Cardioembolic Stroke From Atrial Septal Aneurysm

Giuseppe Di Pasquale, MD, Alvaro Andreoli, MD, Paola Grazi, MD, Paola Dominici, MD, and Giuseppe Pinelli, MD

Atrial septal aneurysm is an uncommon occult cardiac source of cerebral embolism. It is usually asymptomatic, and clinical cardiologic examination and electrocardiography fail to reveal its presence. We report a case of a 34-year-old woman with sudden right hemiplegia and aphasia from occlusion of the left carotid siphon in whom an atrial septal aneurysm was detected by two-dimensional echocardiography. The absence of atherosclerotic risk factors and vascular lesions proximal to the carotid occlusion strengthened a causal relation between atrial septal aneurysm and cerebral infarction. Consequently, two-dimensional echocardiography may be advisable in every patient with unexplained ischemic stroke to detect possible occult embolic cardiac abnormalities. (Stroke 1988;19:640–643)

Case Report

A 34-year-old right-handed woman was admitted 4 hours after abrupt occurrence of right hemiplegia and aphasia. Her medical history was unremarkable; no risk factors for atherosclerosis were present. Cardiac physical examination and electrocardiogram (ECG) were normal; blood pressure was 130/80 mm Hg. Chest x-ray was normal. Computed tomography of the brain was normal on admission but revealed a left frontotemporal low-density area on the second day (Figure 1). Left carotid angiography, performed on admission, showed occlusion of the carotid siphon in the absence of atherosclerotic lesions proximal to the occlusion (Figure 2). Two-dimensional echocardiography (ATL MK 300 IC, phased-array sector scanner) performed in the apical four-chamber and subcostal views showed abnormal systolic bulging of the midportion of the interatrial septum. The motion of the aneurysm from the left into the right atrium occurred during mid- and end-systole of the cardiac cycle. The aneurysmal dilatation protruded about 1.5 cm beyond the plane of the interatrial septum. No other echocardiographic abnormalities were noted (Figure 3). Laboratory investigations, including blood rheology, coagulation tests, and antibodies for lupus, were normal. Twenty-four-hour Holter monitoring did not reveal cardiac arrhythmias.

Since a causal relation between stroke and atrial septal aneurysm was deemed very likely, anticoagulant treatment with warfarin was started. After 7 months' follow-up the patient is moderately disabled. No recurrence of systemic embolism has occurred.

Discussion

Cardiac embolic lesions account for about 15% of ischemic strokes in clinical stroke registries. Medical history, physical examination, and ECG permit us to recognize most embolic cardiac abnormalities so routine echocardiography in unselected patients with cerebral ischemia is probably unnecessary. Atrial septal aneurysm is a striking example of an occult embolic cardiac source of cerebral ischemia. The aneurysm, when it occurs in isolation, is not in fact associated with abnormalities of either physical examination or ECG. Also, in our experience, this lesion turned out to be an unexpected echocardiographic finding.

The embolic potential of atrial septal aneurysm has been claimed by several authors reporting systemic or pulmonary embolism. Anatomic findings support such a clinical assumption; Silver and Dorsey documented the presence of a thrombus at the base of an aneurysm at autopsy, and Grosogol et al described the histologic appearance of a partly organized thrombus in a resected aneurysm. Another cause of systemic
A causal relation between atrial septal aneurysm and stroke in our case is supported by clinical and radiographic findings. The clinical features that suggest embolization include abrupt onset of maximal neurologic deficit in a young active woman without atherosclerotic risk factors. The angiographic features that reinforce the diagnosis of cardiogenic embolism are the occlusion of the internal carotid artery in the siphon in the absence of atherosclerotic lesions proximal to the occlusion.

The association of atrial septal aneurysm with cerebral ischemia has been rarely reported in the literature, and a causal relation cannot be assumed with certainty in every case. Between 1973 and 1986, 13 cases of atrial septal aneurysm associated with cerebral ischemia have been described (Table I). However, in only five cases was a causal relation between that cardiac lesion and cerebral ischemia strongly suggested.

The diagnosis of atrial septal aneurysm has been made occasionally in the past as an unexpected finding at necropsy. Angiocardiography, an invasive technique, is obviously not practical as a routine examination for the detection of the aneurysm. Furthermore, angiocardiography does not allow direct visualization of the interatrial septum but shows a filling defect that may be mistaken for an intra-atrial tumor or thrombus. Intravenous digital subtraction angiography and nuclear imaging have been recently employed for the visualization of atrial septal aneurysm. However, two-dimensional echocardiography is the most useful diagnostic examination for the detection of this lesion. The reliability of this
**FIGURE 3.** Two-dimensional echocardiogram in apical four-chamber (top) and subcostal (bottom) views showing aneurysmal systolic bulging of middle portion of interatrial septum into right atrium.

**TABLE 1. Reported Cases of Atrial Septal Aneurysm Associated With Cerebral Ischemia**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age/sex</th>
<th>Diagnostic detection</th>
<th>Neurologic features</th>
<th>Cerebral angiography</th>
<th>Computed tomography</th>
<th>Therapy</th>
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<tbody>
<tr>
<td>Grosgeaat et al</td>
<td>33/F</td>
<td>AGC</td>
<td>L hemiplegia</td>
<td>R ICA occlusion</td>
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<td>Anticoagulant</td>
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<tr>
<td></td>
<td>39/M</td>
<td>AGC</td>
<td>R hemiplegia</td>
<td>Normal</td>
<td>—</td>
<td>Cardiac surgery</td>
</tr>
<tr>
<td>Yiannikas et al</td>
<td>68/F</td>
<td>DSA/2D echo</td>
<td>R hemiplegia</td>
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</tr>
<tr>
<td>Canny et al</td>
<td>53/F</td>
<td>AGC</td>
<td>L hemiplegia</td>
<td>R MCA occlusion</td>
<td>—</td>
<td>Cardiac surgery</td>
</tr>
<tr>
<td>Gallet et al</td>
<td>61/F</td>
<td>2D echo/AGC</td>
<td>R hemiplegia</td>
<td>Normal*</td>
<td>Ischemic frontoparietal infarct</td>
<td>Anticoagulant</td>
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<td>Hanley et al</td>
<td>64/F</td>
<td>2D echo/NS</td>
<td>CVA (SH)</td>
<td>—</td>
<td>—</td>
<td>Cardiac surgery</td>
</tr>
<tr>
<td></td>
<td>56/M</td>
<td>2D echo</td>
<td>TIA (SH)</td>
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<td></td>
<td>68/F</td>
<td>2D echo</td>
<td>CVA</td>
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<tr>
<td>Belkin et al</td>
<td>75/F</td>
<td>2D echo</td>
<td>CVA</td>
<td>Normal in 1, not performed in 3</td>
<td>Ischemia in 3, not performed in 1</td>
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<tr>
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<td>69/F</td>
<td>2D echo</td>
<td>CVA</td>
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<tr>
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<td>71/F</td>
<td>2D echo</td>
<td>CVA</td>
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<td>68F</td>
<td>2D echo</td>
<td>CVA</td>
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<tr>
<td></td>
<td>59/F</td>
<td>2D echo</td>
<td>TIA</td>
<td>—</td>
<td>Normal</td>
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</table>

Present report: 34/F 2D echo R hemiplegia L ICA occlusion Ischemic frontoparietal infarct Anticoagulant

F, female; M, male; AGC, angiography; DSA, digital subtraction angiography; 2D echo, two-dimensional echocardiography; NS, nuclear scan; L, left; R, right; CVA, cerebrovascular accident; SH, systemic hypertension; TIA, transient ischemic attack; ICA, internal carotid artery; MCA, middle cerebral artery.

*Venous digital subtraction angiography of cervical vessels only.
Atrial Septal Aneurysm and Stroke

Di Pasquale et al

References


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