Transesophageal Echocardiographic Detection of Aortic Arch Disease in Patients With Cerebral Infarction

Pierre Amarenco, MD; Ariel Cohen, MD; Marielle Baudrimont, MD; and Marie-Germaine Bousser, MD

Background and Purpose: Ulcerated plaques in the aortic arch are frequent autopsy findings in patients with cerebral infarctions, particularly those of unknown cause. It has been suggested that they could be a source of cerebral emboli. Using transesophageal echocardiography, we prospectively studied 12 consecutive patients with cerebral infarction of undetermined cause after noninvasive workup to evaluate the frequency of aortic plaques or mural thrombi that could embolize in cerebral arteries.

Summary of Review: Six patients (50%) had an intraluminal echogenic mass of the aortic arch, mainly located at the junction of the ascending aorta and arch. This material was pedunculated (in one patient) or broad based (in five patients) with a markedly irregular surface and intraluminal extension from 3 to 15 mm. In addition, we found cholesterol emboli in two of the four patients who underwent quadriceps biopsy.

Conclusions: These results show that transesophageal echocardiography has capabilities in detecting such lesions and point to the aortic arch as a possible source of cerebral emboli in patients with cerebral infarction of unknown cause.

KEY WORDS • cardiovascular diseases • cerebral infarction • echocardiography

Cerebral angiography, echo Doppler of extracranial carotid arteries, transcranial Doppler ultrasonography,1,2 and transesophageal echocardiography (TEE)3-4 have improved our ability to detect arterial or cardiac causes of cerebral infarction. However, in stroke data banks, no cause can be found in 26–40% of all cerebral infarcts,5-6 pointing to an underrecognition of sources of cerebral emboli other than those detected by techniques currently used. Several single reports have recently emphasized the ability of TEE to detect atherosclerotic debris in the aorta, particularly in the arch.3-7-9 In an autopsy data bank of 500 neurological patients we previously found a prevalence of ulcerated plaques in the aortic arch of 28% in patients with stroke and of 61% in patients with stroke of unknown cause, and such lesions were present only in patients older than 60 years.10 We therefore decided to prospectively examine the aortic arch in vivo using TEE in patients with cerebral infarcts and in control subjects, a multicenter study currently in progress (French Study of Aortic Plaques in Stroke [FAPS]). In the present pilot study, we prospectively enrolled 12 consecutive patients with cerebral infarct of undetermined cause during a 6-month period to evaluate the ability of TEE in detecting aortic arch atherosclerotic disease in this subgroup of patients. Four of these patients also had a muscle biopsy for detection of cholesterol emboli.

Subjects and Methods

Patients older than 60 years were prospectively recruited if they had a cerebral infarct proven by computed tomography and no known cause of stroke using noninvasive studies, i.e., normal transcranial Doppler, no ipsilateral >30% stenosis of the origin of the internal carotid artery, and no cardiac source of emboli after examination with 12-lead electrocardiography and transthoracic and transesophageal echocardiography. Twelve patients remained for study (six men and six women) aged 64–83 years (mean age, 72.5±6.2 years). Transthoracic echocardiography was performed to evaluate the aortic arch using the suprasternal view, then TEE was performed. Patients with a history of dysphagia, preexisting esophageal pathology, or severe bronchopneumonia were excluded. Transesophageal echocardiography was performed with video recording, enabling retrospective review on two separate occasions by the same observer. Transesophageal echocardiography was performed within 1 week of the stroke according to standard techniques. With the patient in the left lateral decubitus position, a peripheral intravenous catheter was inserted while the patient was in a fasting state (>4 hours). Local anesthesia of the oropharynx (aerosol and viscous lidocaine) was administered. In anxious patients, midazolam hydrochloride injection (1-2 mg) was given. After the cardiac phase of the examination, the probe was rotated 120–180° counterclockwise to visualize the cross section of the entire...
thoracic descending aorta (40–45 cm from incisors). The arch was then imaged by slowly withdrawing the probe up to 18–20 cm from the incisors. We used the Vingmed and Hewlett-Packard SONOS-100 ultrasound imaging systems, with a 5-MHz single-plane transesophageal transducer. In addition to the cardiac examination, the aorta was specifically examined for 5–10 minutes. In four patients with large aortic lesions, biopsy of the quadriceps muscle was performed after informed consent was obtained.

**Results**

Transesophageal echocardiography showed no atheroma or only a moderate degree of atheroma in the aortic arch in six of 12 patients. The other six (50%) had protruding echogenic lesions in the aortic arch ≥2 mm. Baseline characteristics of the patients are indicated in Table 1. In five of these patients, the mechanism of infarction was likely embolic on clinical grounds (sudden onset, multiple or single small cortical infarcts, cerebellar or posterior cerebral artery territory infarct). The sixth patient (patient 2) had pontine infarction of sudden onset with no evidence of intracranial vessel disease at transcranial Doppler, which could be due to either branch disease or small emboli. These patients had normal transcranial Doppler assessing intracranial carotid arteries, middle, anterior, and posterior cerebral arteries, vertebral and basilar arteries, normal neck ultrasound examination, and no cardiac source of emboli. All six had vascular risk factors for atheroma: hypertension in four, cigarette smoking in three, and peripheral vascular disease in two. In addition, high serum fibrinogen, elevated erythrocyte sedimentation rate, or both were noted at admission in four patients. One of these patients had stroke postoperatively. In three patients TEE showed a large protruding echogenic lesion (6, 9, and 15 mm in diameter), either pedunculated with narrow base (in one patient) (Figure 1A) or immobile on a broad base (in two patients) (Figures 1B and 1D) with marked irregularities of the intimal surface. One of them was reevaluated 6 months later, and the protruding echo was no longer seen, which was interpreted as the disappearance of a thrombus superimposed on an ulcerated plaque of the arch. This patient also had cholesterol embolii in muscle biopsy (Figure 2). The other two patients had no cholesterol embolii on muscle biopsy. The three remaining patients had smaller plaques (3, 4.5, and 5 mm in diameter). One of these had intraluminal echoes characteristic of a mural thrombus (Figure 1C) and cholesterol emboli on quadriceps biopsy. Four of the six protruding echogenic lesions were located at the junction of the ascending aorta and arch layered on the dorsal wall of the aorta, with one of these extending over the arch and down the descending aorta. In the two remaining patients, the lesion was found in the arch of the aorta. The following case is illustrative.

A 64-year-old right-handed hypertensive man (patient 1) had sudden onset of left-sided paresthesia involving the face and arm lasting 10 minutes and occurring five times. On the third day he had left-sided numbness, which lasted 10 days. A month later, while walking he had blurring of vision, right-sided numbness involving the face, arm, and leg, amnesia, and confusion. On admission, blood pressure was 150/80 mm Hg with pulse 84 and regular. Neurological examination showed disorientation, abulia, anterograde amnesia, dysarthria, right-sided spatial and motor neglect, and a severe right-sided arm and leg sensory deficit with loss of deep touch, temperature and pain sensations, synkinesia, and ataxia. Computed tomography and magnetic resonance imaging showed left medial and lateral thalamic infarcts, a left internal temporal infarct in the deep territory of the posterior cerebral artery, and a small left anterior cerebral artery territory infarct. Single-photon emission computed tomography showed a marked left occipital and thalamic hypoperfusion. Ultrasound examination of the extracranial carotid and

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**Table 1. Baseline Characteristics of Patients With Cerebral Infarction With No Known Cause and Intraluminal Aortic Arch Disease at Transesophageal Echocardiography**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)/sex</th>
<th>Risk factors</th>
<th>Topography of cerebral infarct</th>
<th>Ultrasound study</th>
<th>Site and size of aortic echos</th>
<th>Muscle biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64/M</td>
<td>Hypertension</td>
<td>Thalamic, ACA</td>
<td>Nonstenotic plaque of ICAO; TCD normal</td>
<td>Ascending aorta Diameter, 15 mm</td>
<td>+ Cholesterol emboli</td>
</tr>
<tr>
<td>2</td>
<td>82/F</td>
<td>↑ Fibrinogen, ESR 70 mm</td>
<td>Pontine</td>
<td>40% left ICAO stenosis; TCD normal</td>
<td>Ascending aorta Diameter, 6 mm</td>
<td>+ Normal</td>
</tr>
<tr>
<td>3</td>
<td>65/M</td>
<td>Hypertension, postoperative stage, ESR 47 mm</td>
<td>Multiple small infarcts (left MCA, right PCA, cerebellum)</td>
<td>No atheroma; TCD normal</td>
<td>Arch of aorta Diameter, 4.5 mm; length, 25 mm</td>
<td>–</td>
</tr>
<tr>
<td>4</td>
<td>71/M</td>
<td>Hypertension, smoking, ↑ fibrinogen, ESR 40 mm</td>
<td>Left cerebellar</td>
<td>Nonstenotic plaque of ICAO</td>
<td>Arch, ascending and descending aorta Diameter, 9 mm</td>
<td>+ Normal</td>
</tr>
<tr>
<td>5</td>
<td>83/F</td>
<td>PVD, ↑ fibrinogen</td>
<td>Left MCA</td>
<td>30% left ICAO stenosis</td>
<td>Ascending aorta Diameter, 5 mm</td>
<td>–</td>
</tr>
<tr>
<td>6</td>
<td>67/M</td>
<td>Hypertension, PVD</td>
<td>Multiple small infarcts (deep MCA, cerebellum)</td>
<td>&lt;30% left ICAO stenosis</td>
<td>Arch of aorta Diameter, 3 mm</td>
<td>+ Cholesterol emboli</td>
</tr>
</tbody>
</table>

M, male; ACA, anterior cerebral artery; ICAO, origin of internal carotid artery; TCD, transcranial Doppler ultrasonography; +, muscle biopsy performed; F, female; ESR, erythrocyte sedimentation rate; MCA, middle cerebral artery; PCA, posterior cerebral artery; –, muscle biopsy not performed; PVD, peripheral vascular disease.
FIGURE 1. Transesophageal echography of the aortic arch showing intraluminal protruding echoes, pedunculated, interpreted as a thrombus (panel A, patient 1); broad-based protruding echoes, immobile, in the arch of the aorta (panel B, patient 2); a mural thrombus in the arch (panel C, patient 8); and large intraluminal protruding echoes in the aortic arch (panel D, patient 4).

vertebral arteries showed nonstenotic plaques of the left internal carotid artery and a 60% stenosis of the origin of the right internal carotid artery contralateral to the anterior cerebral artery infarct. Transcranial Doppler was normal, in particular the basilar, intracranial vertebral, and posterior cerebral arteries. Electrocardiogram showed sinus rhythm. Transthoracic echocardiography showed left ventricular hypertrophy and was otherwise normal. Transesophageal echocardiography showed an intraluminal echogenic lesion that was pedunculated and lay on the aortic wall at the junction of the ascending aorta and arch (Figure 1A). The luminal surface of the mass was markedly irregular with focally increased echogenicity. Muscle biopsy showed cholesterol emboli in small arteries with organized thrombi and small periarterial inflammatory infiltrates (Figure 2). The patient improved but had persistent hemisensory loss, thalamic pain, and amnesia 27 days after the stroke. He was treated with aspirin. Six months later the patient had improved markedly, and TEE showed that the aortic lesion had disappeared.

Discussion

This pilot study demonstrates that TEE has capabilities in detecting such lesions and suggests a high frequency of protruding echogenic structures in the aortic arch of patients with ischemic stroke of undetermined cause after a noninvasive workup. The aorta was long recognized as the first and most severe site of atheromatous changes before coronary, internal carotid, and cerebral arteries. In a necropsy study, Fisher et al noted that the aorta was two to four times more involved by severe atherosclerosis than extracranial arteries. Atheromatous embolization from the aortic arch has been reported but considered to be most unusual. These studies were published mainly in the late 1950s and early 1960s. In that era, the stroke community was concerned primarily with the new possibility of angiographic detection and surgical treatment of internal carotid artery stenosis, and there was no technique other than angiography to visualize the aortic arch. However, cardiac surgeons have long been con-
cerned with preoperative and postoperative cerebral emboli and are accustomed to look for atheroma of the ascending aorta during the surgical procedure, either by manual or intraoperative echographic evaluation. The link between the presence of atheroma of the ascending aorta and a perioperative cerebral event was thus statistically demonstrated in patients who underwent coronary artery bypass grafting. In these patients with coronary disease, the prevalence of atherosclerotic plaques of the ascending aorta >8 mm was 38% in a pathological study of 97 patients. Unlike angiography, which only shows the arterial lumen contour delineation, TEE shows most of the thoracic aorta, including the aortic arch, and provides high-quality imaging of the intimal surface and wall structure, allowing plaque and wall characterization. Several TEE reports and our recent necropsy study have emphasized this possible donor site of cerebral emboli. In this study, we found ulcerated plaques in the aortic arch in 26% of patients with cerebrovascular disease and in 5% of patients with other neurological disease; we showed that their presence did not correlate with carotid artery disease and that they were much more frequent in patients with infarcts otherwise unexplained. In the present series, the frequency of protruding echogenic mass (six of 12 patients [50%]) that we found in the arch with TEE was not far from the 61% of ulcerated plaques in the aortic arch that we previously found in patients with stroke of unknown cause at necropsy.

In the present series, none of the patients had angiography to exclude intracranial arterial occlusive disease. Although an intracranial branch disease cannot be excluded on the basis of a normal transcranial Doppler examination, in most of these patients infarction was likely embolic on clinical grounds. The presence of cholesterol emboli in the muscle biopsy of two patients supports the possibility of a common source of emboli for both recipient sites (brain and muscle). The material seen with TEE in our patients could have been a protruding atherosclerotic plaque, a thrombus superimposed on an ulcerated plaque, or both. We found the same echogenic material as did other authors, i.e., pedunculated or broad-based lesions in the aortic lumen, with marked irregularities of the intimal surface. In necropsy studies of the arch, although atherosclerosis is very severe, there is usually no stenosis but mainly ulcerated atheroma. Interestingly, in our first patient we found a very large protruding echogenic mass in the ascending aorta that disappeared 6 months later. Thus, in this patient we can speculate that there had been a thrombus superimposed on an ulcerated plaque in the aortic arch. Another patient had an intraluminal aortic mass whose echogenicity suggested mural thrombus. In their study of 178 necropsies, Fisher et al found 42 mural thrombi; half of these were found in the aorta. A single observation was reported of a floating thrombus in the arch of the aorta surgically removed in a patient with recurrent peripheral emboli. Thus, as reported in the literature and as we observed in the present study, a thrombus may develop in the aortic arch. Four of our present patients had elevated erythrocyte sedimentation rates and high serum fibrinogen at the onset of stroke that could indicate a hypercoagulable state. It can be hypothesized that such a hypercoagulable state may favor thrombus formation on an ulcerated plaque in the aortic arch.

Transthoracic echocardiography showed no aortic lesion in our study. Transesophageal echocardiographic visualization of the aortic arch was subject to several technical problems, and we improved the technique before we began the study. Usually in standard TEE investigation of the heart, examination of the aorta and especially the arch is quickly undertaken at the time of the withdrawal of the transducer. In the present study the duration of the examination of the arch was 5-10 minutes in all patients. In some cases this procedure was shortened if gag and cough reflexes caused patient discomfort. The aortic valve was identified in cross section; the probe was then slowly withdrawn up to the level of the pulmonary artery bifurcation to visualize the arch and proximal ascending aorta in short axis.
Extreme anteflexion of the tip of the transverse plane scanning probe can display the proximal ascending aorta in long axis. The distal ascending aorta above the level of the right pulmonary artery was not visualized by our monoplane probe because of the interposition of the main stem bronchi and trachea between the upper ascending aorta and the esophagus. This is a limitation of the single-plane transducer. Biplane transducers, allowing a longitudinal view at this level, might provide a clear visualization of the whole ascending aorta. In our patients we therefore saw the arch and the first centimeter of the ascending aorta but not the upper centimeter. Only one patient had magnetic resonance angiography of the aorta and no correlation was possible, but this technique could be of interest in the investigation of the arterial wall of the entire aorta.

In conclusion, in six of 12 patients (50%) older than 60 years of age presenting with cerebral infarct and no detectable cardiac or arterial cause at noninvasive workup, TEE showed intraluminal echogenic masses in the ascending aorta and the arch. This, together with previous pathological and TEE data, points to ulcerated plaques in the aortic arch as a possible source of cerebral emboli. However, case-control studies (such as FAPS) are needed to firmly establish such a causal relationship.

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References

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