Diagnostic Usefulness of High Resolution Cross Sectional MRI in Symptomatic Middle Cerebral Arterial Dissection

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Dissection of the middle cerebral artery (MCA) is less frequent compared with dissection of the vertebrobasilar system or carotid artery. Recently, high-resolution cross sectional MR imaging (HRMRI) has emerged as a potential technique for atherosclerotic plaque imaging in MCA. We introduce the findings of HRMRI in a 56-year-old woman with traumatic MCA dissection. HRMRI showed an intimal flap and tapered pseudolumen with intraluminal hemorrhage. We performed stent deployment about MCA dissection after failed medical treatment. Three months later, there was no instant restenosis and no further neurological deficit were noted.

Key Words : Dissection · Magnetic resonance imaging · Stent.

INTRODUCTION

Dissection of the middle cerebral artery (MCA) is an uncommon cause of stroke, and less frequent compared with dissection of the vertebrobasilar system or carotid artery7). Most dissections of the anterior circulation are associated with cerebral ischemia3,5), and result in complete stroke due to arterial stenosis or occlusion8,10). Angiography is the gold standard for the diagnosis of dissection of the MCA. Magnetic resonance imaging (MRI) is also recognized as a useful diagnostic tool, as axial MRI has detected intramural hematoma in some cases of intracranial artery dissection11,16).

Recently, high-resolution cross sectional MR imaging (HRMRI) has emerged as a potential technique for atherosclerotic plaque imaging in the MCA8,12). We report a patient of traumatic MCA dissection with intimal flap identified by HRMRI and stent deployment about MCA dissection after failed response with medical treatment.

CASE REPORT

A 56-year-old woman was seen in the emergency department with left side weakness and dysarthria. She had a history of head trauma one-month prior by the traffic accident and did not show abnormal findings such as hemorrhage or infarction on brain computed tomography (CT) at that time. She did not have a history of cigarette smoking, drinking but had mild untreated hyperlipidemia.

At admission in emergency room, blood pressure was 130/70 mm Hg. Initial brain CT without contrast revealed no sign of hemorrhage. The patient visited to our institution within 2 hours of the onset of stroke symptoms and had serious neurological symptoms as defined by a National Institutes of Health Stroke Scale score of 5. We started intravenous tissue-plasminogen activator.

And then, brain MRI was performed for confirmation of cerebral ischemia. MRI was performed with a 3-tesla MRI scanner and an eight-channel phased array coil. DWI showed focal infarctions in the right basal ganglia and perfusion MR detected large mismatched area in the right MCA territory (Fig. 1A, B). Also, MR angiography revealed focal and severe stenosis of the right MCA. We performed HRMRI for evaluation of MCA stenosis. The imaging parameters for the TOF-MRA scan were: repetition time (TR)/echo time (TE)=25/3.45 ms, flip angle= 20°, field of view (FOV)=197×249 mm, matrix size=416×208, sensitivity encoding factor=2.5, slice thickness=0.60 mm, and number of average (NEX)=1. The TOF-MRA scan time was 4 min. T1-weighted MRI was acquired using a two-dimensional turbo spin echo (TSE) sequence with the following imaging parameters : TR/TE=581/20 ms, FOV=120×105 mm, matrix size=320×220,
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Hyperintensity around the flow void of the distal portion of the lesions on T1-weighted image was suggestive of intramural hematoma. However, MR findings of an intimal flap and false lumen are uncommon.

Recently, HRMRI has emerged as a potential technique for atherosclerotic plaque imaging in MCA. Especially, some plaques showed the inhomogeneous components with high signal intensity within themselves. At 3.0T, the radiofrequency wavelength is estimated to be about 25-30 cm within the body and becomes comparable to or even smaller than the anatomic structures. As a result, radiofrequency waves interact strongly with global structures in the imaging plane, especially in regions with high permeability and low conductivity. In theory, an increase in field strength from 1.5T to 3.0T should result in a

echo train length (ETL)=6, and NEX=4. For both the PD-weighted and T2-weighted HR-MRI scans, the TSE sequence used a TR/TE=3000/80 ms for T2-weighted images and a TR/TE=2500/30 for PD-weighted images. Both scans were run with FOV=120×105 mm, matrix size=320×220, ETL=16, and NEX=4. The black blood technique with preregional saturation pulses of 80 mm thickness to saturate incoming arterial flow was used for all three scans. The longitudinal coverage of each artery was 14-16 mm (7-8 slices) for three scans. The scan time was 3 min 30 s for each scan. HRMRI showed an intimal flap and tapered pseudolumen with intraluminal hemorrhage (Fig. 1C). We could easily diagnose MCA dissection with using HRMRI.

Comparison to HRMRI, we performed digital subtraction angiography (DSA) that showed a definite double lumen, diagnostic of arterial dissection, at the M1 segment of the right MCA (Fig. 1D). The true lumen appeared to become severe stenosis due to intimal flap and tapered pseudolumen and distal flow of MCA was delayed due to M1 stenosis. Surprisingly, angiography showed same results as HRMRI did. After appropriate medical treatment, we carried out follow-up DSA that the true lumen remained severe stenosis due to pseudolumen, distal flow of MCA was continuously delayed, and contrast flow of perforators was decreased. Therefore, we decided to attempt intracranial stent. A 2.5×15-mm, balloon-expandable stent (FLEXMASTER, Abbott, Rangendingen, Germany) was placed in the true lumen to cover the dissection. Final angiography did not show an intimal flap, pseudoaneurysm, or delay flow of MCA (Fig. 2A). Three months later, there was no in-stent restenosis (Fig. 2B) and no further neurological deficit were noted.

DISCUSSION

Arterial dissection is an important cause of stroke, particularly in young victims, and most of them have been reported to involve the extracranial carotid and vertebral arteries. It is known that the annual incidence of the carotid artery dissection is 2.5 to 3 per 100,000 and that of vertebral artery dissection is 1 to 1.5 per 100,000. However, in clinical practice, isolated MCA dissection is an extremely rare clinical entity. Although etiology of MCA dissection remains uncertain or idiopathic, trauma is important in the development of extracranial dissection, but its role in the intracranial dissection is less clear. Some authors have suggested that the impact of the MCA against the sphenoid ridge causes an intimal tear and even rupture, which resulted in a posttraumatic dissection.

Typical angiographic findings of MCA dissection are similar to those observed with dissection of the extracranial arteries including string sign, irregular stenosis, pseudoaneurysm and even total occlusion. Angiographic demonstration of an intimal flap, false lumen, or pseudoaneurysm is relatively uncommon. Segmental stenosis of the involved vessel segment is the most common angiographic finding. In particular, diagnosis of MCA dissection on stroke MR protocol in patients with acute infarction at emergency room is very difficult. Hyperintensity around the flow void of the distal portion of the lesions on T1-weighted image was suggestive of intramural hematoma. However, MR findings of an intimal flap and false lumen are uncommon.

Fig. 1. Initial brain magnetic resonance image (MRI) and digital subtraction angiogram (DSA) of this patient. A : Diffusion weighted MRI shows focal acute cerebral infarction of the right basal ganglia. B : Perfusion weighted MRI reveals severe perfusion decrease on the right middle cerebral artery (MCA) territory. C : HRMRI shows a definite double lumen (arrow) with intramural hemorrhage. D : DSA presents MCA dissection due to intimal flap.

Fig. 2. A : Digital subtraction angiogram (DSA) after stent deployment shows complete recanalization of the right middle cerebral artery (MCA) and no visualization of the intimal flap. B : Follow up DSA after 3 months, there is no in-stent restenosis.
doubling of SNR, which can potentially allow for improved spatial resolution or reduced imaging time. However, both \( B_0 \) and \( B_1 \) field inhomogeneities tend to be more severe at 3.0T. In our study, HRMRI showed an intimal flap and tapered pseudo-lumen with intraluminal hemorrhage. These findings on HRMRI can confirm a MCA dissection.

The appropriate management of MCA dissection remains controversial. Treatment for ischemic symptoms due to stenosis or occlusion by the dissection or subarachnoid hemorrhage due to dissection aneurysms should be selected based on the severity of the symptoms and the location of the dissection. Among patients with intracranial dissecting aneurysms associated with ischemia, there was no statistically significant difference between numbers of patients chosen for surgical and conservative treatment\(^1\). Both surgical and conservative therapy led to good outcomes in patients with anterior cerebral artery dissection aneurysms\(^7\). Early stent placement for intracranial artery dissection was reported for iatrogenic lesions occurring as a result of angioplasty\(^11\). In addition, stent placement has been reported for the treatment of symptomatic intracranial carotid artery dissections\(^6\). Suh et al.\(^10\) reported that balloon-expandable intracranial stent placement revealed a low adverse effect and good outcome rate at 6 months. In our case, successful stent placement into the true lumen of MCA dissection in patient without response of anticoagulation therapy could preserve the MCA flow without adverse effect.

**CONCLUSION**

As in our case, we can easily detect the wall structure of MCA and double lumen to use of HRMRI in MCA dissection. Also, stent deployment can possibly be treated more effectively in patients of MCA dissection without response after medical treatment.

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