Middle Cerebral Artery Stenosis Is a Major Clinical Determinant in Striatocapsular Small, Deep Infarction

Oh Young Bang, MD; Ji Hoe Heo, MD; Jung Yeon Kim, MD; Jae Hyun Park, MD; Kyoon Huh, MD

Background: The significance of the stenotic lesions of the middle cerebral artery (MCA) in Asian patients with striatocapsular small, deep infarctions (SSDIs) remains undetermined.

Objectives: To investigate the frequency of stenotic lesions of the MCA in patients with SSDIs and to evaluate clinical and radiological features in those same patients.

Setting: Acute stroke registry of a university hospital.

Patients and Methods: One hundred two Korean patients with acute symptomatic SSDIs underwent cerebral angiography or magnetic resonance angiography and echocardiography. We divided these patients into 2 groups—patients with and without MCA occlusive lesions. The clinical and magnetic resonance image features were compared between these 2 groups.

Results: Thirty-seven patients (36%) had an ipsilateral proximal MCA lesion, whereas 65 patients (64%) showed no MCA abnormality on cerebral angiography or magnetic resonance angiography. Among 65 patients without an MCA lesion, 18 had an embolic source; the remaining 37 patients had no demonstrable embolic source. There were significant differences in the temporal profile and magnetic resonance imaging findings between the groups. Although the type of lacunar syndrome and the volume of infarcts did not differ between the groups, the unstable temporal profile and magnetic resonance imaging findings of multiple small infarcts in the symptomatic hemisphere were frequently observed in patients with MCA lesions.

Conclusions: The proximal MCA lesion was a common cause of SSDIs in Korean patients. Depending on the existence of an MCA lesion, the clinical course and magnetic resonance imaging feature of the patients with SSDIs were different.

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**PATIENTS AND METHODS**

From January 6, 1996, to May 25, 2001, we retrospectively studied consecutive patients with acute symptomatic SS-DIs. The inclusionary criteria were as follows: patients who had suffered traditional lacunar syndrome and who had been observed within 1 week after the onset of symptoms, those who underwent cerebral angiography or MRA within 2 weeks after the onset of symptoms, and those who had MRI-confirmed relevant focal small (<15-mm), deep infarcts within the striatocapsular area. Of the patients who had multiple lesions or no abnormalities on MRI, we selected only those who underwent diffusion-weighted MRI.

Of 292 patients who suffered from lacunar syndrome and had small, deep infarcts on MRI during the study period, 102 composed the cohort. The primary reasons for exclusion were as follows: (1) small, deep infarcts located outside striatocapsular area (ie, the territory of the posterior circulation, superficial pial MCA, or anterior choroidal artery) (n = 142 patients); (2) no documentation of a relevant lesion on MRI but the patient did not undergo diffusion-weighted MRI (n = 6 patients); (3) SS-DIs associated with specific disease process (n = 7 patients); or (4) no undergo either cerebral angiography or MRA within 2 weeks after the onset of symptoms (n = 35 patients). We divided the patients into 2 groups according to angiographic findings. The first group included patients with mural atheroma at the proximal MCA (M1), which showed an occlusive lesion at the origin of MCA penetrating arteries on cerebral angiography or MRA. The second group included those who had a normal MCA on cerebral angiography or MRA.

We reviewed the medical history, general physical and neurological examination findings, and laboratory test results. National Institutes of Health Stroke Scale (NIHSS) score was checked at admission to the hospital as well as on days 1, 3, and 7 after admission. Clinical course was determined after a 1-week follow-up period and defined as follows: improved, when the NIHSS score decreased more than 2 points; stable, when the score decreased less than 2 points; worsening, when the score increased after admission; and fluctuating, when the score episodically increased and then decreased or vice versa. Electrocardiography was performed in all patients, and echocardiography was performed in 77 patients, especially when the possibility of cardioembolic sources was suggested or when no other cause of stroke was found.

Brain MRI (1.5 T) was performed in all patients and diffusion-weighted MRI in 78 patients. The diagnosis of infarcts in the striatocapsular distribution was made with the use of previously published templates. The volume of the lesion was calculated from the MRI using the methods of Nelson et al. Cerebral angiography was performed in 40 patients and MRA in the remaining 62 patients. We measured M1 stenosis on MRA or conventional angiography by previously suggested methods. The M1 stenosis was calculated according to the residual luminal diameter measured at the site of maximal narrowing and the diameter of the adjacent normal vessel, from which a percentage of stenosis was calculated. The degree of stenosis was graded as follows: lower degree of stenosis (50%-69%) or higher degree of stenosis (≥70%). All patients who were classified as having a higher degree of stenosis on MRA had segmental loss of signal at the stenotic site. In the patients who underwent MRA, ultrasonography or MRA of the carotid artery was also performed. Internal carotid artery stenosis was calculated using the North American Symptomatic Carotid Endarterectomy Trial study method. χ² Test and t test were used to compare clinical and radiological findings between the 2 groups.

### Table 1. Angiographic Classification of 102 Patients With Striatocapsular Small, Deep Infarctions

<table>
<thead>
<tr>
<th>Source of Embolism</th>
<th>No M1 Stenosis Group (n = 65)</th>
<th>M1 Stenosis Group (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>47</td>
<td>30</td>
</tr>
<tr>
<td>Associated sources</td>
<td>81</td>
<td>7</td>
</tr>
</tbody>
</table>

**CARDIOEMBOLISM**

- AF
- AF with AAA
- Akinetic left ventricular segment
- Left ventricular thrombus
- CHF with AAA
- ICA lesion, % of stenosis†
  - <50
  - 50-74
  - ≥75
- Both cardioembolism and ICA lesion†
  - MVP and ICA stenosis
    - of <50%
    - of ≥50%-74%
[†]All values indicate that the stenotic lesion was on the symptomatic side.

**RESULTS**

Among the 102 patients, 68% were men and 32% were women, and their ages ranged from 35 to 85 years (mean age, 58 years). The angiographic and echocardiographic findings are listed in Table 1.

Thirty-seven (36%) of the 102 patients had a symptomatic MCA lesion. The lesions were disclosed in 20 patients by cerebral angiography and in 17 patients by MRA. Twenty-one patients had a high degree of stenosis; 16 patients had a lower degree of stenosis.

Embolic sources from the internal carotid artery, aorta, and heart were found in 23 patients (25%). Cardiac sources included atrial fibrillation, congestive heart failure with aortic arch atheroma (AAA), AAA with patent foramen ovale, AAA with atrial fibrillation, akinetic left ventricular segment, and aortic stenosis with cardiac thrombi. The patients who had MCA more often had embolic sources than those who had a stenotic MCA.

**CLINICAL FEATURES**

None had cortical dysfunction, such as aphasia or agnosia. Faciobrachialocrural involvement was characteristic, except for the patients with dysarthria clumsy hand syndrome or ataxic hemiparesis (Table 2). The age, sex
Table 2. Clinical and Radiological Features in Each Group of Patients

<table>
<thead>
<tr>
<th>Clinical Presentation and Course</th>
<th>No Embolic Source (n = 47)</th>
<th>Associated Embolic Source (n = 18)</th>
<th>Lower Degree, 50%-69% (n = 16)</th>
<th>Higher Degree, &gt;70% (n = 21)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporal profile, No. (%) of patients†</td>
<td>Improving or stable</td>
<td>41 (87)</td>
<td>16 (89)</td>
<td>12 (75)</td>
<td>11 (52)</td>
</tr>
<tr>
<td>Worsening or fluctuating</td>
<td>6 (13)</td>
<td>2 (11)</td>
<td>4 (25)</td>
<td>10 (48)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Previous TIA episodes, No. (%) of patients</td>
<td>7 (15)</td>
<td>4 (22)</td>
<td>7 (44)</td>
<td>7 (33)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Lacunar syndrome type</td>
<td>Pure motor hemi</td>
<td>33</td>
<td>10</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>Sensory motor stroke</td>
<td>8</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Dysarthria clumsy hand syndrome</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Ataxic hemiparesis</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>&gt;.05</td>
</tr>
</tbody>
</table>

MRI Findings

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Embolic Source (n = 18)</th>
<th>Associated Embolic Source (n = 18)</th>
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<th>Higher Degree, &gt;70% (n = 21)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume of infarction, mean (SD), cm³</td>
<td>0.7 (0.5)</td>
<td>1.0 (0.7)</td>
<td>0.8 (0.8)</td>
<td>1.1 (0.8)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Laterality of small, deep infarcts</td>
<td>Unilateral multiple lesions, No. (%) of patients‡</td>
<td>3 (11)</td>
<td>2 (9)</td>
<td>1 (2)</td>
<td>8</td>
</tr>
<tr>
<td>Restricted to striatocapsular area</td>
<td>4 (22)</td>
<td>1 (6)</td>
<td>3 (17)</td>
<td>3</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Associated lesion on Cx or subCx WM</td>
<td>4 (25)</td>
<td>3 (19)</td>
<td>1 (6)</td>
<td>7 (33)</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Unilateral multiple lesions, No. (%) of patients‡</td>
<td>3 (11)</td>
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</tbody>
</table>

*Data are given as the number of patients unless otherwise indicated. M1 indicates the proximal portion of the middle cerebral artery; ellipses, not applicable; TIA, transient ischemic attack; MRI, magnetic resonance imaging; Cx, cortex; and subCx WM, subcortical white matter.
†The National Institutes of Health Stroke Scale (NIHSS) was used to score the patient’s clinical course after a 1-week follow-up period. The scores indicated the following status: improved, a more than 2-point decrease in the NIHSS score; stable, a less than 2-point decrease in the NIHSS score; fluctuating, episodic change in NIHSS score; and worsening, any increase in NIHSS score after admission to the hospital.
‡All lesions were recently developed small infarcts.

M1 Stenosis Group

In this study, about 40% of the patients with SSDs had stenotic lesions of the MCA, which is a parent artery of the lenticulostriate arteries. Intracranial stenosis has been reported to be neglected as a cause of small, deep infarction; however, about half of the patients who had MCA stenosis also had small, deep infarctions.13,14 Findings from our present study suggest that large-artery atherosclerosis and small arterial abnormalities may present with symptomatic SSDs in a significant fraction of patients. Since intracranial atherosclerosis is more common in Asians than in Westerners, who are known to have extracranial stenosis more frequently,15 it is conceivable that intracranial stenosis may play an important role in the development of small, deep infarctions in Asians.13

Patients with stenotic MCA lesions showed clinical characteristics distinct from those with the normal MCA.
A history of TIA and temporal profile of fluctuation or worsening of symptoms were frequent in the patients with stenotic MCA lesions. Fisher reported that vascular stenosis was associated with an unstable clinical course. Capsular lacunar syndromes with prior ipsilateral TIA were not benign and indicated large-vessel disease of either the MCA or the internal carotid artery. Our findings, in agreement with published data, suggest that patients with small, deep infarction associated with occlusive diseases of the parent artery may experience unstable clinical courses.

With the introduction of the diffusion-weighted imaging, silent infarctions at different sites from the symptomatic small, deep infarction could be detected. Multiple infarctions on the ipsilateral striatocapsular area or cortical and subcortical areas supplied by the MCA were other distinct features of severe MCA stenosis. Thromboembolism from the atherosclerotic plaque of the MCA might be responsible for the frequent association of multiple infarctions in the patients with severe MCA stenosis. Multiple small lesions on diffusion-weighted MRI were reported to be more likely to harbor an identifiable stroke mechanism than a single small, deep infarction.

Several limitations of our present study deserve to be mentioned. Although proximal embolic sources were not found in most of the patients with an MCA lesion, a signal gap in the MRA or even a narrowing in the angiogram may represent only a partially recanalized clot. Also, there may be a discrepancy in the degree of stenosis between MRA and angiogram. None of our patients underwent both MRA and conventional angiogram.

**CONCLUSIONS**

Striatocapsular small infarctions were frequently associated with occlusive lesions of the MCA, which may require diagnostic and therapeutic approach different from the small arterial disease of good prognosis. A history of preceding TIA, fluctuating or progressive clinical courses,
and ipsilateral multiple infarctions on MRI are findings that strongly suggest occlusive lesions of the MCA.

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**Author contributions:** Study concept and design (Drs Bang and Huh); acquisition of data (Drs Bang, Kim, and Park); analysis and interpretation of data (Drs Bang and Heo); drafting of the manuscript (Drs Bang, Kim, and Park); critical revision of the manuscript for important intellectual content (Drs Heo and Huh); statistical expertise (Dr Bang); administrative, technical, and material support (Dr Kim, Park, and Huh); and study supervision (Drs Heo and Huh).

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**REFERENCE**