Single Subcortical Infarction and Atherosclerotic Plaques in the Middle Cerebral Artery High-Resolution Magnetic Resonance Imaging Findings

Youngshin Yoon, MD; Deok Hee Lee, MD, PhD; Dong-Wha Kang, MD, PhD; Sun U. Kwon, MD, PhD; Jong S. Kim, MD, PhD

Background and Purpose—Single subcortical infarction (SSI) may be classified as proximal SSI (pSSI) or distal SSI (dSSI) according to its extension to the middle cerebral artery (MCA). We investigated the differences between pSSI and dSSI in terms of their clinical features, lesion size, and the frequency of MCA plaques detected by high-resolution MRI.

Methods—Thirty-nine patients with SSI (20 pSSI and 19 dSSI) were prospectively enrolled who did not show relevant MCA disease on MR angiography. Lesion size, neurological status (initial National Institutes of Health Stroke Scale score and modified Rankin Scale at 3 months), and the presence and location (superior versus inferior) of high-resolution MRI–identified plaques were evaluated.

Results—The frequencies of MCA plaques did not differ between patients with pSSI and those with dSSI (8 [40%] versus 12 [63.2%]; P=0.205); however, superiorly located plaques were significantly more common in patients with pSSI than in those with dSSI (6 [75%] versus 2 [16.7%]; P=0.019). Initial lesion volumes were larger (1.96±1.18 versus 1.11±1.11 mm³; P=0.025), National Institutes of Health Stroke Scale scores were higher (5 [3–6.75] versus 3 [1–3] points; P=0.017), and microbleeds were fewer (1 [5%] versus 10 [52.6%]; P=0.001) in patients with pSSI than in those with dSSI. Three-month modified Rankin Scale scores were higher in patients with superior plaques than in those with inferior plaques.

Conclusions—Compared with dSSI, pSSI is closely associated with large lesions, severe clinical symptoms, and superiorly located MCA plaques, suggesting that the location, rather than simple presence of plaques, determines the SSI location. (Stroke. 2013;44:2462-2467.)

Key Words: cerebral infarction – high-resolution MRI – plaque, atherosclerotic

Single subcortical infarction (SSI) in the middle cerebral artery (MCA) territory is a heterogeneous condition. SSI may be caused by lipohyalinotic small-vessel disease, atherosclerotic small-vessel disease, and the occlusion of perforators because of parental artery atherothrombosis. On the basis of location of a MRI-identified lesion, SSIs may be classified as those extending to the surface of the MCA (proximal SSI [pSSI]) or those that do not (distal SSI [dSSI]; Figure 1A and 1B). Previous studies have shown that dSSI is associated with the characteristics of small-vessel disease, including abundant microbleeds and white matter ischemic changes, whereas pSSI is more often associated with atherogenic characteristics, such as the concomitant presence of atherosclerotic cerebral vessels.

Recently, studies have reported that high-resolution MRI (HR-MRI) can show the vessel wall structure and, therefore, detect early atherosclerotic changes such as plaques, wall thickening, and arterial remodeling, even in patients with normal findings on magnetic resonance angiography (MRA). Because the lesion characteristics of pSSI are similar to those of SSI associated with parental artery disease, pSSI may be more closely associated with HR-MRI–identified MCA plaques than dSSI in patients who do not show parental artery disease on MRA.

The aim of the present study is to investigate the differences in the clinical features, lesion size, and HR-MRI–identified MCA plaques between patients with pSSI and those with dSSI. We also investigated whether the SSI characteristics (eg, lesion location, volume, and diameter) can predict the presence of MCA plaques in these patients.

Methods

Patients
We prospectively enrolled consecutive patients who were admitted to the Department of Neurology of Asan Medical Center between July 2011 and August 2012 with the following characteristics: (1)
infarction in the lenticulostriate arterial territory (basal ganglia, corona radiata, and internal capsule) that was identified using diffusion-weighted imaging (DWI) performed within 72 hours of symptom onset; (2) no relevant MCA disease that was confirmed by MRA; and (3) no identified source of the embolism (eg, embolicigenic cardiac disease, atrial fibrillation, recent myocardial infarction, dilated cardiomyopathy, valvular heart disease, and infective endocarditis) or significant (≥50%) stenosis of the relevant internal carotid artery (ICA). All of the enrolled patients underwent follow-up MR imaging using HR-MRI on the fifth day after the initial MRI.

Imaging Analysis

Follow-up images of the MCA main trunk supplying the infarcted region were acquired using 3.0-T HR-MRI (Achieva scanner; Philips, Eindhoven, Netherlands). DWI (axial and coronal), 3-dimensional time-of-flight MRA, T1-weighted, T2-weighted, proton density–weighted, and contrast-enhanced proton density images were obtained. The parameters of the imaging sequences were as follows: T1W (repetition time/echo time, 600/12 ms; slice thickness, 1.5 mm; 512×512 matrix); T2W (repetition time/echo time, 3000/80 ms; slice thickness, 1.5 mm; 512×512 matrix); and proton density (repetition time/echo time, 1000/20 ms; field of view, 200×200 mm; matrix size, 720×720 matrix; slice thickness, 1 mm; interslice gap, 0.5 mm; average, 1). Sagittal images were taken perpendicular to the M1 segment of the relevant MCA. Postenhanced proton density images were obtained using gadoterate meglumine if there were no contraindications.

The HR-MRI images were reviewed and interpreted by consensus between 1 neurologist (Y.Y.) and 1 neuroradiologist (D.H.-L.) both of whom were blind to the findings of the initial MRI and clinical characteristics of the patients. Plaques were identified on the basis of the presence of eccentric wall thickening, where thin sections were identified if they were estimated to be <50% of the thickness of the thickest point by visual inspection (Figure 1C and 1D). The locations of atherosclerotic plaques were classified as centered on the superior side (the usual origin of MCA perforators) or the inferior side of the vessel (Figure 2). The lesion location relative to the parental artery was classified as pSSI (extending to the surface of the MCA) or dSSI (not extending to the surface of the MCA). Lesion size was defined as large when the infarct was shown in >4 axial slices in DWI. The infarction volume on the initial and follow-up DWI examinations was defined as the total hyperintense area of each slice multiplied by the slice thickness, as determined using...
### Table 1. Characteristics of Patients With pSSI and Those With dSSI

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>pSSI (n=20)</th>
<th>dSSI (n=19)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y (mean±SD)</strong></td>
<td>61.8±10.19</td>
<td>65.1±10.94</td>
<td>0.335</td>
</tr>
<tr>
<td><strong>Men, %</strong></td>
<td>13 (65.0)</td>
<td>7 (36.8)</td>
<td>0.113</td>
</tr>
<tr>
<td><strong>Vascular risk factors, %</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>10 (50.0)</td>
<td>13 (68.4)</td>
<td>0.333</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1 (5.0)</td>
<td>5 (26.3)</td>
<td>0.091</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>2 (10.0)</td>
<td>5 (26.3)</td>
<td>0.235</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>0</td>
<td>2 (10.5)</td>
<td>0.231</td>
</tr>
<tr>
<td>Current smoker</td>
<td>3 (15.0)</td>
<td>3 (15.8)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Previous history of stroke</td>
<td>2 (10.0)</td>
<td>5 (26.3)</td>
<td>0.235</td>
</tr>
<tr>
<td><strong>MR findings</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large infarcts, %</td>
<td>17 (85.0)</td>
<td>1 (5.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Initial diameter, mm (mean±SD)</td>
<td>1.7±0.56</td>
<td>1.3±0.59</td>
<td>0.083</td>
</tr>
<tr>
<td>Follow-up diameter, mm (mean±SD)</td>
<td>1.88±0.64</td>
<td>1.81±0.63</td>
<td>0.806</td>
</tr>
<tr>
<td>Initial volume, mm³ (mean±SD)</td>
<td>1.96±1.18</td>
<td>1.11±1.11</td>
<td>0.025</td>
</tr>
<tr>
<td>Follow-up volume, mm³ (mean±SD)</td>
<td>2.97±1.80</td>
<td>1.94±1.88</td>
<td>0.090</td>
</tr>
<tr>
<td>(Follow-up)-initial volume, mm³ (mean±SD)</td>
<td>1.01±1.08</td>
<td>0.84±1.10</td>
<td>0.631</td>
</tr>
<tr>
<td>Concomitant cerebral arterosclerosis, %</td>
<td>7 (35.0)</td>
<td>8 (42.1)</td>
<td>0.748</td>
</tr>
<tr>
<td>Presence of microbleeds, %</td>
<td>1 (5.0)</td>
<td>10 (52.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Fazekas grade on DWM (median [IQR])</td>
<td>0 (0–2)</td>
<td>1 (1–2)</td>
<td>0.112</td>
</tr>
<tr>
<td>Fazekas grade on PVWM (median [IQR])</td>
<td>1 (1–1.75)</td>
<td>1 (1–2)</td>
<td>0.503</td>
</tr>
<tr>
<td>Vessel wall findings, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presence of plaque</td>
<td>8 (40.0)</td>
<td>12 (63.2)</td>
<td>0.205</td>
</tr>
<tr>
<td>Plaques in superior side (among patients with plaque)</td>
<td>6 (75.0)</td>
<td>2 (16.7)</td>
<td>0.019</td>
</tr>
<tr>
<td>Clinical assessments</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIHSS score at admission (median [IQR])</td>
<td>5 (3–6.75)</td>
<td>3 (1–3)</td>
<td>0.017</td>
</tr>
<tr>
<td>Patients with neurological progression, %</td>
<td>1 (5.0)</td>
<td>2 (10.5)</td>
<td>0.605</td>
</tr>
<tr>
<td>3-month mRS (median [IQR])</td>
<td>0.5 (0–1)</td>
<td>0</td>
<td>0.064</td>
</tr>
</tbody>
</table>

**dSSI** indicates distal single subcortical infarction; **DWM**, deep white matter; **mRS**, modified Rankin Scale; **NIHSS**, National Institutes of Health Stroke Scale; **pSSI**, proximal single subcortical infarction; **PVWM**, periventricular white matter; and **IQR**, interquartile range.

### Table 2. Patient Characteristics According to the Presence of MCA Plaques

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Plaque+ (n=20)</th>
<th>Plaque− (n=19)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y (mean±SD)</strong></td>
<td>65.8±10.6</td>
<td>60.9±10.2</td>
<td>0.818</td>
</tr>
<tr>
<td><strong>Men, %</strong></td>
<td>10 (50.0)</td>
<td>10 (52.6)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td><strong>Vascular risk factors, %</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (55.0)</td>
<td>12 (63.2)</td>
<td>0.748</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4 (20.0)</td>
<td>2 (10.5)</td>
<td>0.661</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>4 (20.0)</td>
<td>3 (15.8)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>0</td>
<td>2 (10.5)</td>
<td>0.231</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1 (5.0)</td>
<td>5 (26.3)</td>
<td>0.091</td>
</tr>
<tr>
<td>Previous history of stroke</td>
<td>3 (15.0)</td>
<td>4 (21.1)</td>
<td>0.695</td>
</tr>
<tr>
<td><strong>MR findings</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large infarcts, %</td>
<td>8 (40.0)</td>
<td>10 (52.6)</td>
<td>0.527</td>
</tr>
<tr>
<td>Initial diameter, mm (mean±SD)</td>
<td>1.48±0.51</td>
<td>1.62±1.48</td>
<td>0.445</td>
</tr>
<tr>
<td>Follow-up diameter, mm (mean±SD)</td>
<td>1.81±0.60</td>
<td>1.85±0.67</td>
<td>0.836</td>
</tr>
<tr>
<td>Initial volume, mm³ (mean±SD)</td>
<td>1.24±0.87</td>
<td>1.87±1.48</td>
<td>0.100</td>
</tr>
<tr>
<td>Follow-up volume, mm³ (mean±SD)</td>
<td>2.34±1.93</td>
<td>2.61±1.89</td>
<td>0.667</td>
</tr>
<tr>
<td>(Follow-up)-initial volume, mm³ (mean±SD)</td>
<td>1.11±1.32</td>
<td>0.73±0.73</td>
<td>0.287</td>
</tr>
<tr>
<td>Concomitant cerebral arterosclerosis, %</td>
<td>9 (45.0)</td>
<td>6 (31.6)</td>
<td>0.514</td>
</tr>
<tr>
<td>Presence of microbleeds, %</td>
<td>8 (40.0)</td>
<td>3 (15.8)</td>
<td>0.155</td>
</tr>
<tr>
<td>Fazekas grade on DWM (median [IQR])</td>
<td>1.5 (1–2)</td>
<td>1 (0–2)</td>
<td>0.142</td>
</tr>
<tr>
<td>Fazekas grade on PVWM (median [IQR])</td>
<td>1 (1–2)</td>
<td>1 (1–1)</td>
<td>0.229</td>
</tr>
<tr>
<td>Clinical assessments</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIHSS score at admission (median [IQR])</td>
<td>4 (2–6)</td>
<td>4 (2–6)</td>
<td>0.835</td>
</tr>
<tr>
<td>Patients with neurological progression, %</td>
<td>1 (5.0)</td>
<td>2 (10.5)</td>
<td>0.605</td>
</tr>
<tr>
<td>3-month mRS (median [IQR])</td>
<td>1 (0–1)</td>
<td>1 (0–1)</td>
<td>0.923</td>
</tr>
</tbody>
</table>

**DWM** indicates deep white matter; **mRS**, modified Rankin Scale; **MCA**, middle cerebral artery; **NIHSS**, National Institutes of Health Stroke Scale; **PVWM**, periventricular white matter; and **IQR**, interquartile range.

Stroke Scale (NIHSS) score. Neurological progression was defined as ≥2-point increase in NIHSS score that was not attributed to other conditions, such as infection, pain, or other serious medical comorbidities. Clinical outcomes were assessed using the modified Rankin Scale (mRS), and evaluations were performed 3 months after stroke onset using telephone or in-person interviews. This study was approved by the institutional review board of our institution, and informed consent was obtained from each patient.

### Data Analysis

The Fishers exact test or χ² test was performed to assess the categorical variables. Differences in continuous variables were evaluated using the Student t test, and ANOVA was used to assess age, lesion volume, and diameter. The Mann–Whitney U test was used to assess NIHSS, Fazekas Grading Scale, and mRS scores. Statistical analyses were conducted using SPSS for Windows (version 18.0). A 2-sided P value of <0.05 was considered statistically significant.
Table 3. Patient Characteristics According to the Location of MCA Plaques

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Superior Plaque (n=8)</th>
<th>Inferior Plaque (n=12)</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men, %</td>
<td>5 (62.5)</td>
<td>5 (41.7)</td>
<td>0.650</td>
</tr>
<tr>
<td>Age, y (mean±SD)</td>
<td>66.2±8.7</td>
<td>65.4±12.1</td>
<td>0.869</td>
</tr>
<tr>
<td>Vascular risk factors, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>3 (37.5)</td>
<td>8 (66.7)</td>
<td>0.362</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (25.0)</td>
<td>2 (16.7)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>2 (25.0)</td>
<td>2 (16.7)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>0</td>
<td>0</td>
<td>...</td>
</tr>
<tr>
<td>Current smoker</td>
<td>0</td>
<td>1 (8.3)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Previous history of stroke</td>
<td>2 (25.0)</td>
<td>1 (8.3)</td>
<td>0.537</td>
</tr>
<tr>
<td>MR findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal lesion, %</td>
<td>6 (75.0)</td>
<td>2 (16.7)</td>
<td>0.019</td>
</tr>
<tr>
<td>Large infarcts, %</td>
<td>5 (62.5)</td>
<td>3 (25.0)</td>
<td>0.167</td>
</tr>
<tr>
<td>Initial diameter, mm (mean±SD)</td>
<td>1.42±0.52</td>
<td>1.51±0.53</td>
<td>0.733</td>
</tr>
<tr>
<td>Follow-up diameter, mm (mean±SD)</td>
<td>1.88±0.64</td>
<td>1.76±0.59</td>
<td>0.680</td>
</tr>
<tr>
<td>Initial volume, mm(^3) (mean±SD)</td>
<td>1.27±0.78</td>
<td>1.21±0.86</td>
<td>0.877</td>
</tr>
<tr>
<td>Follow-up volume, mm(^3) (mean±SD)</td>
<td>2.59±2.22</td>
<td>2.61±1.89</td>
<td>0.655</td>
</tr>
<tr>
<td>(Follow-up)-initial volume, mm(^3) (mean±SD)</td>
<td>0.32±1.53</td>
<td>0.97±1.22</td>
<td>0.577</td>
</tr>
<tr>
<td>Concomitant cerebral atherosclerosis, %</td>
<td>5 (62.5)</td>
<td>4 (33.3)</td>
<td>0.362</td>
</tr>
<tr>
<td>Presence of microbleeds, %</td>
<td>1 (12.5)</td>
<td>7 (58.3)</td>
<td>0.070</td>
</tr>
<tr>
<td>Fazekas grade on DWM (median [IQR])</td>
<td>1.5 (0.25–2)</td>
<td>1.5 (1–2)</td>
<td>0.513</td>
</tr>
<tr>
<td>Fazekas grade on PVWM (median [IQR])</td>
<td>1 (0.25–2)</td>
<td>1 (1–2)</td>
<td>0.567</td>
</tr>
<tr>
<td>Clinical assessments</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIHSS score at admission (median [IQR])</td>
<td>4.5 (1.5–6.75)</td>
<td>3.5 (2–5.75)</td>
<td>0.613</td>
</tr>
<tr>
<td>Patients with neurological progression, %</td>
<td>0</td>
<td>1 (8.3)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>3-month mRS (median [IQR])</td>
<td>1 (0–1.75)</td>
<td>0</td>
<td>0.028</td>
</tr>
</tbody>
</table>

DWM indicates deep white matter; MCA, middle cerebral artery; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; PVWM, periventricular white matter; and IQR, interquartile range.

Results

Patient Characteristics

We enrolled 39 patients during the course of the study period, including 20 patients with pSSI and 19 with dSSI. Nineteen (48.7%) were men, and the mean age (±SD) of the study participants was 63.4 (±10.6) years. The identified vascular risk factors included hypertension (23 patients; 59.0%), diabetes mellitus (6 patients; 15.4%), hyperlipidemia (7 patients; 17.9%), coronary artery disease (2 patients; 5.1%), cigarette smoking (6 patients; 15.4%), and previous history of stroke (7 patients; 17.9%). MCA plaques were detected in 20 (51.3%) patients and, of these, 8 (40%) patients had superiorly located plaques. Fifteen (38.5%) patients had concomitant cerebral artery atherosclerosis. Cerebral microbleeds were observed in 11 (28.2%) patients, and the median (interquartile range) scores on the Fazekas scales for deep and periventricular white matter were 1.5 (1–2) and 1 (1–2), respectively. The median (interquartile range) NIHSS score was 4 (2–6), and neurological progression was observed in 3 (7.7%) patients. The median (interquartile range) mRS at 3 months was 0 (0–1).

Comparison of pSSI and dSSI

As shown in Table 1, the demographic characteristics and vascular risk factors did not differ between the groups. The frequency of large infarcts was higher (17 [85.0%] versus 1 [5.3%]; \(P<0.001\)), initial lesion volume was bigger (1.96±1.18 versus 1.11±1.11; \(P=0.025\)), the prevalence of microbleeds was lower (1 [5%] versus 10 [52.6%]; \(P=0.001\)), NIHSS scores on admission were higher (5 [3–6.75] versus 3 [1–3]; \(P=0.017\)), and mRS scores at 3 months tended to be higher (0.5 [0–1] versus 0 [0–0]; \(P=0.064\)) in patients with pSSI than in patients with dSSI, respectively. The prevalence of HR-MRI–identified MCA plaques did not differ between patients with pSSI (8 patients; 40%) and those with dSSI (12 patients; 63.2%; \(P=0.205\)). However, among patients with plaques, the prevalence of superiorly located plaques was higher in patients with pSSI (6 patients; 75.0%) than in those with dSSI (2 patients; 16.7%; \(P=0.019\)).

Characteristics of the Patients With and Without HR-MRI–Identified Atherosclerotic Plaques

There were no significant differences in terms of the demographic characteristics, vascular risk factors, lesion patterns, or clinical findings between patients with and without plaques (Table 2). However, among the patients who had MCA plaques, mRS scores at 3 months (1 [0–1.75] versus 0 [0–0]; \(P=0.028\)) and the proportion of patients having proximal lesion (6 [75%] versus 2 [16.7%]; \(P=0.019\)) were significantly higher in patients with superior plaques than in those with inferior plaques (Table 3).

Sensitivity and Specificity of MCA Plaque Prediction

To predict the presence of MCA plaques, sensitivity and specificity were calculated using the variables that are possibly associated with MCA plaques, such as lesion location, lesion size, and volume. Sensitivity and specificity values were as follows: lesion location (pSSI), 40% and 36.8%, respectively; lesion size (large lesion), 44.4% and 42.9%, respectively; lesion volume (>1.54 mm\(^3\) [mean value of the measured lesions]), 30.8% and 42.3%, respectively; and maximum lesion diameter (>1.5 cm), 50% and 47.4%, respectively (Figure 3). Next, we calculated the sensitivity and specificity of predicting superiorly located MCA plaques. Although the sensitivity (sensitivity 2: 30%, 42.2%, 37.5%, and 25.0%) was still low, the specificity (specificity 2: 89.5%, 85.9%, 90.1%, and 87.0%) was found to be higher than specificity 1.

Discussion

We classified SSI as either pSSI or dSSI, and found that patients with dSSI significantly have more microbleeds and tend to demonstrate more intense ischemic changes in the
white matter. These results are consistent with previous studies reporting that dSSI is more often associated with the characteristics of small-vessel disease than pSSI. In addition, we found that as many as 51.3% of patients with SSI demonstrated HR-MRI–identified MCA plaques, even if the patient’s MRA findings were normal. This result agrees with recent studies reporting that MCA plaques are present in 42% to 60% of patients with lacunar infarction. Unexpectedly, however, the frequencies of MCA plaques were not different between patients with pSSI and dSSI. This result may be in accordance with a previous study that reported that the prevalence of basilar artery plaques was not different between lesions associated with branch atheromatous disease or lesions associated with small-vessel disease in patients with pontine infarction. Nevertheless, we observed that superiorly located plaques were significantly more frequently observed in patients with pSSI than in those with dSSI.

It has been reported that perforators usually branch out from the superior portion of the MCA. Moreover, according to a recent study that investigated patients with symptomatic and asymptomatic MCA stenosis, symptomatic MCA stenosis was closely associated with superiorly located atherosclerotic plaques, although plaques were generally more prevalent on the inferior side of the MCA wall. We have also observed that HR-MRI–identified plaques were found on the superior portion of the MCA in 7 of 8 SSI patients with symptomatic MCA stenosis (unpublished observation). Taken together, it seems that superiorly located plaques are associated with pSSI, whereas inferiorly located plaques are more often incidental findings that are not associated with infarction. Therefore, our results suggest that the location, rather than the simple presence, of plaques is important for determining the location of SSI.

It was also surprising to see that there were no differences between patients with HR-MRI–identified plaques and those without identified plaques in terms of the markers of atherosclerosis and small-vessel disease, whereas differences between pSSI and dSSI were obvious. This apparent discrepancy could be explained by the possibly confounding effects of the presence of more significant atherosclerotic characteristics of plaque-negative pSSI in comparison with plaque-negative dSSI. In a previous study, patients with pSSI more often demonstrated markers of atherosclerosis than those with dSSI, although HR-MRI was not performed in that study.

Here, we found that characteristics, such as lesion size and location, demonstrate relatively low sensitivity and specificity for the prediction of MCA plaques. This may be related to (1) as discussed above, the fact that some plaques, especially inferiorly located plaques, may be incidental and unrelated to the infarction; (2) the branching patterns of MCA perforators vary greatly in terms of type, and infarction size and location may also be related to these variations; and (3) although most perforating arteries arise from the main trunk of the MCA, some originate from the superior or inferior branches. In this study, we could not examine the M2 branches because the HR-MRI protocol used in this study was focused on the main trunk of the MCA. Nevertheless, we found that specificity was significantly increased when we limited our analysis to only superiorly located plaques, which again supports the strong association between superiorly located plaques and infarction location and size.

Finally, we found that pSSI was associated with large infarction size, severe initial neurological status, and marginally higher mRS scores at 3 months than was dSSI. Interestingly, functional outcomes at 3 months were significantly worse in patients with superior plaques than those with inferior plaques.
plaques, regardless of infarction location. Perhaps, a plaque that is located close to the orifice of the branching arteries gives rise to a microembolism or hypoperfusion in the relevant subcortical tissue, leading to unfavorable clinical outcomes.10

Our study has a merit in that consecutive patients with SSI were prospectively enrolled and thoroughly assessed by DWI, MR angiogram, and HR-MRI. The limitations of this study were the number of patients was relatively small and the M2 portion of the MCA was not examined by HR-MRI. Nevertheless, our results illustrate that the superiorly located plaques are associated with pSSI and that pSSI is closely associated with large lesions, and unfavorable clinical manifestations as compared with dSSI. Therefore, the identification of atherosclerotic plaques using HR-MRI may help clinicians to assess stroke mechanism, patterns, and clinical outcomes. Further studies with a larger number of patients are needed to confirm our findings.

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Disclosures
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References
Single Subcortical Infarction and Atherosclerotic Plaques in the Middle Cerebral Artery: High-Resolution Magnetic Resonance Imaging Findings
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