO. The patients had been evaluated by conventional studies, including transthoracic echocardiography (TTE) and TEE.

Results TEE findings were abnormal in 13 (59%) of the 22 patients. The findings revealed a decrease of flow velocity in the left atrial appendage (n=7), atrial septal aneurysm (n=4), patent foramen ovale (n=2), spontaneous echo contrast (n=1), ascending aortic plaque (n=1) and left atrial thrombus (n=1). Evaluations, including TEE, disclosed cardiac abnormalities in 16 (73%) of these 22 patients. However, excluding the analysis by TEE, cardiac abnormalities were revealed in only 6 (27%) patients.

Conclusion In patients with RAO, TEE may be a useful examination for detecting potential cardiac diseases.

Key words: patent foramen ovale, atrial septal aneurysm, embolism

Introduction

Atherosclerotic lesions in the carotid bifurcation are often seen in patients with retinal artery occlusion (RAO) (1–5). Therefore, it has been suggested that RAO is mainly caused by emboli originating from carotid lesions. On the other hand, cardiac diseases which can be embolic sources are also seen in some patients with RAO (2, 3, 5–9). Transthoracic echocardiography (TTE) is commonly used to detect cardiac sources of embolism. In addition, transesophageal echocardiography (TEE) is a sensitive method for detecting cardiac sources of embolism, including patent foramen ovale (PFO) and atrial septal aneurysm (ASA), in patients with brain infarction or transient ischemic attack, and has been reported to be superior to TTE (10–15). TEE has detected cardiac disease in some patients with RAO who had no abnormal findings by TTE (16, 17). We therefore analyzed the usefulness of TEE in detecting cardiac diseases in patients with RAO.

Subjects and Methods

We retrospectively reviewed the charts of 59 consecutive patients with acute RAO who visited Kumamoto City Hospital between April 1, 1993 and March 31, 1997. A diagnosis of central retinal arterial occlusion (CRAO) was made if the patient had experienced a sudden, painless loss of vision in one eye associated with the typical ophthalmoscopic findings of CRAO including a pale retina, narrowing of the retinal arteries, and a macular cherry red spot. A patient was diagnosed with branch retinal arterial occlusion (BRAO) if the territory of a branch retinal artery appeared pale on ophthalmoscopic examination. The inclusion criteria were well-described full studies including electrocardiogram (ECG), 24-hour Holter monitoring, carotid ultrasonography, TTE and TEE within a month after the onset. Although we explained the importance of TEE, 34 patients refused the evaluation, and the descriptions of TTE findings were inadequate in 3 patients. Thus, the study group was comprised of 22 patients (13 men, 9 women) with a mean age of 63.4±13.8 years.

The carotid arteries of all study patients were examined in the longitudinal and transverse planes from the anterior, lateral, and posterior approaches using B-mode imaging with color flow imaging using the Ultramark 9 carotid ultrasonography instrument (Advanced Technology Laboratories, Washington, DC, USA). The common carotid artery, carotid bifurcation, internal carotid artery, and external carotid artery were studied in all patients in an attempt to diagnose the underlying cardiac or arterial disease which may have caused the RAO. The percentage of stenosis of the external carotid arteries was mea-
TTE and TEE were performed by SSD-870 (Aloka, Tokyo). In both studies, we used the B-mode, M-mode, color Doppler or the contrast echo method with or without the Valsalva maneuver. A patient was diagnosed with PFO if a right-to-left atrial shunt was detected by color Doppler or by the contrast echo method. A patient was diagnosed to have ASA if the atrial septum appeared abnormally redundant and mobile and exhibited invasion into the left atrium, right atrium, or both at a distance of at least 7 mm. Spontaneous echo contrast was defined as an amorphous echo composed of numerous microechos which curled up slowly in the left atrial cavity like billows of smoke or clouds. The severity of aortic regurgitation (AR) was estimated with color Doppler by measuring the distance in the left ventricle that turbulent flow signals were seen. If a small area of high velocity signals was localized near the aortic valve, the AR was classified as mild, and if these signals extended to the level of the anterior mitral leaflet, it was classified as moderate. Turbulent signals extending beyond the mitral valve to the level of the papillary muscles or toward the left ventricular apex indicated severe AR. The severity of mitral regurgitation (MR) was also estimated with color Doppler by noting the portion of the left atrium occupied by regurgitant signals. If the regurgitant jet occupied less than 20% of the left atrial cavity, the MR corresponded to the mild MR. When the jet occupied between 20 and 40% or more than 40% of the left atrium, the MR corresponded respectively to moderate or severe MR. Normal ranges of the other clinical parameters at our hospital are: left atrial dimension (LAD) ≤ 50 mm, left ventricular internal dimension at end-diastole (LVIDd) ≤ 55 mm, left ventricular internal dimension at end-systole (LVIDs) ≤ 40 mm, left ventricular thickness at end-diastole (LVTd) ≤ 11 mm, and ejection fraction of the left ventricle (EF) ≥ 50%. Normal range of the peak flow velocity in the left atrial appendage (LAA) was defined as ≥ 35 cm/s according to the data of the control group in the literature (19).

**Results**

Data on eligible patients are summarized on Table 1. Fourteen of the 22 patients with acute RAO had CRAO and 8 patients had BRAO. According to clinical history, 4 (18%) of the 22 patients had cardiac disease; 2 had atrial fibrillation, 1 myocardial infarction, and 1 had both myocardial infarction and atrial fibrillation. Fourteen (64%) of these 22 patients had one or more diseases which are risk factors for arteriosclerosis. Three (14%) of these 22 patients had been given anticoagulant

<table>
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<th>Age/sex</th>
<th>Type of RAO</th>
<th>Risk factor</th>
<th>Cardiac history</th>
<th>Anticoagulant</th>
<th>% carotid stenosis</th>
<th>TTE findings</th>
<th>TEE findings</th>
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<tbody>
<tr>
<td></td>
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<td>HT DM HL TO</td>
<td>LAD (mm)</td>
<td>LVIDd/LVIDs (mm)</td>
<td>LVTd/LVTs (mm)</td>
<td>EF (%)</td>
<td>Others</td>
<td>Flow in LAA (in/out, cm/s)</td>
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<td>BRAO</td>
<td>+ - - - -</td>
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<td>100</td>
<td>39</td>
<td>51/29</td>
<td>13/18</td>
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<tr>
<td>68/F</td>
<td>CRAO</td>
<td>+ - - - -</td>
<td>-</td>
<td>75-90</td>
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<td>46/37</td>
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<td>-</td>
<td>25-50</td>
<td>NR</td>
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<tr>
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<td>35</td>
<td>51/22</td>
<td>11/NE</td>
<td>68</td>
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<tr>
<td>48/M</td>
<td>BRAO</td>
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<td>23</td>
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<td>s-af warfarin</td>
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<td>9/14</td>
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<td>p-af warfarin</td>
<td>0</td>
<td>42</td>
<td>70/40</td>
<td>14/17</td>
<td>73</td>
</tr>
</tbody>
</table>

| 76/F    | CRAO        | - - - - -   | s-af, MI        | 0              | 66                | 56/42        | 9/10         | 50          | 10/9        |
| 69/M    | CRAO        | - + + - +   | MI warfarin     | 0              | 40                | 48/26        | 10/12        | 50          | 81/46       |
| 64/M    | BRAO        | + + + - -   | -               | 0              | 32                | 46/24        | 11/14        | 86          | 55/55       |
| 55/F    | CRAO        | - + + - -   | -               | 0              | 33                | 48/30        | 9/14         | 63          | 106/62      |
| 64/M    | BRAO        | - + + - -   | -               | 0              | NR                | 51/35        | 9/15         | 67          | NE/ASA      |
| 48/M    | BRAO        | + - + - -   | -               | 0              | 36                | 47/32        | 10/14        | 61          | 51/82       |
| 54/M    | BRAO        | + - - - -   | -               | 0              | 33                | 46/28        | 9/15         | 70          | 60/NE       |
| 21/F    | CRAO        | - - - - +   | -               | 0              | 31                | 46/26        | 7/11         | 74          | 53/45       |
| 51/M    | CRAO        | - - - - +   | -               | 0              | 35                | 43/19        | 9/15         | 66          | 40/19       |

HT: hypertension, DM: diabetes mellitus, HL: hyperlipidemia, TO: tobacco, NE: not examined, NR: not remarkable, s-(p-) af: sustained (paroxysmal) atrial fibrillation, MI: myocardial infarction, see text for other abbreviations.
therapy prior to RAO.

Carotid ultrasonography revealed stenosis or occlusion of the ipsilateral internal carotid artery in 11 (50%) of the 22 patients. Total occlusion was found in one patient.

TTE disclosed 12 abnormalities in 6 (27%) of the 22 patients: aortic valvular regurgitation (n=3), dilation of the left atrium (n=3), thickened left ventricular wall (n=2), dilation of the left ventricular cavity (n=2), decrease of the ejection fraction (n=2), and mitral regurgitation (n=1).

Findings were abnormal by TEE in 13 (59%) of the 22 patients. There was a decrease of flow velocity in the left atrial appendage in 7 patients, ASA in 4 patients, PFO in 2 patients, and spontaneous echo contrast, plaque in the ascending aorta and left atrial thrombus in 1 patient, respectively. Clinical history and TTE disclosed cardiac abnormalities or diseases in only 6 (27%) of the 22 patients (Table 2). However, TEE disclosed cardiac abnormalities or diseases in 16 (73%) of the 22 patients (Table 3).

**Discussion**

**Usefulness of TEE**

TTE has been used to detect potential cardiac embolic sources in patients with RAO (2, 3, 5–9). TEE, which was recently developed, is significantly better than TTE in visualizing potential intracardiac sources of emboli in patients with cardioembolic stroke or transient ischemic attack (10–15). TEE must be performed for the detection of PFO (14, 20–23), ASA (12, 15, 21, 24), smoke-like echo or spontaneous echo-contrast (25–27), and atherosclerotic plaque in aorta (10). Among patients with acute RAO who are at low cardioembolic risk, TTE can find few diseases (8). In some reported cases of RAO, PFO was detected by TEE after TTE failed to show any abnormalities (16, 17).

In the present study, TEE detected cardiac abnormalities which were not detected by TTE, including a decrease of flow velocity in the left atrial appendage, ASA, PFO, left atrial thrombus, spontaneous echo contrast, and aortic plaque.

ASA was detected with TEE in 0.21% of normal people (28) and with TEE in 8.9% of normal people (12). The prevalence of ASA among the 22 patients in the present study was greater than that found in those reported studies. One possible explanation for this difference is that our criteria of ASA was a redundant and mobile atrial septum with a more than 7 mm invasion, according to a previous study (28). In more recent reports (12, 15, 21, 24), ASA was defined as more than 10–15 mm of redundant and mobile atrial septum. In the present study no ASA that TEE disclosed was found by TTE. Possibly, our criteria of ASA may allow inclusion of minimal changes that can only be detected by TEE. Also, although our criteria allowed for an increase in the prevalence of ASA, the prevalence of ASA in patients with RAO may be greater than in the normal population.

Decrease of flow velocity in LAA was seen in 7 patients in our study. This finding may be associated with left atrial thrombus or atrial fibrillation (19, 27, 29, 30). Four of the 7 patients, however, did not have any other cardiac abnormalities. It is suggested that a decrease of flow velocity in LAA is less significant for embolic sources by itself.

In previous studies, PFO was detected by TTE in 10% of normal people (20) and with TEE in 18% of normal subjects (14). We found fewer patients with PFO among the present patients. In our study, 50% of patients had carotid plaque. The prevalence was similar to the results of the previous studies in which carotid plaque was seen in 27 to 85% of patients with RAO (1–5). Aortic plaque was also found in only one patient. A larger study of TEE in patients with RAO is necessary.

Although our study was small, examinations for the detection of cardiac diseases which included TEE found twice the number of abnormalities in comparison with those that did not include TEE. TEE may be a useful examination for detecting potential cardiac diseases in patients with RAO.

**Prevalence of cardiac disease as an embolic source in patients with RAO**

Cardiac diseases are seen in 2–45% of patients with RAO (2, 3, 5–9). However, cardiac diseases in these studies have included non-rheumatic valvular disease and cardiac hypertro-
phy, which do not cause embolism. In addition, TEE was not used for the detection of the embolic source in all of the RAO patients in any of the previous studies as well as in the present study. In our study, many abnormal cardiac findings, including dilation of the left ventricle, valvular diseases, ASA and PFO, have not been defined as having an embolic source. It is important to design controlled studies in which these findings would be defined as having an embolic source.

**Limitations of the present study**

TEE was performed on only 22 of 59 patients (37%) who were admitted to our hospital with acute RAO. Most of the excluded patients were those who refused an evaluation by TEE. All 59 patients were equally informed of the importance of TEE; therefore, the selection of eligible patients was not biased through a lack of information provided to patients. However, certain cardiac abnormalities that would have been detected by TEE may have been missed by TTE alone. TEE is a rather invasive study for detecting cardiac embolic source(s) and is more invasive than ECG and TTE. Actually, 34 of 59 patients refused TEE. However, the risks of TEE are acceptably low if performed by experienced technicians under proper and safe conditions (31).

In conclusion, TEE may be a useful examination for detecting potential cardiac sources of cerebral emboli in patients with RAO. We propose a larger study of TTE versus TEE and a controlled study of the prevalence of cardiac abnormal findings to define their significance as embolic sources.

**References**