Application of High-Resolution CUBE Sequence in Exploring Stroke Mechanisms of Atherosclerotic Stenosis of Middle Cerebral Artery

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Background: This study aimed to analyze the vascular wall and atherosclerotic plaques of the middle cerebral artery (MCA) and compare their differences between patients with cerebral infarction and transient ischemic attack (TIA) using 3-dimensional fast-spin-echo T1-weighted sequence (namely CUBE). *Methods:* Forty-seven patients with atherosclerotic stenosis of the MCA were included in this study. They received magnetic resonance examinations with routine T1WI, T2WI, 3-dimensional time-of-flight magnetic resonance angiography and diffusion-weighted imaging, as well as high-resolution CUBE T1WI sequence. Two physicians independently observed the location and degree of enhancement of the atheromatous plaques. The vessel area and lumen area at the maximal-lumen-narrowing and reference site were measured to calculate the plaque area, rate of stenosis, and remodeling index of the MCA. The chi-squared test was used to compare the differences of degree of enhancement between the cerebral infarction and TIA groups. The differences of rate of stenosis and remodeling index were compared by independent sample t test. *Results:* Twenty-five lesion vessels in the infarction group and 22 in the TIA group were analyzed. The difference of stenosis rate between the groups was not statistically significant. The lesion vessels of infarction group had a significantly larger remodeling index and plaque area, and the plaques had a significantly higher degree of enhancement, compared to the TIA group. *Conclusions:* CUBE T1WI can be used to characterize the MCA vessel wall and atherosclerotic plaque. Positive remodeling and enhanced plaques are closely correlated with the occurrence of brain stroke.

Key Words: CUBE T1WI—plaque—middle cerebral artery—infarction—transient ischemic attack—stenosis rate—remodeling index

Introduction

Atherosclerosis is a systemic disease that can lead to cerebral infarction or transient ischemic attack (TIA), when the carotid or intracranial arteries are involved. Carotid atherosclerosis is the most common cause of cerebrovascular ischemic events in Europeans and

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Americans,[1] whereas intracranial atherosclerosis is more common in Asians.[2] In China, 33%-50% of patients with cerebral infarctions and more than 50% of patients with TIA reportedly experienced symptomatic intracranial atherosclerotic stenosis, in which the middle cerebral artery (MCA) was the most commonly involved vessel.[3] Atherosclerotic stenosis of intracranial arteries can be evaluated using traditional angiography techniques including digital subtraction angiography, computed tomography angiography, and magnetic resonance angiography (MRA).[4] These approaches may be insufficient to diagnose atherosclerotic diseases only according to the degree of vascular stenosis. Incorporation of plaque stability in the carotid artery into the hierarchical management of ischemic events provides greater accuracy than the evaluation of luminal stenosis. Thus, further knowledge of the structure of the vascular wall may be valuable and clinically relevant.

High-resolution (HR) magnetic resonance imaging (MRI) is currently the only imaging method for the intracranial vascular wall in vivo, and has become a topic of interest in studies of intracranial atherosclerotic stenosis.[5,6] Three-dimensional (3D) isotropic fast-spin-echo (FSE) T1-weighted sequence (namely CUBE T1WI in GE) is a novel HR black-blood imaging method based on an ultra-long echo train length (ETL) and multiple flip angles (FAs) 3D-FSE sequence.[7] Volume data can be reconstructed in all directions with a high signal-to-noise ratio, contrast-to-noise ratio, and isotropic resolution. Compared with traditional angiography, CUBE T1WI can not only evaluate the degree of arterial stenosis, but also provide more information about atherosclerotic plaques, arterial dissection, vascular wall remodeling, and vasculitis.

Previous studies had found that expansile reconstruction pattern in carotid arteries indicates the instability of plaques, which is closely correlated with the occurrence of acute ischemic events.[8] In this study, we compared the remodeling index (RI) and plaque burden of the MCA between the patients with infarctions and TIA, according to the results of diffusion-weighted imaging (DWI), and compared the degree of enhancement after administration of contrast agent, which could also determine the stability of plaques because of the newborn capillaries and inflammation inside the plaques.[9]

Materials and Methods

Patients

We initially recruited 60 patients with relevant neurological symptoms of ischemic stroke, or patients suspected of ischemic stroke, from January 2016 to December 2017. They all provided informed consent before MRI examination. The study was approved by the ethics committee of our hospital. The infarction group of patients had a high DWI signal in the territory supplied by MCA. Patients who had ischemic neurological symptoms without relevant imaging findings comprised the TIA group. The inclusion criteria were as follows: stenotic MCA was the responsible vessel detected by MRA; absence of stenosis of the ipsilateral internal carotid artery; at least 1 risk factor of atherosclerosis including hypertension, diabetes, dyslipidemia, obesity, and smoking;[10] and no history of cardiovascular events or evidence of cardioembolism. Patients with nonatherosclerotic vasculopathy, such as vasculitis, arterial dissection, perforating artery disease, and Moyamoya disease were excluded.[11] After eliminating 2 patients with Moyamoya disease and 11 with other artery stenoses, 47 patients were finally included. They included 21 males and 26 females ranging in age from 45 to 75 years (mean age, 58.5 ± 8.2 years). Detailed clinical characteristics are provided in Table 1.

Imaging Protocol

A 3-Tesla scanner with an 8-channel head coil (MR750; GE Healthcare, Milwaukee, WI) was used. The imaging protocols included T1-weighted imaging (T1WI), T2-weighted imaging (T2WI), DWI, 3-dimensional time-of-flight magnetic resonance angiography, and CUBE T1WI. The parameters of T1WI were set as follows: repetition time (TR) 1750 ms, echo time (TE) 25 ms, thickness 5 mm, spacing 1.5 mm, field-of-view (FOV) 24 × 18 cm, and matrix 320 × 256. The T2WI parameters were as follows: TR 6818 ms, TE 106 ms, thickness 5 mm, spacing 1.5 mm, FOV 24 × 18 cm, and matrix 512 × 512. The DWI parameters were as follows: TR 3000 ms, TE 71 ms, thickness 5 mm, spacing 1.5 mm, FOV 24 × 24 cm, and matrix 160 × 160, and b value 1000 s/mm^2. Three-dimensional time-of-flight magnetic resonance angiography was obtained using the following parameters: TR 21 ms, TE 3.1 ms, thickness

Table 1. *Clinical characteristics between the infarction group and TIA group*

	Age (years)	Males (%)	Hypertension	Diabetes	Dyslipidemia	Obesity	Smoking
Infarction (n = 25)	59.3 ± 9.4	14 (56%)	17 (68%)	14 (56%)	9 (36%)	14 (56%)	8 (32%)
TIA (n = 22)	57.7 ± 8.1	7 (32%)	14 (63%)	16 (72%)	9 (41%)	13 (59%)	6 (27%)
P	0.467	**0.036**	0.218	**0.021**	0.536	0.348	0.072

Abbreviation: TIA, transient ischemic attack.

Two independent sample t test and chi-squared test are used for comparison. $P < 0.05$ is considered statistically significant.

0.6 mm, FOV 22.0 × 17.8 cm, and matrix 480 × 448. CUBE T1WI[12] was acquired in an oblique coronal plane with the following parameters: TR 800 ms, TE 16 ms, thickness 0.6 mm, FOV 23.0 × 18.4 cm, matrix 480 × 320, ETL 24, phase acceleration 2, and initial/minimum FA, 130°/20°. We applied variable refocusing FAs to compensate for the signal decay resulting from the long ETL used to reduce the scanning time in the FSE sequence. The techniques of propeller k-space filling, fat suppression, and parallel acceleration along the phase direction were also used in CUBE T1WI. The enhanced CUBE T1WI was performed after administration of contrast medium (Magnevist, 0.2 mmol/kg; Bayer Schering Pharma, Berlin, Germany) at a rate of 3 mL/s, followed by a 15-mL saline flush. The voxel size, which represented spatial resolution, was 0.48 × 0.57 × 0.60 mm^3. Nearly 200 slices covering both the anterior and posterior circulation were obtained in 6 minutes, 32 seconds.

Image Analysis and Postprocessing

The image quality was divided into 3 levels; cases with image quality at Level 1 and Level 2 were included in this study. In the standards of classification, Leve1 1 were high-definition images without motion artifacts, Level 2 were less clear images with mild motion artifacts that were still useful for diagnosis, and Level 3 were poor quality images with obvious motion artifacts and obscure vessel. The CUBE T1WI images were sent to an Advantage Workstation 4.5 for postprocessing (GE Medical Systems). Multiplanar reconstruction along the route of the MCA was applied to the CUBE T1 images. The degree of enhancement of the MCA plaques was observed. The vessel area (VA) and lumen area (LA) were manually measured at the maximal-lumen-narrowing (MLN) site and reference site (Fig 1). The reference site was defined as lesion-free or minimally diseased segments proximal or distal to the stenosis. The vessel-blood interface and vessel-cerebrospinal fluid interface were used to trace the LA and VA, respectively. The means of the proximal and distal values are taken as the reference LA and VA. The wall area (WA) was calculated as (VA-LA) and the plaque area (PA) was estimated as ($WA_{MLN}-WA_{reference}$). The degree of stenosis was calculated as $(1 - LA_{MLN} / LA_{reference}) \times 100\%$. VA_{MLN} meant vessel area at the maximal-lumen-narrowing site, with LA_{MLN} defined as the lumen area at the maximal-lumen-narrowing site, $VA_{reference}$ defined as the vessel area at the reference site, and $LA_{reference}$ defined as the lumen area at the reference site. RI was calculated as $VA_{MLN} / VA_{reference} \times 100\%$. As previously described, RI greater than or equal to1.05 is regarded as positive remodeling (PR), RI less than or equal to 0.95 indicates negative remodeling (NR); while 0.95 less than RI less than1.05 is regarded as

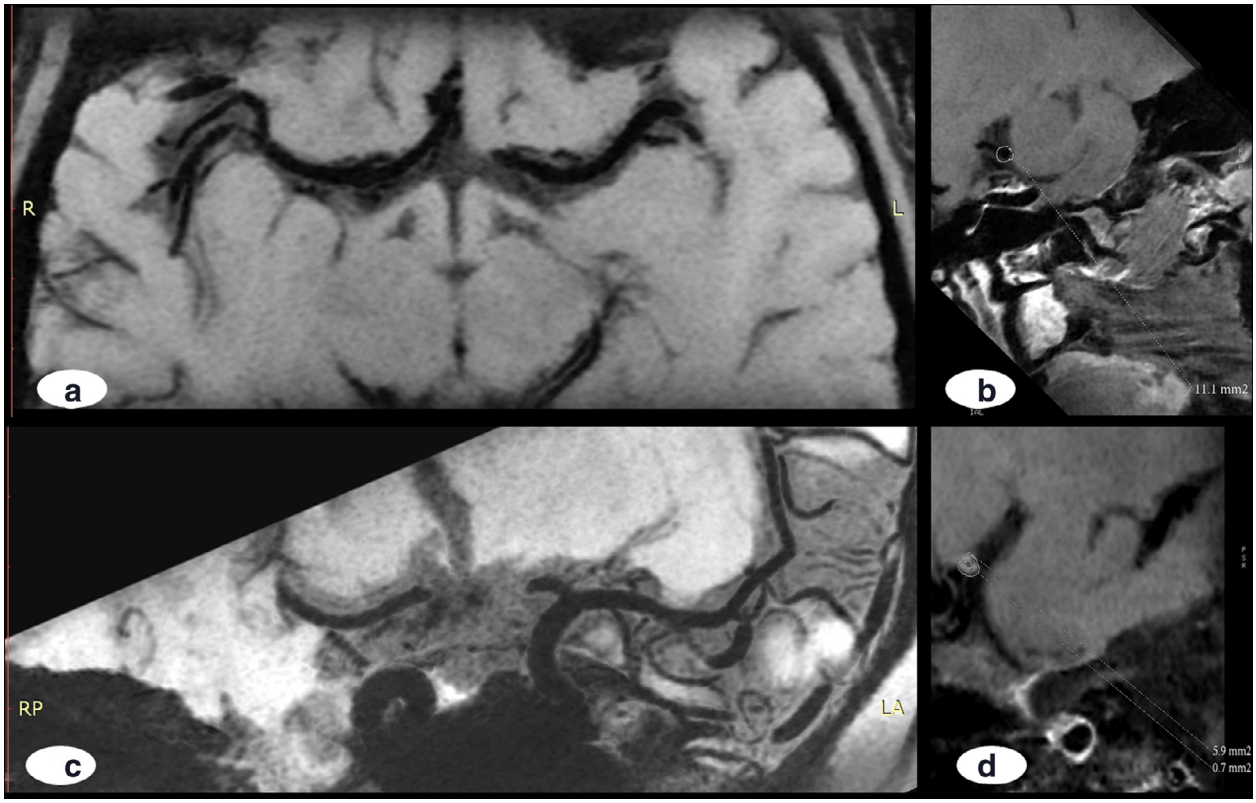

Figure 1. *Multiplanar reconstruction and measurement method used in this study. The vascular wall and lumen were clearly evident in the axial (a) and oblique plane (c) of CUBE T1WI. The vessel area at a reference site (b), vessel area and the lumen area at the maximal-lumen-narrowing site (d) were measured in the sagittal plane after zooming in to an appropriate field of view.*

Table 2. *Stenosis rate and remodeling index between infarction group and TIA group*

	Infarction group	TIA group	*P*
VA_{MLN} (mm^2)	8.65 ± 1.93	7.10 ± 1.38	**<0.001**
LA_{MLN} (mm^2)	2.64 ± 1.55	2.72 ± 1.26	0.696
$VA_{reference}$ (mm^2)	8.13 ± 1.60	8.06 ± 1.42	0.906
$LA_{reference}$ (mm^2)	5.21 ± 2.31	5.07 ± 1.85	0.863
PA (mm^2)	3.62 ± 1.86	3.14 ± 1.55	**0.032**
Stenosis rate	0.68 ± 0.08	0.66 ± 0.14	0.584
Remodeling index	1.06 ± 0.11	0.88 ± 0.23	**<0.001**

Abbreviation:VA_{MLN}, vessel area at the maximal-lumen-narrowing site; LA_{MLN}, lumen area at the maximal-lumen-narrowing site; $VA_{reference}$, vessel area at the reference site; $LA_{reference}$, lumen area at the reference site; PA, plaque area; TIA, transient ischemic attack.

Two independent sample t test is used for comparison. $P < 0.05$ is considered statistically significant.

Table 3. *Degree of enhancement of plaques between infarction group and TIA group*

Number of plaques	Infarction group (n = 25)	TIA group (n = 22)	*P*
Mild enhancement	2 (8%)	6 (29%)	**0.027**
Moderate enhancement	9 (36%)	13 (57%)	
Obvious enhancement	14 (56%)	3 (14%)	

Abbreviation: TIA, transient ischemic attack.

Chi-squared test was used for comparison. $P < 0.05$ is considered statistically significant.

nonremodeling.[13] We measured the signal intensity (SI) of plaques before and after enhancement, and calculated $(SI_{pre} - SI_{post})/SI_{pre}$ as the enhanced percentage, SI_{pre} and SI_{post} meant signal intensity of plaques before and after enhancement, respectively.[14] Mild enhancement was defined as enhanced percentage less than 10%, moderate as 10%-50%, and obvious as higher than 50%.

Statistical Analyses

The SPSS 13.0 statistical software package (SPSS, Chicago, IL) was used for statistical analysis. Measurement data were expressed as mean ± standard deviation and compared by 2 independent sample t test. Chi-squared or Fisher's exact test was used to compare the differences in the degree of enhancement between the cerebral infarction group and TIA group. $P < .05$ was considered statistically significant.

Results

After preliminarily evaluation of the image quality of all cases, 7 cases displayed mild motion artifacts caused by patient anxiety or discomfort during the examinations. We talked to these 7 patients and rescanned using the minimum number of slices as possible to cover the MCA. All the images were qualified. Among the 47 patients, 28 cases occurred in the left side, and 19 in the right side. Twenty-five lesion vessels in the infarction group and 22 in the TIA group were analyzed. VA at the reference sites and LA at the MLN sites were similar between the 2 groups ($P = .906$ and $P = .696$, respectively). VA and PA of infarction group at the MLN sites was significantly larger than that of TIA group ($P < .001$ and $P = .032$, respectively). The stenosis rate between infarction group and TIA group was not significantly different. Compared with the TIA group, the lesion vessels of the infarction group had a significantly larger remodeling index and plaque burdens, and the plaques had a significantly higher degree of enhancement. The details were provided in Tables 2 and 3. Representative cases in the infarction group and TIA group were showed in Fig. 2 and 3, respectively.

Discussion

Intracranial atherosclerotic stenosis is an important cause of ischemic stroke and TIA. It commonly occurs in the MCA and basilar artery. The territory supplied by the MCA has a higher incidence of ischemic stroke because the MCA is the most easily involved artery and supplies a large region of the brain.[15] Clinical treatment of ischemic stroke is mainly based on the degree of vascular stenosis. However, ischemic events may still recur in the patients with mild to moderate stenosis after medical treatment. As a result, investigation of plaque stability seems more valuable to the neurologist. Compared with traditional imaging methods, HR-MRI has a unique advantage in visualizing the vessel wall and provides more details about the plaques, which has been successfully used in carotid atherosclerosis.[16] Similarly, we investigated the stroke mechanisms of atherosclerotic stenosis of the MCA with a 3.0-T MR scanner using the CUBE T1WI sequence. The signal-to-noise ratio and resolution were acceptable in this study.

The present findings suggest that the difference in the stenosis rate was not significant between the infarction and TIA groups, indicating that the degree of vascular stenosis was not the decisive factor in the occurrence of cerebral infarction. However, the lesion vessels of infarction group

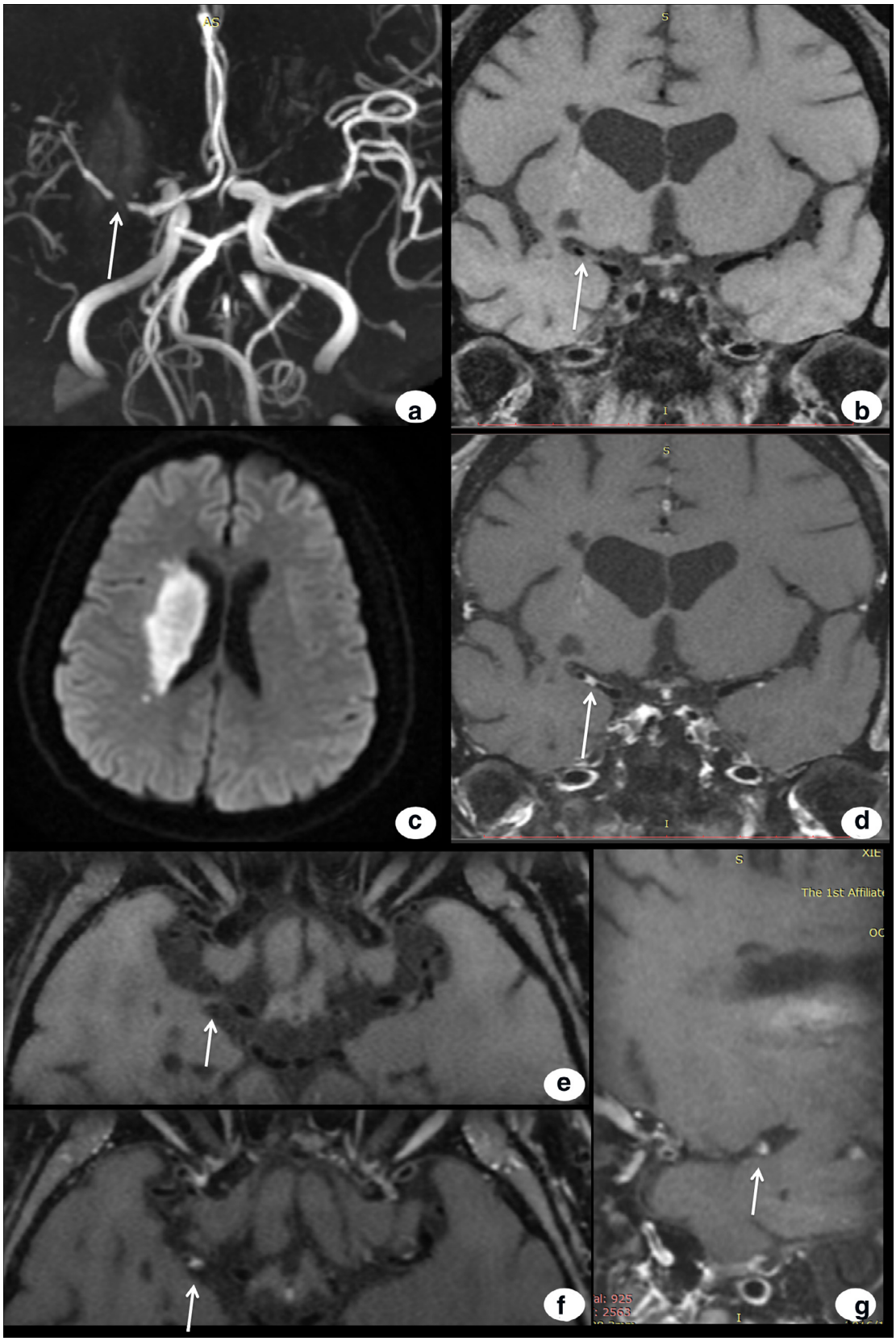

Figure 2. *Case of a 64-year-old male patient 5 hours after an ischemic stroke. MRA showed obvious stenosis at the M1 segment of the right MCA (a). DWI showed a large area of high signal in the right basal ganglia (c). The plaques in the lesion vessel showed significant enhancement on a coronal (d), axial (f), and sagittal plane (g), compared with the nonenhanced images (b, e).*

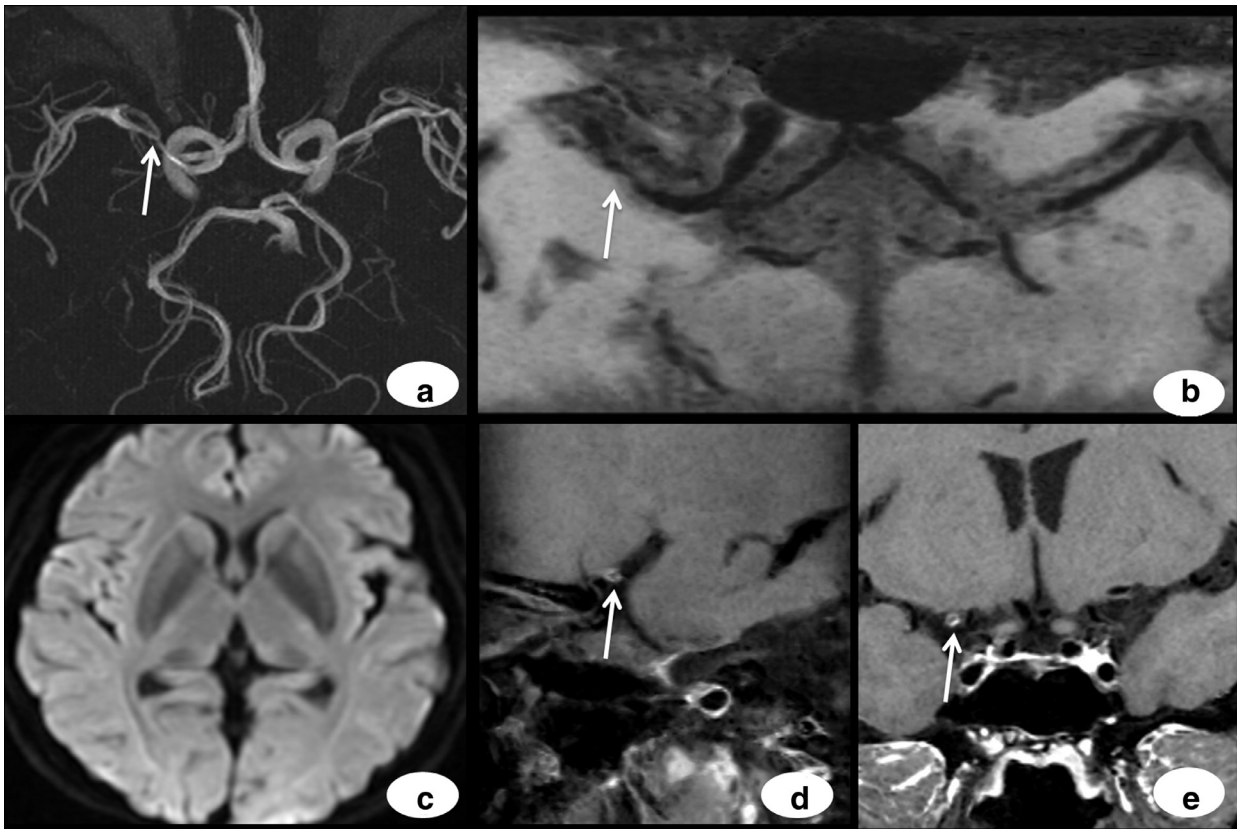

Figure 3. *Case of a 47-year-old male patient with a 1-week history of headache and dizziness. MRA revealed mild to obvious stenosis of the M1 segment of the right MCA (a). No lesion could be seen on a DWI image (c). Plain CUBE T1WI showed a plaque with broad base and relatively low signal intensity locating at the back wall of the MCA (b). The plaque had a moderate enhancement on a sagittal (d) and coronal (e) enhanced image.*

had a higher RI and PA than that of the TIA group. The MCA of stroke patients tended to be PR, whereas the TIA patients were likely to be NR. Dilation of the artery may have resulted from the physiological compensatory responses to ensure the blood supply in an early ischemic stage of atherosclerotic stenosis. Since the PR can maintain vascular patency or relieve ischemic symptoms for a certain time, the potential stroke risks of these patients may be underestimated if evaluated solely by the degree of arterial stenosis. The study of Zhao et al[11] indicated that the MCA with PR had a larger plaque burden; the result was similar with our study. Another study reported that patients with PR were inclined to have microemboli and a larger VA and WA at the MLN site than those without PR.[17] These pathological changes may have resulted from repeated reparation of ruptured plaques, and increased blood deposited in the dilated vessels under the stimulus of the remodeling patterns. The high biological activity would lead to the instability and vulnerability of the plaques. Previous studies[18-20] reported that patients with PR in their coronary arteries display higher levels of low-density lipoprotein, C-reactive protein, and homocysteine, which are correlated with advanced atherosclerosis and unstable plaques, as well as PR of the MCA in this study. Furthermore, ulceration and hemorrhage were more commonly seen in the unstable plaques, which may easily fall off under the repeated flushing of blood flow. Other authors described intraplaque hemorrhage as an independent risk factor of ischemic stroke, with an important role in the progress of plaque-related events.[21] These collective risk factors may account for the prevalence of ischemic stroke in the infarction group.

Previous studies have revealed the relationship between the signal changes and pathological components of plaques in HR-MRI examinations of the carotid and cerebral arteries.[22, 23] However, the enhanced characteristics of plaques and the relation with stroke risk had not been clearly interpreted. In this study, the enhanced CUBE T1WI improved the contrast of plaques and vessel walls for accurate measurement, and provides additional information for stability assessment. A statistical difference in the degree of enhancement was evident between the infarction group and TIA group. The plaques in PR MCA had a higher degree of enhancement than the plaques in NR MCA. The neovascularization inside the plaque may account for the obvious enhancement of a plaque. With higher biological activity, the degree of enhancement is increased. The increased endothelial cell permeability and extracellular interstitial volume caused by inflammation also lead to significant enhancement in the plaques. Contrast enhancement has been closely related with the stability and vulnerability of the intracranial

atherosclerotic plaques, and it may serve as a surrogate marker for predicting stroke risk.[14] In another study, plaques in symptomatic patients had a distinctly higher signal enhancement compared with asymptomatic patients in human carotid atherosclerotic plaques using gadofosveset-enhanced MRI.[24] On the other hand, the lesion vessels with constricted changes in TIA patients may result from the calcification and sclerosis of the plaques at a relatively late stage of atherosclerosis, indicating a close relationship between NR and stable plaque.[25]

There were some limitations in this study. First, we mainly concentrated on the plaque burden, enhancement, and arterial remodeling pattern of the MCA. Signal changes of T1WI, T2WI, and PDWI of the plaques were not investigated, since numerous prior studies addressed these signal changes. Second, the image findings were mainly referenced to other relevant coronary and carotid studies without pathologic confirmation, and the reliability of the results may decrease, to a certain extent. The pathologic mechanisms of enhancement need further verification. Third, the scanning time of CUBE T1WI sequence was somewhat long, which may introduce motion artifacts to the images. Fourth, we could not identify the special contents of the small plaques using CUBE T1WI, and only made a global observation on plaque enhancement.

In conclusion, just assessing the vascular stenosis is not sufficient for patients with intracranial atherosclerosis, and evaluating the plaque stability is more important in exploring the stroke mechanisms. HR CUBE T1WI can be used to characterize the vessel wall and atherosclerotic plaque of MCA. Positive remodeling and significant enhancement are closely correlated with the unstable plaques, which may increase the stroke risk in atherosclerotic patients. Assessment of plaque stability should be included in the hierarchical management of ischemic events.

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