**Atherosclerotic middle cerebral artery plaque and subsequent stroke: a high-resolution MRI study**

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Aims and objectives

To assess the reproducibility of HR-MRI for the identification of MCA atherosclerotic plaque components and quantification of stenosis;

To compare the plaque characterization and vessel wall properties between symptomatic and asymptomatic atherosclerotic MCA using HR-MRI.
Methods and materials

Subjects

High-resolution MRI in atherosclerotic stenosis of the MCA was partial of an ongoing multicenter prospective study analyzing the multimoding diagnosis of acute ischemic stroke and assessing the prognostic value. Patients with the following features were included: (1) MCA stenosis #50% at the M1 segment on conventional angiography; (2) without any other major cause of stroke; and (3) #2 risk factors (hypertension, diabetes mellitus, hyperlipidemia, cigarette smoking, and obesity) for atherosclerotic disease. Patients were considered symptomatic if they had an ischemic stroke or transient ischemic attack in the territory of the stenotic MCA within the previous 30 days. Patients were considered asymptomatic if they did not have a history of cerebrovascular events or if an ischemic event occurred in a vascular distribution outside the stenotic MCA. The exclusion criteria were: (1) evidence of cardioembolism; (2) complete MCA occlusion; (3) arteritis; (4) dissection; (5) after intra-arterial or intravenous thrombolysis; and (6) poor image quality secondary to motion artifacts. The study was approved by the local Medical Ethics Committee and all patients signed informed consent forms.

Magnetic Resonance Imaging Protocol

All subjects were imaged with a 3.0 T MRI Scanner (Signa Excite HD, GE Healthcare, Milwaukee, WI, United States), with a peak gradient strength of 50 mT/m, using a standard 8-channel head coil. A standardized stroke MR protocol was performed including a routine MRI (T1WI, T2WI, and DWI) and three-dimensional (3D) time-of-flight (TOF) MRA. The lesion site on the MCA M1 segment for HR-MRI scanning was determined by a neuroradiologist, after reviewing both the MRA and traditional MR images. The HR-MRI protocol included three sequences. First, pre- and post-contrast T1WI image scanning used a double inversion recovery measurement of the black blood two-dimensional (2D) fast spin echo sequence parameters: TR equal to two heart beats (1200 to 2000 ms, depending on heart rate), TI/TE 3000/50 ms, echo train length (ETL) 22, band width 14.7, and number of average (NEX) 4. Next, the high resolution T2WI image scanning was acquired also using fast-spin echo sequence parameters: TR/TE 3000/50 ms, ETL 50, band width 62.5, and NEX 8. Both acquisitions were run with a field of view of 13 cm, matrix size of 256, slice thickness of 2 mm, and spacing of 0.2 mm. The scan direction was parallel or perpendicular to the M1 segment. Three minutes later, for the post-contrast T1-weighted imaging, the contrast agent (gadopentetate dimeglumine, Gd-DTPA, Magnevist; Bayer Schering Pharma, Berlin, Germany) was injected at an intravenous dose of 0.1 mmol/kg (flow rate: 2 mL/s).

Image Review

All images were reviewed by two experienced neuroradiologists independently blinded to the patient’s clinical findings and history on a digital picture archiving and communication
(PACS) workstation. The quality of each image from the four sequences (3D-TOF, T2WI and pre-and post-contrast T1WI) for every subject was graded on a three-point scale according to the method described by Ryu et al.[9] based on the conspicuity of the vessel margins, lumen and the wall architecture. The grades were: (1) grade1 was that outer boundary of artery and lumen couldn’t be identifiable; (2) Grade 2 was that outer boundary and/or lumen was partially obscured; and Grade 3 was that wall architecture depicted in detail and outer boundary and lumen could be clearly identified [9]. Subjects in which image quality was grade 1 were excluded from the study. The MR images of the qualifying MCA artery were assessed using a unified form and published criteria [2]. Plaques were defined if there were markedly eccentric or focal wall thickenings, at which the thickest point of the wall was estimated to be > 200% the thinnest part on visual inspection of T2-weighted images [6, 7]. Wall thickening was identified if the wall was abnormal on inspection but did not reach the criteria that defined plaque.

**Tissue Components (qualitative assessment)**

The variety of tissue components was identified based on previous postmortem MCA, carotid artery, and coronary artery pathological control studies [2, 11]. Both recent and fresh intra-plaque hemorrhages were identified as hyperintense areas on T1WI and 3D-TOF images (> 150% of the signals of adjacent gray matter). The fibrous cap appeared as a bright band adjacent to the lumen in MCA atherosclerosis on T2W images. Both intra-plaque hemorrhages and fibrous cap were considered present if they were observed on at least one imaging slice. All plaque signal intensities were compared with the adjacent brain parenchymal gray matter on each sequence. The degree of plaque enhancement was evaluated qualitatively via comparison of the pre- and post-contrast cross-sectional T1W images.

**Quantitative Analysis**

The quantitative measurements were performed at the narrowest lumen location on cross-sectional HR-MRI T2W images, including vessel area, lumen area, reference vessel area, and the wall area. In order to calculate the cross-sectional area of the vessel and lumen, two regions of interest of the outer vessel wall boundary and the lumen were manually drawn in the same slice (Fig 1). The outer vessel wall boundary was traced along the interface between the vessel wall and meninges (or cerebrospinal fluid). The wall area was calculated by subtracting the lumen area from the outer-wall boundary area. The degree of MCA stenosis on HR-MRI was stated as the following: degree of stenosis = (1#narrow lumen area/reference lumen area) × 100% [12]. The reference MCA segment site was selected, with the nonoccluded lumen preferably close to the stenotic segment. If a proximal reference site was also abnormal, the neighboring distal site could be used instead.

All the qualitative assessment and quantitative measurement data from both readers were used to calculate the inter-observer reproducibility. To assess intra-observer reproducibility, reader 1 reevaluated all images which were presented in a different
order four weeks after the first reading session. For further comparison of the plaque characterization and vessel wall properties between the symptom group and asymptom group, the measurement was averaged and the tissue component differences between the first set of reader 1 and reader 2 were solved by consensus.

**Statistical Analysis**

Statistical analysis was performed with SPSS 13.0 statistical software. Agreements were assessed by calculating kappa (κ) for dichotomous data and intraclass correlation coefficients (ICC) with a one-way random effect for intra-observer continuous variables and a two-way random effect for inter-observer continuous variables. All agreement parameters had calculated 95% confidence intervals (CIs). Values were graded according to the method proposed by Landis and Koch (<0.0 = poor agreement; 0.0-0.2 = slight agreement; 0.21-0.40 = fair agreement; 0.41-0.60 = moderate agreement; 0.61-0.80 = substantial agreement; 0.81-1.0 = almost perfect) [13]. Intraclass correlation coefficient values that were >0.75 were considered excellent agreement [14]. Besides, we analyzed the level of agreement by plotting the differences between the two area measurements against the averages of the two area measurements according to the method described by Bland and Altman (i.e. Bland-Altman plots) [15]. Finally, qualitative data comparison was conducted with Chi-square test. A t-test was used to quantitative data comparison. A two-sided level of 0.05 was used to infer the statistically significant difference.
Results

Between March 2010 to February 2013, seventy-three subjects (46 males and 27 females) were enrolled in the study. Median age of the subjects was 64 years (range: 30-84 years). Among them 38 had a stroke, 3 had a transient ischemic attack and 32 were asymptomatic. The median time from stroke onset to MRI examination in symptomatic patients was 10 days (range, 4-17).

A total of 65 plaques were found on HR-MRI. Plaques were found in each of the 41 symptomatic cases (41/41) and 24 of the asymptomatic cases (24/32). These were displayed as either focal or eccentric wall thickening with homogenous (28 cases) or heterogeneous (37 cases) intensity. Within the 65 plaques, 11 hemorrhage cases (Fig 1) and 55 fibrous cap cases (Fig 2) were observed. The intra-observer reproducibility was substantial for the fibrous cap (#=0.65) and for intra-plaque hemorrhage (#=0.79); inter-observer reproducibility was moderate for the fibrous cap (#=0.58), and substantial for intra-plaque hemorrhage (#=0.68) (Table 1).

Table 1. Intra-observer and inter-observer reproducibility

<table>
<thead>
<tr>
<th>Plaque Components</th>
<th>Intra-observer</th>
<th>Inter-observer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Agreement</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>97%</td>
<td>0.79 (0.57-0.96)</td>
</tr>
<tr>
<td></td>
<td>84%</td>
<td>0.65 (0.42-0.86)</td>
</tr>
<tr>
<td></td>
<td>92%</td>
<td>0.89 (0.78-0.95)</td>
</tr>
<tr>
<td>Quantitative Measurements</td>
<td>Intraclass Coefficient Correlation (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Vessel area</td>
<td>0.95 (0.92 to 0.97)</td>
<td>0.87 (0.78 to 0.93)</td>
</tr>
<tr>
<td>Lumen area</td>
<td>0.97 (0.95 to 0.98)</td>
<td>0.96 (0.94 to 0.98)</td>
</tr>
<tr>
<td>Wall area</td>
<td>0.96 (0.35 to 0.98)</td>
<td>0.91 (0.85 to 0.94)</td>
</tr>
<tr>
<td>Reference Vessel area</td>
<td>0.91 (0.85 to 0.94)</td>
<td>0.90 (0.84 to 0.94)</td>
</tr>
</tbody>
</table>

Seven (10.8%) patients didn’t receive T1 contrast enhancement images after performing plain scan because of kidney failure (n=3) or too bad condition to continue the
Finally, there were 58/65 patients who received T1 contrast enhancement images; 40 were from symptomatic cases and 18 were from asymptomatic cases. Focal, irregular, and eccentric vessel wall enhancement was present in 43 of the 58 patients with atherosclerotic disease (Fig 2); 39 patients had enhancement only in the vessel supplying the acute ischemic injury area, and four patients did not have evidence of ischemic injury in the territory supplied by the vessels. Intra-observer agreement was almost perfect ($\kappa = 0.89$) and inter-observer agreement was substantial ($\kappa = 0.80$) for the identification of contrast enhancement (Table 1).

The frequency of intra-plaque hemorrhage and contrast enhancement in symptomatic group was significantly higher than that in the asymptomatic group. However, the difference in the frequency of fibrous cap between two groups did not reach statistical significance (Table 2). Intra-observer and inter-observer reproducibility for quantitative area measurements were excellent, with the ICC ranging from 0.91 to 0.97 and 0.87 to 0.96, respectively (Table 1). Bland-Altman plots showed small absolute differences in intra-observer (Fig 3) and inter-observer (Fig 4) measurements. However, for the vessel and wall area measurements, the Bland-Altman plots showed a wide interval compared with the mean. The lumen area, wall area and degree of stenosis in the symptomatic group were significantly higher than that in the asymptomatic group. However, there was no significant difference of vessel area between two groups (Table 2).

### Table 2. Morphology analysis of MCA atherosclerotic plaque components and stenosis

<table>
<thead>
<tr>
<th></th>
<th>Symptomatic MCA stenosis (n=41)</th>
<th>Asymptomatic MCA stenosis (n=32)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plaque</strong></td>
<td>41</td>
<td>24</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Hemorrhage</strong></td>
<td>11</td>
<td>0</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>Fibrous cap</strong></td>
<td>37</td>
<td>18</td>
<td>0.154</td>
</tr>
<tr>
<td><strong>Enhancement</strong></td>
<td>39(39/40)*</td>
<td>4(4/18)*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Vessel area</strong></td>
<td>8.64±2.59 mm$^2$</td>
<td>7.88±2.07 mm$^2$</td>
<td>0.206</td>
</tr>
<tr>
<td><strong>Lumen area</strong></td>
<td>2.86±2.18 mm$^2$</td>
<td>4.67±2.42 mm$^2$</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>Wall area</strong></td>
<td>5.78±3.12 mm$^2$</td>
<td>3.21±2.93 mm$^2$</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Reference Vessel area</strong></td>
<td>6.59±2.25 mm$^2$</td>
<td>7.43±2.47 mm$^2$</td>
<td>0.159</td>
</tr>
<tr>
<td><strong>Degree of stenosis</strong></td>
<td>55.86±27.40%</td>
<td>38.52±21.21%</td>
<td>0.005</td>
</tr>
</tbody>
</table>
40 patients received T1 contrast enhancement images in symptomatic group, while 18 patients in asymptomatic group.
**Fig. 1:** Visualization of intra-plaque hemorrhage and quantitative measurement of the lumen for symptomatic middle cerebral artery stenosis using HR-MRI. A 56-year-old male patient with infarction in right basal ganglia. (A) Sagittal 3D-TOF showed a plaque with heterogenous signal intensity (arrow).

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Fig. 2: (B) Cross-sectional T1W image showed a plaque with heterogeneous signal intensity (arrow).

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Fig. 3: (C) Cross-sectional T2W image showed a plaque with heterogeneous signal intensity (arrow).

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Fig. 4: (D) Coronal (parallel to the M1 segment) T1W image showed a plaque with heterogeneous signal intensity (arrow).

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Fig. 5: Quantitative measurement of the vessel and lumen. (E) Two regions of interest of the outer vessel wall boundary and the lumen were manually drawn in the same slice.

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Fig. 6: (F)Coronal projection of MRA showed stenosis in the right MCA (arrow head).
Fig. 7: Eccentric narrowing and enhancement of symptomatic middle cerebral artery stenosis. A 78-year-old female patient with infarction in right corona radiata and cortical. (A)MRA showed severe narrowing of the right M1 segment (arrow head).

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Fig. 8: (B) Multiple infarcts were observed in the distribution of stenotic right MCA on DWI.
**Fig. 9:** (C) T2WI showed wall abnormality (arrow).

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Fig. 10: (D) non-enhanced T1WI HR-MR image showed wall abnormality (arrow).

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**Fig. 11:** (E) Contrast-enhanced T1W image in cross-section showed eccentric right MCA enhancement, indicating an active atherosclerotic plaque (arrows).

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Fig. 12: (F) Normal wall as a reference site.

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Fig. 13: Figure 3. Bland-Altman plots of intra-observer reproducibility for vessel area at the level of the M1 segment.

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**Fig. 14:** Figure 3. Bland-Altman plots of intra-observer reproducibility for lumen area (B) at the level of the M1 segment.

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Fig. 15: Bland-Altman plots of intra-observer reproducibility for wall area (C) at the level of the M1 segment.

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Fig. 16: Figure 3. Bland-Altman plots of intra-observer reproducibility for reference vessel area (D) at the level of the M1 segment.

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Fig. 17: Bland-Altman plots of inter-observer reproducibility for vessel area (A) at the level of the M1 segment.

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Fig. 19: Bland-Altman plots of inter-observer reproducibility for wall area (C) at the level of the M1 segment.

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Fig. 20: Figure 4. Bland-Altman plots of inter-observer reproducibility for reference vessel area (D) at the level of the M1 segment.

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Fig. 18: Bland-Altman plots of inter-observer reproducibility for lumen area (B) at the level of the M1 segment.

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Conclusion

Feasibility and reproducibility of HR-MRI for identifying MCA atherosclerotic plaque components is generally acceptable. Plaque characterization and vessel wall properties on HR-MRI were different between symptomatic and asymptomatic MCA stenosis. The presence of vulnerable plaque is closely related to stroke. Hence, HR-MRI may provide a useful tool for clinical risk evaluation in MCA atherosclerosis.
Personal information

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References


