The value of high-resolution MRI and perfusion weighted imaging in the middle cerebral artery atherosclerotic stenosis

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Aims and objectives

The ischemic cerebrovascular disease is a very common clinical disease which possess high incidence and cause severe disability rate, seriously affecting the human health. Middle cerebral artery (MCA), the main intracranial artery perfused in the cerebral hemispheres, is one of the most common locations for intracranial stenosis and is associated with a high mortality rate in symptomatic patients(1).

Currently, mainly available imaging modalities for the assessment of intracranial artery stenosis include digital subtraction angiography (DSA), computed tomography angiography (CTA), transcranial doppler ultrasonography (TCD) and magnetic resonance angiography (MRA). Despite there have been various imaging modality in clinical application, every individual imaging modality has its own merit and deficiency. DSA has long been considered the gold standard for evaluation of cerebral artery stenosis, but it is limited by availability, expense and risk of periprocedural complications (2). CTA is more widely used, quicker and offers excellent information about the degree of stenosis, but exposes patients to ionizing radiation (3-5). TCD often plays as an inexpensive, non-invasive screening technique while this method is operator-dependent and often limited by the inadequate acoustic temporal bone window (6, 7). MRA as another alternative, non-invasive method with demonstrated effectiveness but suffers from technical limitations such as overestimation of stenosis in post stenotic regions of low flow (8, 9). Currently, high-resolution MRI (HRMRI) has emerged as a new technique for evaluating intracranial artery stenosis. It is noninvasive and able to depict both the vessel lumen and vessel wall. In recent years, with the rapid development of high-field magnetic resonance, susceptibility perfusion-weighted imaging (PWI) plays an important role in evaluating regional cerebral transient ischemic attack (TIA) blood supply, especially echo planar imaging (EPI) technology, as well as the development of paramagnetic contrast agent. Magnetic resonance quantitative or semi-quantitative measuring the brain tissue perfusion technology has become more mature. Among all the examination methods, PWI can find cerebral hemodynamics abnormal earlier. Early detection and diagnosis of cerebral blood flow reduction is helpful to guid clinical work.

Herein the study seeks to explore the assessment value of HRMRI and PWI in patients of TIA with unilateral MCA atherosclerotic stenosis.

Methods and materials

Subjects:
Consecutive 43 referral patients (male 30, female 13, mean age 44.7±10.4 years old) presenting with symptoms of recent MCA territory transient ischemic attack (TIA) either clinically or on the basis of imaging, such as CTA or MRA, from March 2014 to March 2015 were recruited in this study. All these patients underwent MRA, HRMRI, and PWI. The study was approved by the First Affiliated Hospital of Zhengzhou University institutional ethics committee and written informed consent was obtained before each examination.

Inclusive criteria: all cases occurred TIA. TIA diagnostic criteria: transient neurological dysfunction caused by the brain, spinal cord or retinal focal cerebral ischemia without acute infarction. The conventional MRI scans and DWI show no acute cerebral infarction or large area of old infarction, and MRA examination revealed unilateral MCA stenosis. Exclusive criteria: MRA suggesting carotid artery, bilateral middle cerebral artery or anterior cerebral artery stenosis, moyamoya disease and other non-atherosclerotic stenosis disease; MRI scan showing intracranial hemorrhage, intracranial tumors, brain trauma, inflammatory diseases or other neurological diseases. All patients first underwent MRA to localize the unilateral M1 section stenosis of the MCA, then underwent HRMRI within the diseased section, and last went PWI. All examinations were carried out in the incidence of intermittent period. The HRMRI and PWI data were transported to the Siemens syngo workstation for processing. All the data were analyzed by two experienced radiologists who were blind to the clinical information of the patients. First they analyzed atherosclerotic plaques, distinguished plaque stability and atherosclerotic plaque AHA type.
Fig. 1: Plaque AHA Classification

**References:** American Heart Association, Inc and calculated the degree of stenosis on HRMRI according to the Samuel's standards (10). Then, they subdivided into mild stenosis (29%), moderate stenosis (30~69%), severe stenosis (70~99%), occlusion (100%). And last they processed the PWI data on the Siemens syngo workstation to ger a series of parameters including the relative cerebral blood volume (rCBV), relative cerebral blood flow (rCBF), relative mean transit time (rMTT), time to peak (TTP) of the regions (ROI) of interest.

**Imaging Protocol:**

MRI was performed with a 3-tesla MRI scanner (Siemens Verio, Germany) and a 16-channel phased-array head coil. The MR protocol included three parts: three-dimensional TOF-MRA, high-resolution MRI, which is T1-, T2-weighted black blood MRI and PWI. First, MRA was obtained in the axial plane, and data were reconstructed at the Siemens Verio post-processing workstation. Both the raw MRA data and the reconstructed blood vessel data were used for localizing the subsequent HRMRI. The image plane was acquired in an oblique sagittal plane in the patients with MCA steno-occlusive lesions along the short axes of the stenotic segments. Last the patients went PWI.
The imaging parameters for MRA were: TR/TE=21/3.6ms, FOV=181mm×200mm, Thk/Sp=0.7/0mm, Matrix=580×640. Parameters for T₁WI and T₂WI were as follows: TR/TE=861/18ms for T₁WI and TR/TE=903/83ms for T₂WI, FOV=130mm×130mm, Thk/Sp=2.0/0.2mm, Matrix=512×512. Fat suppression was used to reduce the signal intensity from surrounding fatty tissues. All the HRMRI sequences were acquired pulse-gated during the end-diastolic phase. Parameters for PWI were as follows: TR/TE=1500/30ms, FOV=230mm×230mm, Thk/Sp=4.0/1.2mm, Matrix=128×128, NEX=1, Flip angle=90°. PWI collected 60 times in a row, the first five scans without contrast injection, as the platform of the time signal intensity curve, and at the end of the fifth scan the patient was given fast bolus injection of Gd-DTPA, the dose of 0.2ml / kg, injection speed of 4ml / s, then the same dose of saline. There were 19 levels for each scan image, obtaining a total of 1140 layer images, the scanning time of 1 minute 38 seconds.

Statistical analysis

Quantitative data are expressed as mean ± SD. All data were analysed by SPSS17.0 package in statistics, using the paired t-test between the two groups, the relationships between variables were analyzed by Pearson correlation, P<0.05 as statistically significant difference.

Results

HRMRI got 155 positive slices type # of plaque 49 (31.6%), IV~#a 41 (26.5%), #b 4 (2.5%), # 13 (8.4%), #c 48 (31.0%). The soft plaques (type #~#a and VI) adds up to 54 (34.8%), hard plaques (type III,#b and#c) 101 (65.2%). There was no significant difference between hard and soft plaque distribution in the affected and normal MCAs (chi-square test P = 0.257). HRMRI diagnosed 4 cases of mild stenosis, 11 moderate , 22 severe stenosis, and 6 occlusion.

42 of the 43 patients had hemisphere perfusion difference between the affected and normal MCA perfusion districts, with lower rCBF , longer rMTT and TTP (P &lt 0.05) in the affected side. The degree of artery stenosis had no significant correlation with the values of rCBF and rCBV (P &gt 0.05), but it had significant correlation with the values of rTTP and rMTT in the affected side(P&lt0.05) in 11 cases of moderate stenosis, the Pearson's r were 0.760, 0.731 respectively(refer to table 1, 2 and figure 2, 3).

Table 1: Perfusion differences in patients of unilateral MCA moderate stenosis

<table>
<thead>
<tr>
<th>parameters</th>
<th>affected side</th>
<th>normal side</th>
<th>t value</th>
<th>P value</th>
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Table 2: correlation between stenosis rate and perfusion in patients of MCA moderate stenosis

<table>
<thead>
<tr>
<th></th>
<th>rCBV</th>
<th>rCBF</th>
<th>rMTT</th>
<th>rTTP</th>
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<tbody>
<tr>
<td>r value</td>
<td>0.115</td>
<td>0.134</td>
<td>0.760</td>
<td>0.688</td>
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<tr>
<td>P value</td>
<td>0.735</td>
<td>0.693</td>
<td>0.007</td>
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</table>
Fig. 2: Figure 2 MRA showed moderate stenosis within the M1 segment of the left MCA; HRMRT1WI and T2WI also showed moderate stenosis with diffuse thickness of the vessel wall; the plaque was obviously enhanced on post-contrast T1WI (AHA type#). References: MRI, The First Affiliated Hospital of Zhengzhou University - Zhengzhou/CN

Fig. 3: Figure 3 and 2 were the same patient. Figure 3 CBV and CBF demonstrated perfusion decrease in the brain parenchyma besides the left ventricle; MTT and TTP suggested perfusion delay in the perfusion area of the left MCA. References: MRI, The First Affiliated Hospital of Zhengzhou University - Zhengzhou/CN
While in the 22 cases of severe stenosis, the artery stenosis had significant correlation with the ratio of the affected side to the normal side of rMTT/rTTP \( (P<0.05) \). The Pearson’s \( r \) were 0.450, 0.857 respectively (refer to table 3, 4 figure 4, 5).

Table 3: Perfusion differences in patients of unilateral MCA severe stenosis

<table>
<thead>
<tr>
<th>parameters</th>
<th>affected side</th>
<th>normal side</th>
<th>( t ) value</th>
<th>( P ) value</th>
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</thead>
<tbody>
<tr>
<td>rCBV/mL/100g</td>
<td>384.99±150.94</td>
<td>389.82±257.40</td>
<td>-0.466</td>
<td>0.646</td>
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<tr>
<td>rCBF (mL/100g/min)</td>
<td>82.17±41.00</td>
<td>106.45±46.46</td>
<td>-5.694</td>
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<tr>
<td>rMTT (s)</td>
<td>1155.24±408.47</td>
<td>810.60±263.83</td>
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<tr>
<td>rTTP (s)</td>
<td>3122.13±186.42</td>
<td>2793.11±138.49</td>
<td>7.827</td>
<td>0.000</td>
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</table>

Table 4: Correlation between stenosis rate and perfusion in patients of MCA severe stenosis

<table>
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<tr>
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<th>rCBF</th>
<th>rMTT</th>
<th>rTTP</th>
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</thead>
<tbody>
<tr>
<td>( r ) value</td>
<td>-0.204</td>
<td>0.280</td>
<td>-0.099</td>
<td>0.313</td>
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<tr>
<td>( P ) value</td>
<td>0.363</td>
<td>0.207</td>
<td>0.660</td>
<td>0.156</td>
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</table>
Fig. 4: Figure 4 MRA showed severe stenosis within the M1 segment of the left MCA; HRMRT1WI and T2WI also showed severe stenosis with eccentric thickness of the vessel wall; the plaque was enhanced on post-contrast T1WI (AHA type#a). 

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Fig. 5: Figure 5 and 4 were the same patient. Figure 5 CBV and CBF demonstrated perfusion decrease in the brain parenchyma besides the left ventricle; MTT and TTP suggested obvious perfusion delay in the perfusion area of the left MCA.

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Images for this section:
<table>
<thead>
<tr>
<th>Pathologic features</th>
<th>Progression</th>
<th>Mechanism</th>
<th>Course</th>
<th>Manifestation</th>
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<tbody>
<tr>
<td>I (initial) Foam cells increase</td>
<td>I</td>
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<td></td>
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<tr>
<td>II (fatty streak) Intracellular lipid accumulation</td>
<td>II</td>
<td>Lipid accumulation</td>
<td>about 10 years</td>
<td>Recessive</td>
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<tr>
<td>III (intermediate) Changes of II &amp; small extracellular lipid pools</td>
<td>III</td>
<td>Smooth muscle and collagen increase rapidly</td>
<td>about 30 years</td>
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</tr>
<tr>
<td>IV (atheromatous plaque) Changes of II &amp; extracellular lipid core</td>
<td>IV</td>
<td></td>
<td>about 40 years</td>
<td></td>
</tr>
<tr>
<td>V (fibrous plaque) Single or multiple lipid core &amp; fibrous layer or mainly containing calcification or fibrous tissue</td>
<td>V</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI (complicated plaque) Fibrous cap rupture, hematoma-hemorrhage, thrombosis</td>
<td>VI</td>
<td>Hematoma hemorrhage</td>
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</tbody>
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**Conclusion**

HRMRI can assess the AHA sub-type and stability of the atherosclerotic plaque, and the degree of stenosis of MCA. MTT, TTP can be found changes in early ischemic events of the brain, which are sensitive parameters to diagnose TIA.

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**References**


