Angiographic Evaluation of Brain Infarction Limited to the Anterior Cerebral Artery Territory

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Background and Purpose: Brain infarction localized in the anterior cerebral artery territory is rather uncommon, and its etiology has not yet been fully elucidated.

Methods: Based on computed tomographic findings, 17 patients with solitary anterior cerebral artery territory infarction were selected from among 3,619 patients admitted consecutively to our institute. Patients without angiographic examinations were excluded. The angiographic findings and clinical category of stroke were analyzed in each patient.

Results: Angiographic abnormalities were revealed in all patients. These consisted of occlusive changes (n=10) or reversible segmental dilatation (n=3) of the anterior cerebral artery, Al hypoplasia (n=5), and occlusive changes of the carotid artery (n=3). In one patient with anterior cerebral artery occlusion, the occluded artery was reopened and subsequently became reoccluded. The clinical category of stroke was classified as atherothrombotic in 10 patients, cardioembolic in three, and undetermined in the remaining four. In eight of the 10 patients with atherothrombotic infarction, the anterior cerebral artery was narrowed or occluded. In all patients with cardioembolic infarction, the Al segment contralateral to the infarction was hypoplastic.

Conclusions: In our series, solitary anterior cerebral artery territory infarction was attributable most commonly to local atherothrombosis and occasionally to cardiogenic embolism. A hypoplastic Al segment may facilitate the occurrence of embolism in the anterior cerebral artery. Reversible dilatatory and occlusive changes of this artery may be another important cause of infarction. (Stroke 1993;24:549-553)

Key Words • angiography • cerebral arteries • cerebral infarction

Brain infarction localized in the territory of the anterior cerebral artery (ACA) is rather uncommon, and its etiology has not yet been fully elucidated. According to Ethelberg, autopsy findings indicated that the great majority of ACA occlusions were due to atheromatous changes of the vessel and secondary thrombosis, and angiography suggested that the latter condition may appear during a patient's lifetime with a great degree of probability. In contrast, several other workers have claimed that local thrombosis of the ACA should be regarded as a rarity, stating that ACA occlusion was mainly a result of various other mechanisms, such as propagation of thrombotic materials from an occluded internal carotid artery (ICA), artery-to-artery embolism from a site of ICA occlusion or stenosis, and cardioembolic stroke. In the present study, we selected patients with solitary ACA infarction from among more than 3,600 consecutive patients, and reviewed their angiograms in an attempt to elucidate the angiographic features of patients with ACA infarction and to clarify the potential causes of the ACA infarction.

Subjects and Methods

During the period from May 1, 1978, to April 30, 1991, 3,619 patients with cerebral infarction were admitted to our institute. From among these patients, those with cerebral infarction localized in the ACA territory alone (i.e., solitary ACA infarction) were selected retrospectively on the basis of their computed tomographic (CT) findings. The ACA territory was classified according to the atlas of Damasio. We excluded cases of localized infarction in the territory of the recurrent artery of Heubner. We adhered to Fischer's subdivision of the ACA into five segments (A1–A5).

Solitary ACA infarction was found in 23 cases, which amounted to 0.6% of the total. Bilateral carotid angiography was undertaken in 17, and only these 17 patients were subjected to the subsequent analysis. They ranged in age from 38 to 75 (mean±SD, 59±11) years and comprised 11 men and six women.

The transfemoral catheter method was used to perform cerebral angiography in the majority of the patients. The catheter tip was usually lodged in the common carotid artery to opacify the extracranial and intracranial carotid systems. If the transfemoral technique was unsuccessful, direct injection was carried out.
FIGURE 1. Diagrams of topography of infarcts revealed by computed tomographic scans in horizontal plane.

In patient 17, extracranial arteriograms were not obtained, and duplex carotid ultrasonography data were used to estimate the extracranial carotid artery changes.

The risk factors examined included age, sex, smoking habits, hypertension, hypercholesterolemia, diabetes mellitus, and heart disease. Hypertension was judged to be present if the patient had a history of antihypertensive administration or if the blood pressure readings on the medical charts had exceeded 160/90 mm Hg at least twice before the onset of stroke. Hypercholesterolemia was defined as a total serum cholesterol concentration of >220 mg/100 mL on admission. Diabetes mellitus was diagnosed according to the definition cited by the National Diabetes Data Group.8 A diagnosis of heart disease was made on the basis of the patient's history, electrocardiography, echocardiography, and Holter monitoring. Fifteen patients underwent transthoracic echocardiography; only one also underwent transesophageal echocardiography. Because our study dated back to 1978, duplex carotid ultrasonography was performed only in nine patients. We were unable to compare angiographic findings with the data from noninvasive techniques.

We classified the infarctions according to the system of the National Institute of Neurological Disorders and

FIGURE 2. Case 1. Panels A and B: Computed tomographic scans showing hypodense regions with areas of increased density (hemorrhagic infarction) in the bilateral anterior cerebral artery (ACA) territories. Panels C and D: Right and left carotid angiograms. Hypoplasia of right A1 segment of ACA and tight stenosis of left A1 segment (arrow) are observed.

Stroke as being atherothrombotic, cardioembolic, lacunar, or undetermined.9

Results

The topographical distribution of the individual infarcts as revealed on CT slices is shown diagrammatically in Figure 1. Nine patients had left-sided infarcts, six had right-sided infarcts, and two had bilateral infarcts.

The clinical characteristics of the patients are summarized in Table 1. Four patients displayed atrial fibrillation; patient 1 had long QT syndrome, patient 10 had mitral stenosis, and the remaining two patients exhibited nonvalvular atrial fibrillation. One patient had a history of myocardial infarction.

Angiographic abnormalities were revealed in all patients. Ten patients had in situ occlusion or stenosis of the ACA ipsilateral to the infarct. Three patients displayed ACA dilatation with no other abnormalities. The remaining four patients had no ACA lesions with or without occlusive changes in the carotid arteries.

Among the 10 patients with in situ occlusion or stenosis of the ACA (patients 1–10), one demonstrated stenosis of the A1 segment, six had occlusion or stenosis of A2, one had occlusion of A3, and two had occlusion of A4. The clinical classification of the infarcts was atherothrombotic in eight patients, cardioembolic in
TABLE 1. Clinical Characteristics of 17 Patients With Isolated Anterior Cerebral Artery Territory Infarction

<table>
<thead>
<tr>
<th>Patient/age/sex</th>
<th>Pattern of onset</th>
<th>Risk factors</th>
<th>Heart disease</th>
<th>Lesion side</th>
<th>Angiographic findings</th>
<th>Other findings</th>
<th>Clinical category</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/72/F</td>
<td>I</td>
<td>HT</td>
<td>Long QT, AF</td>
<td>B</td>
<td>L A1 stenosis, 90%</td>
<td>B stenosis, &lt;25%</td>
<td>R A1 hypoplasia</td>
</tr>
<tr>
<td>2/64/M</td>
<td>P, 3 days</td>
<td>DM, SM</td>
<td>L</td>
<td>L</td>
<td>L A2 occlusion</td>
<td>L stenosis, &lt;25%</td>
<td>R MCA stenosis</td>
</tr>
<tr>
<td>3/67/M</td>
<td>I</td>
<td>HT, SM</td>
<td>L</td>
<td>L</td>
<td>L A2 occlusion</td>
<td>Normal</td>
<td>L MCA stenosis</td>
</tr>
<tr>
<td>4/38/M</td>
<td>I</td>
<td>HT</td>
<td>R</td>
<td>R</td>
<td>R A2 occlusion</td>
<td>Normal</td>
<td>ROP &amp; ROC</td>
</tr>
<tr>
<td>5/50/M</td>
<td>I</td>
<td>DM, SM</td>
<td>L</td>
<td>L</td>
<td>L A2 stenosis, 99%</td>
<td>L stenosis, &lt;25%</td>
<td></td>
</tr>
<tr>
<td>6/50/F</td>
<td>I</td>
<td>HT, SM</td>
<td>R</td>
<td>R</td>
<td>R A2 stenosis, 99%</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>7/68/F</td>
<td>P, 8 days</td>
<td>...</td>
<td>L</td>
<td>L</td>
<td>L A2 stenosis, 90%</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>8/71/F</td>
<td>P, 7 days</td>
<td>HT, DM</td>
<td>L</td>
<td>L</td>
<td>L A3 occlusion</td>
<td>B irregularity</td>
<td></td>
</tr>
<tr>
<td>9/62/M</td>
<td>P, 9 days</td>
<td>SM</td>
<td>L</td>
<td>L</td>
<td>L A4 occlusion</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>10/57/M</td>
<td>I</td>
<td>MS, AF</td>
<td>R</td>
<td>R</td>
<td>R A4 occlusion</td>
<td>Normal</td>
<td>L A1 hypoplasia</td>
</tr>
<tr>
<td>11/53/F</td>
<td>I</td>
<td>HT</td>
<td>B</td>
<td>L</td>
<td>L A2 dilatation</td>
<td>Normal</td>
<td>Normalized later</td>
</tr>
<tr>
<td>12/43/M</td>
<td>I</td>
<td>HT, SM</td>
<td>R</td>
<td>R</td>
<td>R A2 dilatation</td>
<td>Normal</td>
<td>Normalized later</td>
</tr>
<tr>
<td>13/51/F</td>
<td>I</td>
<td>MS, AF</td>
<td>L</td>
<td>L</td>
<td>L A2 dilatation</td>
<td>Normal</td>
<td>Normalized later</td>
</tr>
<tr>
<td>14/69/M</td>
<td>I</td>
<td>DM, OMI</td>
<td>L</td>
<td>Normal</td>
<td>Normal</td>
<td>R stenosis, 50%</td>
<td>R A1 hypoplasia</td>
</tr>
<tr>
<td>15/62/M</td>
<td>I</td>
<td>DM, OMI</td>
<td>L</td>
<td>Normal</td>
<td>R occlusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16/43/M</td>
<td>I</td>
<td>HC, SM</td>
<td>R</td>
<td>Normal</td>
<td>Normal</td>
<td>L stenosis, 50%</td>
<td>R A1 hypoplasia</td>
</tr>
<tr>
<td>17/75/M</td>
<td>I</td>
<td>HT</td>
<td>R</td>
<td>Normal</td>
<td>L stenosis, 50%†</td>
<td>R A1 hypoplasia</td>
<td></td>
</tr>
</tbody>
</table>

ACA, anterior cerebral artery; CCA, common carotid artery; ICA, internal carotid artery; MCA, middle cerebral artery; M, male; F, female; I, immediately complete; P, progressive; HT, hypertension; DM, diabetes mellitus; SM, smoking habit; HC, hypercholesterolemia; Long QT, long QT syndrome; AF, atrial fibrillation; MS, mitral stenosis; OMI, old myocardial infarction; R, right; L, left; B, bilateral; ROP & ROC, reopening followed by reocclusion.

*After cardiac arrest.
†By duplex carotid ultrasonography.

one, and undetermined in one. Stenosis of the left A1 segment resulted in extensive bilateral infarction in patient 1 because both ACAs originated from this segment as a result of hypoplasia of the right A1 segment (Figure 2). A hemodynamic mechanism was thought probable in this case because the ischemic symptoms developed after cardiac arrest. Four patients with progressing stroke (patients 2 and 7–9) were classified as having thrombotic infarction. The CT scans and angiograms of patient 2 are presented in Figure 3. Two patients (5 and 6) in whom the neurological deficits were established immediately were also assigned to the

FIGURE 3. Case 2. Panel A: Computed tomographic scan showing hypodense area in left anterior cerebral artery (ACA) territory. Panel B: Left carotid angiogram revealing occlusion of left ACA in A2 segment (arrow), with leptomeningeal collaterals arising from branches of left middle cerebral artery.
thrombotic infarction group because they demonstrated no significant carotid lesions or embolic heart disease. Although patient 3 exhibited nonvalvular atrial fibrillation and sudden onset of stroke, the arteriosclerotic changes of the intracranial arteries were so severe that a thrombotic etiology also seemed most likely. Cardioembolic infarction was diagnosed in patient 10, who had mitral stenosis associated with atrial fibrillation and was admitted with immediately completed stroke. The angiography in this case demonstrated A4 occlusion, contralateral A1 hypoplasia, and normal carotid arteries. The clinical category of stroke was undetermined in one young adult patient (patient 4), in whom unusual angiographic findings were observed. The angiograms demonstrated A2 occlusion at the acute phase and its reopening 3 weeks afterward. Furthermore, the A2 was found to be occluded again 1 year after the stroke (Figure 4).

The second group of patients (patients 11-13) comprised three cases displaying segmental dilatation of the A2 segment at the acute stage of stroke. All of these patients were middle-aged and had no arrhythmia or cardiac disease. Their symptoms developed abruptly and were unrelated to head injury. The infarcts were extensive or saltatory and were hemorrhagic on CT scan. No other abnormalities were found on their angiograms. Repeat angiography 1-6 years after ictus demonstrated restoration of the dilated A2 segment to its normal caliber.

The four patients (14-17) of the final group revealed no significant lesions in the ACA. All suffered immediately completed strokes. There was a diagnosis of cardioembolic stroke in two (patients 14 and 15). One of these (patient 14) exhibited nonvalvular atrial fibrillation, and his angiograms showed normal extracranial and intracranial arteries except for hypoplasia of the A1 segment contralateral to the infarct (Figure 5). Patient 15 had an old myocardial infarction. Angiograms revealed no lesions in the intracranial arteries except for hypoplasia of the A1 segment contralateral to the infarct. This patient also had stenosis of the ICA ipsilateral to the A1 hypoplasia. The occurrence of ACA infarction was thus unrelated to the carotid stenosis. The clinical category of stroke in the remaining two patients (16 and 17) was atherothrombotic. Occlusion of the ICA ipsilateral to the ACA territory infarct was demonstrated in one patient with normal ACAs on his angiograms (patient 16). Although patient 17 did not undergo angiography of the cervical carotid arteries, duplex carotid ultrasonography revealed 50% stenosis of the right ICA. Intracranial angiograms showed normal ACAs except for hypoplasia of the A1 segment contralateral to the ICA stenosis. It appeared that the mechanism of stroke in these two patients was probably artery-to-artery embolism.

Discussion

In our series of 17 patients with solitary ACA infarction, the clinical category of stroke was judged to be atherothrombotic in 10 patients (59%), cardioembolic in three (18%), and undetermined in four (24%). Among the 10 patients with atherothrombotic stroke, eight were presumed to have local thrombosis as the pathogenesis. There were no cases of antegrade thrombus extending from the ICA occlusion to the ACA. Embolism from an occlusive lesion of the ICA was thought to be the etiology in only two patients.

Most studies undertaken in Western countries have indicated that the principal mechanisms of ACA territory infarction are cardioembolic stroke and propagation of thrombotic materials or artery-to-artery embolism from an occlusive ICA lesion to the ACA. The difference between these findings and ours may be
attributable to the fact that sclerosis of the intracranial arteries tends to be more common than extracranial arteriosclerosis in Japanese patients. It is of interest that the Lausanne Stroke Registry showed ACA occlusion without a potential source of embolism in only one Vietnamese patient. Solitary ACA infarction in oriental populations may tend to be based on a thrombotic etiology. In our study, the site most commonly or prevalently affected by atherothrombotic changes was the A2 segment.

A hypoplastic A1 segment was observed in all three patients with cardiogenic embolism and in one of two patients with artery-to-artery embolism from an ICA lesion. The anatomy of the anterior circle of Willis is known to vary considerably. In a pathological study on a series of 350 normal brains, 2% were found to have a string-like A1 segment that was less than 1 mm in external diameter. In an angiographic study, a hypoplastic A1 segment was detected in 8.6% on the right side and in 4.1% on the left side. In patients with hypoplasia of the unilateral A1 segment, the distal areas are usually filled from the contralateral A1 segment that supplies both distal ACAs. In such patients, emboli derived from the heart or carotid arteries may be prone to reach the distal ACAs through the contralateral A1 segment with an increased blood flow compared with that in patients with normal vascular structures. Gacs et al reported that embolic occlusion of the ACA occurred under unusual hemodynamic circumstances, such as an increased flow through the anterior communicating artery due to unilateral ICA occlusion. In this context, a hypoplastic A1 segment may have a similar hemodynamic significance to unilateral ICA occlusion. It is postulated that the hypoplastic A1 segment may facilitate the occurrence of embolism in the ACA territory.

The four patients with an undetermined category of stroke revealed peculiar changes on serial angiography. In one patient, the A2 segment was initially occluded and subsequently reopened. Furthermore, the A2 segment again became occluded subsequently. In the three other patients, the A2 segment was initially dilated and later returned to a normal state. Although the mechanisms remain unknown, the most likely etiologies are considered to be “reversible cerebral segmental vasoconstriction and dilatation,” described by Call et al, and isolated ACA dissections. Reversible arterial lesions confined to the ACA may be important causes of ACA territory infarction.

In conclusion, angiographic abnormalities almost always appear to be found in patients with solitary ACA infarction. Cerebral angiography may thus be of considerable value for evaluating the mechanisms involved in the occurrence of such infarcts.

References

Angiographic evaluation of brain infarction limited to the anterior cerebral artery territory.
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