MRI of Plaque Characteristics and Relationship With Downstream Perfusion and Cerebral Infarction in Patients With Symptomatic Middle Cerebral Artery Stenosis

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Background: Intracranial plaque characteristics are associated with stroke events. Differences in plaque features may explain the disconnect between stenosis severity and the presence of ischemic stroke.

Purpose: To investigate the relationship between plaque characteristics and downstream perfusion changes, and their contribution to the occurrence of cerebral infarction beyond luminal stenosis.

Study Type: Case control.

Subjects: Forty-six patients with symptomatic middle cerebral artery (MCA) stenosis (with acute cerebral infarction, n = 30; without acute cerebral infarction, n = 16).

Field Strength/Sequence: 3.0T with 3D turbo spin echo sequence (3D-SPACE).

Assessment: Luminal stenosis grade, plaque features including lesion T2 and T1 hyperintense components, plaque enhancement grade, and plaque distribution were assessed. Brain perfusion was evaluated on mean transient time maps based on the Alberta Stroke Program Early CT score (MTT-ASPECTS).

Statistical Tests: Plaque features, grade of luminal stenosis, and MTT-ASPECTS were compared between two groups. The association between plaque features and MTT-ASPECTS were assessed using Spearman’s correlation analysis. Multivariate logistic regression and receiver operating characteristic (ROC) curves were constructed to assess the effect of significant variables alone and their combination in determining the occurrence of cerebral infarction.

Results: Stronger enhanced plaques were associated with downstream lower MTT-ASPECTS (P = 0.010). Plaque enhancement grade (P = 0.039, odds ratio [OR] 5.9, 95% confidence interval [CI] 1.1–32) and MTT-ASPECTS (P = 0.003, OR 2.6, 95% CI 1.4–4.7) were associated with a recent cerebral infarction, whereas luminal stenosis grade was not (P = 0.128). The combination of MTT-ASPECTS and plaque enhancement grade provided incremental information beyond luminal stenosis grade alone. The area under the receiver operating characteristic curve (AUC) improved from 0.535 to 0.921 (P < 0.05).

Data Concussion: Strongly enhanced plaques are associated with a higher likelihood of downstream perfusion impairment. Plaque enhancement and perfusion evaluation may play a complementary role to luminal stenosis in determining the occurrence of acute cerebral infarction.

Level of Evidence: 4
Technical Efficacy: Stage 2

Intracranial atherosclerosis (ICAS) is a major cause of ischemic stroke, accounting for about 10% and 30–50% of strokes in Western and Asian populations, respectively.1,2 Patients with ICAS have increased risk of subsequent stroke. The overall annual stroke risk in patients with symptomatic middle cerebral artery stenosis is 12.5%, whereas the annual

View this article online at wileyonlinelibrary.com. DOI: 10.1002/jmri.25879
Received Aug 23, 2017, Accepted for publication Oct 10, 2017.

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incidence in asymptomatic MCA stenosis is only 2.8%\textsuperscript{3,4}. Severe stenosis was traditionally considered to be an indicative of cerebral ischemia. However, it is increasingly recognized that the disconnect between stenosis severity and the presence of ischemic stroke is common.\textsuperscript{5–7} Recent studies using high-resolution intracranial vessel wall imaging (HRVWI) have shown that atherosclerotic plaque characteristics, such as plaque enhancement and positive remodeling, are potential high-risk biomarkers for ischemic stroke.\textsuperscript{6,8–11} Therefore, plaque characteristics may be a link between stenosis and ischemia.

Possible stroke mechanisms arising from ICAS includes branch occlusion, in situ thrombotic occlusion, artery-to-artery embolism, and hyperperfusion.\textsuperscript{12,13} However, these mechanisms often coexist and their pathophysiological features are interactive. A vulnerable atherosclerotic plaque tends to produce plaque fissuring resulting in embolism to distal arteries, and may also lead to hypoperfused status in the distal area.\textsuperscript{12,13} Details on the correlation between intracranial vessel wall pathology and brain perfusion impairment remain elusive. Investigation of the relationship between plaque characteristics and downstream perfusion may facilitate understanding of the mechanisms underlying acute ischemic events in ICAS patients.

Dynamic susceptibility contrast-enhanced perfusion weighted imaging (DSC-PWI) is a well-established technique for estimating the impairment of brain perfusion in daily practice. Parametric maps such as mean transit time (MTT) maps have been used for identifying the hypoperfusion areas.\textsuperscript{14} In the present study, we sought to investigate the following: 1) the association between plaque characteristics and changes of downstream perfusion on MTT maps in patients with symptomatic MCA stenosis; 2) whether plaque characteristics and perfusion evaluation on MTT maps could provide incremental contribution to the occurrence of acute cerebral infarction beyond luminal stenosis severity.

**Materials and Methods**

**Patient Population**

This study was studied and approved by the Institutional Review Board. We reviewed our single-institutional HRVWI database for patients with symptomatic MCA stenosis (>50%) detected by magnetic resonance angiography (MRA) between May 2015 and February 2017. Forty-six patients were recruited based on the following inclusion criteria: 1) symptomatic patients who suffered from a recent transient ischemic attack (TIA) or cerebral infarction in the territory of the stenosed MCA, based on neurological examination and diffusion-weighted imaging (DWI) within the previous week; 2) significant MCA stenosis (>50%) calculated on 3D black blood sequence, and absence of significant ipsilateral internal carotid stenosis (>30%) as assessed by ultrasound; 3) no clinical history of atrial fibrillation; 4) ≥1 atherosclerotic risk factors, including hypertension, diabetes mellitus, hypercholesterolemia, and current cigarette smoking; and 5) image quality sufficient for plaque characteristics and brain perfusion evaluation. Exclusion criteria included: i) nonatherosclerotic vasculopathy, such as dissection, vasculitis, or Moyamoya disease; ii) high-risk factors for cardioembolism, such as atrial fibrillation, mechanical prosthesis valve disease, dilated cardiomyopathy; iii) acute infarcts <2 cm in diameter (defined as lacunae). They were excluded since they are generally thought to have resulted from occlusion of penetrating branches of large cerebral arteries.\textsuperscript{15,16} Based on DWI, symptomatic patients were further divided into two subgroups: 1) patients with acute cerebral infarction (ACI); 2) patients without acute cerebral infarction (NACI).

**Clinical Information**

Patient demographic features and risk factors were defined as follows: 1) hypertension: blood pressure >140/90 mmHg on repeated measurements, or patients receiving medication for hypertension; 2) hyperlipidemia: cholesterol level >200 mg/dL or low-density lipoprotein ≥130 mg/dL; 3) diabetes mellitus: fasting blood sugar ≥7.0 mmol/L, or 2-hour postprandial blood sugar ≥11.1 mmol/L; 4) current smoker.

**MRI Protocol**

All images were acquired with a 3.0T MR (Magnetom Verio or Skyra; Siemens, Erlangen, Germany) equipped with a 32-channel or a 16-channel head-matrix coil. Three-dimensional time-of-flight (3D-TOF) MRA was performed for positioning using the following parameters: repetition time / echo time (TR/TE), 22/3.6 msec; flip angle, 18º; field of view (FOV), 210 × 190 mm; acquired resolution, 0.55 × 0.55 × 0.55 mm; acquisition time, 4.4 minutes. A 3D black blood sequence was performed using a proton-density weighted (PD) or T\textsubscript{1}-weighted (T\textsubscript{1}w) SPACE (Sampling Perfection with Application optimized Contrast using different angle Evolutions) before and after contrast administration. The parameters for PD SPACE (on Verio) were TR/TE, 1500/24.0 msec; FOV, 160 × 130 mm; turbo-spin factor, 77 echoes; echo spacing 4.48 msec; acquired resolution, 0.60 × 0.60 × 0.60 mm; acquisition time, 5.3 minutes. The parameters for T\textsubscript{1} SPACE (on Skyra) were TR/TE, 900/4.2 msec; FOV, 240 × 216 mm; turbo-spin factor, 43 echoes; echo spacing 4.2 msec; acquired resolution, 0.75 × 0.75 × 0.75 mm; reconstruction resolution, 0.4 × 0.4 × 0.75 mm; acquisition time, 7 minutes. Twelve sections of two-dimensional black blood (2D-BB) turbo spin-echo T\textsubscript{2}-weighted (T\textsubscript{2}w) and T\textsubscript{1} images were acquired in the sagittal plane. The details of the protocols were as follows: 2D-BB T\textsubscript{2}, TR/TE, 2400/50 msec; FOV, 130 × 130 mm; matrix, 320 × 320; acquired in-plane resolution, 0.40 × 0.40 mm; slice thickness, 2.0 mm; acquisition time, 3.4 minutes; 2D-BB T\textsubscript{1}, TR/TE, 601/10 msec; FOV, 130 × 130 mm; matrix, 320 × 320; acquired in-plane resolution, 0.40 × 0.40 mm; slice thickness, 2.0 mm; acquisition time, 3.5 minutes.

Axial DWI was performed using a single-shot echo-planar spin-echo sequence. The parameters were as follows: b-value, 0 and 1000 mm\textsuperscript{2}/s, FOV, 230 × 230 mm, section thickness, 5 mm, matrix, 192 × 192; acquisition time, 1 minute. DSC-PWI was performed with a gradient-echo echo-planar imaging sequence. After the first six image acquisitions, a bolus of 0.1 mmol/kg gadodiamide (Omniscan, GE Healthcare, Cork, Ireland) was rapidly administered by an MRI power injector (Mississippi, Ulrich...
Medical, Ulm, Germany) at a rate of 4 mL/s, followed by 20 mL of saline at the same rate. A total of 50 dynamic phases were obtained. The detailed imaging parameters were as follows: TR/TE, 1500/30 msec, FOV, 230 × 230 mm, section thickness 5 mm, matrix, 128 × 128, acquisition time, 1.3 minutes. Contrast-enhanced 2D-BB T1 and 3D SPACE images were repeated immediately after DSC-PWI acquisition. The total acquisition times were 27.7 minutes (on Verio) and 31.1 minutes (on Skyra), respectively.

### Image Analysis

HRVWI images were analyzed on a workstation (Leonardo, Siemens) by two independent neuroradiologists (SS. L and CQ. S, each with 5 and 4 years of experience, respectively) who were blinded to the DWI results and clinical information. A culprit plaque was defined as the most stenotic lesion arising on the ipsilateral MCA to an ischemic stroke. The plaque characteristics evaluated in our study included the following: 1) lesion T2 hyperintense components: presence or absence of T2 hyperintense band. Lesion signal characteristics on T2 were assessed relative to gray matter; 2) lesion T1 hyperintense components: presence or absence of T1 hyperintense on unenhanced T1 images. Lesion signal characteristics on T1 were assessed relative to normal-appearing white matter; 3) plaque enhancement grade: grade 0, enhancement was less than or equal to that of intracranial arterial walls without plaque in the same patient; grade 1, enhancement was greater than that of grade 0 but less than that of the pituitary stalk; grade 2, enhancement was greater than or equal to that of pituitary stalk; and 4) plaque distribution: eccentric, <50% wall involvement; concentric, >50% wall involvement. The degree of MCA luminal stenosis was calculated in 3D SPACE using the following formula: stenosis grade = (1 - narrow lumen area/reference lumen area) × 100%. The reference lumen was defined as the MCA segment of normal appearance proximal to the stenotic site. If a proximal reference site was not available, the neighboring distal site was used instead. The measurement results of two neuroradiologists were averaged for subsequent analysis.

Perfusion data were computed using the singular value decomposition deconvolution method to create MTT maps, which are highly sensitive in identifying tissue at risk. The arterial input function was manually selected from the contralateral middle cerebral artery. The hypoperfused areas on the MTT maps were visually assessed by two independent neuroradiologists who was blinded to the other findings (SS. L and CQ. S, each with 5 and 4 years of experience, respectively) according to the Alberta Stroke Program Early CT score (ASPECTS) as previously described by Barber et al. Each ASPECTS region was scored 0 if abnormal and 1 if normal. These subscores were summed to compute final MTT-ASPECTS for each patient (ranging from 0 to 10). For any discrepancy between the two readers about plaque characteristics or MTT-ASPECTS, another neuroradiologist (XN. H with 20 years of experience) reevaluated the images and assisted in reaching a consensus agreement.

### Statistical Analysis

The interreader reproducibility for plaque characteristics and MTT-ASPECTS assessment was evaluated using kappa analysis. The interreader reproducibility for the measurement of luminal stenosis was evaluated using intraclass correlation coefficient (ICC). Reliabilities <0.4 were characterized as poor, those 0.4–0.75 were fair to good, and those >0.75 were considered excellent.

Variable plaque characteristics, luminal stenosis grade, and MTT-ASPECTS between the ACI and the NACI group were compared using an independent sample t test, or a chi-square test, or a nonparametric Mann–Whitney U-test as appropriate. Variables reaching significance between the ACI and the NACI group on univariate analysis and luminal stenosis grade were entered into the multivariate logistic regression model. Odds ratio (OR) with 95% confidence interval (CI) was calculated for each covariate. A receiver operating characteristic (ROC) curve was constructed to assess the diagnostic performance of each variable and their combinations to determine the occurrence of cerebral infarction. Statistical analyses were performed using SPSS (v. 13.0, Chicago, IL). The ROC curves were analyzed by MedCalc (v. 12.3.0, Mariakerke, Belgium). The P value was two-sided, and P < 0.05 was considered statistically significant.

### Results

#### Patient Demographics

The baseline patient demographics and cerebrovascular risk factors are presented in Table 1. No significant differences were observed between the ACI and the NACI group. Sixteen patients (34.8%) had normal DWI. Thirty patients (65.2%) had DWI-positive infarctions, with the majority

### TABLE 1. Patient Demographics

<table>
<thead>
<tr>
<th>Variables</th>
<th>ACI group (n = 30)</th>
<th>NACI group (n = 16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male, n (%)</td>
<td>21 (70.0%)</td>
<td>10 (62.5%)</td>
<td>0.605</td>
</tr>
<tr>
<td>Age, (Mean, range), y</td>
<td>57.1 ± 14.5</td>
<td>54.1 ± 16.5</td>
<td>0.522</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>23 (76.7%)</td>
<td>12 (75%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>9 (30.0%)</td>
<td>4 (25.5%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>10 (33.3%)</td>
<td>3 (18.8%)</td>
<td>0.493</td>
</tr>
</tbody>
</table>

ACI: patients with acute cerebral infarction; NACI: patients without acute cerebral infarction.
(n = 19) having predominantly subcortical infarcts, five having cortical infarcts with or without subcortical infarcts, and six having border-zone infarcts.

**Luminal Stenosis, Plaque Characteristics, and Brain Perfusion Between Groups**

The ICC for luminal stenosis grade assessment was 0.831. The MCA stenosis grade was 75.3 ± 12.5% and 72.8 ± 13.2% in the ACI group and NACI group, respectively. No significant difference in luminal stenosis grade between groups was observed (P = 0.538).

The interreader agreement for plaque characteristics was 0.828 for plaque enhancement grade, 0.809 for lesion T2 hyperintense components, 0.836 for lesion T1 hyperintense components, and 0.691 for plaque distribution. Forty-four of 46 (95.7%) plaques showed enhancement in all patients. A total of 24 of 30 (80.0%) and 6 of 16 (37.5%) plaques exhibited strong enhancement (grade 2) in the ACI and NACI group, respectively. Plaque enhancement grade in the ACI group was significantly greater than that in the NACI group (P = 0.005). Plaque enhancement grade was significantly correlated with the presence of downstream acute infarction (r = 0.446, P = 0.002). No significant differences were found in other plaque features, including T2 hyperintense components (P = 0.605), T1 hyperintense components (P = 0.498), and plaque distribution (P = 0.309), between the two groups.

The interreader agreement for MTT-ASPECTS was 0.874. The MTT-ASPECTS in the ACI group was 4.60 ± 2.58, which was significantly lower than that in the NACI group (8.19 ± 1.52) (P < 0.001). MTT-ASPECTS was negatively correlated with the presence of cerebral infarction (r = −0.634, P < 0.001). Table 2 shows the detailed differences in the degree of luminal stenosis, plaque characteristics, and MTT-ASPECTS between the ACI and the NACI groups.

**Relationship Between Plaque Characteristics and Brain Perfusion Changes**

The distribution of plaque enhancement grade in relation to MTT-ASPECTS is illustrated in Fig. 1. Plaques enhancement grade was inversely correlated with MTT-ASPECTS (r = −0.378, P = 0.010). The MTT-ASPECTS was 5.10 ± 2.86 in the group with strongly enhanced plaques, which was significantly lower than that with weakly or nonenhanced plaques (7.25 ± 2.27) (P = 0.014). Other plaque characteristics

![FIGURE 1: Distribution of MTT-ASPECTS in relation to plaque enhancement grade. Values shown in the bar are the number of patients in different MTT-ASPECTS groups. n = 46 patients.](image-url)

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**TABLE 2. Luminal Stenosis, Plaque Features and MTT-ASPECTS in ACI and NACI Patients**

<table>
<thead>
<tr>
<th>Variables</th>
<th>ACI group (n = 30)</th>
<th>NACI group (n = 16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenosis grade</td>
<td>75.3 ± 12.5%</td>
<td>72.8 ± 13.2%</td>
<td>0.538</td>
</tr>
<tr>
<td>Plaque enhancement grade</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 0</td>
<td>0 (0.0%)</td>
<td>2 (12.5%)</td>
<td></td>
</tr>
<tr>
<td>Grade 1</td>
<td>6 (26.7%)</td>
<td>8 (50.0%)</td>
<td></td>
</tr>
<tr>
<td>Grade 2</td>
<td>24 (73.3%)</td>
<td>6 (37.5%)</td>
<td></td>
</tr>
<tr>
<td>Plaque distribution</td>
<td></td>
<td></td>
<td>0.309</td>
</tr>
<tr>
<td>Eccentric</td>
<td>18 (60.0%)</td>
<td>12 (75.0%)</td>
<td></td>
</tr>
<tr>
<td>Concentric</td>
<td>12 (40.0%)</td>
<td>4 (25.0%)</td>
<td></td>
</tr>
<tr>
<td>Presence of T2 hyperintense</td>
<td>21 (70.0%)</td>
<td>10 (62.5%)</td>
<td>0.605</td>
</tr>
<tr>
<td>Presence of T1 hyperintense</td>
<td>9 (30.0%)</td>
<td>3 (18.8%)</td>
<td>0.498*</td>
</tr>
<tr>
<td>MTT-ASPECTS</td>
<td>4.60 ± 2.58</td>
<td>8.19 ± 1.52</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

ACI: Patients with acute cerebral infarction; NACI: Patients without acute cerebral infarction. Plaque enhancement grade: grade 0, enhancement was equal to or less than that of intracranial arterial walls without plaque in the same individual; grade 1, enhancement was greater than that of grade 0 but less than that of the pituitary stalk; grade 2, enhancement was similar to or greater than that of pituitary stalk. MTT-ASPECTS: Alberta Stroke Program Early CT score based on mean transit time maps.

*P value calculated from Fisher’s exact test.
including T₂ hyperintense components, T₁ hyperintense components and plaque distribution, were not significantly correlated with MTT-ASPECTS ($P = 0.152$, 0.576, and 0.132, respectively). Case examples demonstrating plaque characteristics and MTT-ASPECTS in the ACI and the NACI groups are shown in Figs. 2 and 3.

FIGURE 2: Acute cerebral infarction in a 43-year-old male patient. (A) On the 3D-TOF MRA, significant stenosis (78.5%) is demonstrated in the right M1 segment of the MCA (arrow). (B) Diffusion-weighted imaging shows acute cerebral infarction involving the cortical and subcortical white matter of the right temporal lobe. (C) Sagittal T₂ image showing an isointense plaque in the right middle cerebral artery. (D) Sagittal contrasted-enhanced T₁ image showing strong enhancement of the plaque (grade 2). (E–H) MTT maps showing a large hypoperfusion area, involving M1, M2, M3, M5, M6, and the insular ribbon according to the Alberta Stroke Program Early CT score. The MTT-ASPECTS is 4.

FIGURE 3: A 40-year-old male patient with a transient ischemic attack. (A) Significant stenosis (63.4%) is indicated in the left M1 segment of the MCA on the 3D-TOF MRA (arrow). (B) No positive lesion is found on diffusion-weighted imaging. (C) Sagittal T₂ image showing an isointense plaque in the left middle cerebral artery. (D) Sagittal contrasted-enhanced T₁ image showing no enhancement of the plaque (grade 0). (E–H) MTT maps showing a small hypoperfusion area, involving M3 and M6 according to the Alberta Stroke Program Early CT score. The MTT-ASPECTS is 8.
Combined Assessment of Plaque Characteristics and Brain Perfusion

In the multivariable logistic model, plaque enhancement grade \( (P = 0.039, \text{OR} = 5.9, \text{95\% CI} \ 1.1–32.0) \) and MTT-ASPECTS \( (P = 0.003, \text{OR} = 2.6, \text{95\% CI} \ 1.4–4.7) \) was associated with acute cerebral infarction, whereas luminal stenosis grade was not \( (P = 0.128) \). The AUC (95\% CI) for determining the occurrence of infarction were 0.535 (0.351–0.720) for luminal stenosis grade, 0.725 (0.582–0.868) for plaque enhancement grade, and 0.881 (0.787–0.976) for MTT-ASPECTS. The combination of plaque enhancement grade and MTT-ASPECTS with luminal stenosis grade improves the AUC to 0.921, which is significantly higher than that of luminal stenosis grade alone.

**Discussion**

In the present study we demonstrated a relationship between the grade of plaque enhancement with perfusion changes and presence of cerebral infarction. A positive relationship between plaque enhancement grade and presence of cerebral infarction in the same vascular territory was observed. Strong enhancement plaques were associated with lower MTT-ASPECTS. MTT-ASPECTS and plaque enhancement grade provided incremental information on determining the likelihood of the occurrence of cerebral infarction beyond luminal stenosis severity.
grade, followed by less efficient collateral circulation establishment; 2) reduced perfusion may limit the ability of bloodstream to clear or wash out emboli and microemboli in the distal arteries.

The limitation of luminal stenosis to assess disease severity has been widely demonstrated in both intracranial and coronary arteries. In our study, there was no significant difference in luminal stenosis grade between the patient groups with or without acute infarction, indicating that luminal stenosis alone is a relatively poor predictor of myocardial ischemia compared to stenosis assessment. CT angiography could provide improved identification of myocardial ischemia compared to stenosis assessment alone.

Our results are consistent with these findings. The present study has some limitations. First, according to the widely used system for establishing the etiology of ischemic stroke, large-artery atherosclerosis is defined as luminal stenosis greater than 50%. Our study only included patients with MCA stenosis >50%. Future studies should include more patients with low-grade stenosis, which may broaden the understanding of stroke mechanisms. Second, the plaque features and MTT maps were visually assessed. Quantitative analysis of plaque features, such as plaque burden, volume, and length may provide incremental information on vulnerable lesions. Quantitative segmentation of the hypoperfused tissues according to an absolute value threshold may also provide more robust results. Third, we only included patients with MCA stenosis.

Atherosclerotic patients with posterior circulation plaques should be studied to determine whether similar results are observed. Finally, this was a retrospective study with a limited number of patients. Further prospective studies are needed to test the complementary value of HRVWI to acute ischemic stroke. Patients with ICAS are at risk of systemic atherosclerotic disease, with a considerable possibility of renal impairment exists. Patients in our study received a nonionic linear contrast agent (gadodiamida), which is a high-risk contrast agent. Patients with severe renal impairment may develop nephrogenic systemic fibrosis after administration of these contrast agents. Although patients with estimated glomerular filtration rates lower than 30 ml/min were excluded from this study, the use of low-risk contrast agents deserves more attention in ICAS patients.

In conclusion, the findings of the present study suggest that strongly enhanced intracranial plaques are associated with a higher likelihood of perfusion impairment and the presence of acute cerebral infarction. Plaque enhancement and perfusion evaluation may play a complementary role to luminal stenosis in determining the occurrence of acute cerebral infarction. Future prospective studies validating the clinical significance of HRVWI for ischemic stroke are warranted.

References


