

CASE REPORTS

Isolated middle cerebral artery dissection with atherosclerosis: A case report

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Abstract

Isolated middle cerebral artery (MCA) dissection with atherosclerosis is a rare entity, and its clinical progression is not well known. We recently came across a case of isolated MCA dissection with atherosclerosis. A 62-year-old man presented to the emergency department with right-sided weakness and mild aphasia. Diffusion-weighted imaging (DWI) showed a multifocal infarction in the left MCA region, and perfusion Magnetic resonance imaging (MRI) detected a moderate time delay in the left MCA region. High-resolution MRI and transfemoral cerebral angiography revealed that the atherosclerotic plaque was accompanied by the dissecting intimal flap. Despite 40 days of antiplatelet therapy, the ischemic stroke recurred and the dissection did not heal. After stenting, the MCA and intracranial circulation revealed a widened lumen and improved flow across the dissection, and no embolic sequelae in the distal intracranial circulation. This case suggest that in MCA dissection with atherosclerosis, early stage intracranial stenting may be a better therapeutic strategy than medical treatment, to prevent recurrent cerebral infarction.

Keywords: Middle cerebral artery dissection with atherosclerosis, middle cerebral artery dissection, atherosclerosis

INTRODUCTION

Isolated middle cerebral artery (MCA) dissection as a cause of stroke has been rarely reported. Dissection of the MCA occurs when the intima tears, and subsequently forms an intramural hematoma. Thus, intracranial artery dissection may result in the formation of an intimal flap or a double lumen. Atherosclerosis is a chronic inflammatory process involving large to medium-sized arteries, with formation of an atheromatous plaque in the arterial tunica intima and consequent artery lumen stenosis/occlusion.

The relationship between atherosclerosis and dissection has not yet been studied.¹ We report here, a case of MCA dissection with atherosclerosis, identified by high resolution - magnetic resonance imaging (HR-MRI) and conventional angiography and discuss the clinical outcome of such patients.

CASE REPORT

A 62-year-old man with a history of ischemic

stroke and taking aspirin for 9 years, presented to the emergency department with right-sided weakness and mild aphasia. He had a history of diabetes, hypertension, and mild untreated hypercholesterolemia. He visited our institution within 24 hours of the onset of stroke symptoms and had a neurological symptoms score of 4 as per the National Institutes of Health Stroke Scale (NIHSS). Initial computed tomography scanning revealed a hypodense area in the left corona radiata and basal ganglia without hemorrhage.

Brain Magnetic resonance imaging (MRI) was performed to confirm cerebral ischemia. Diffusion-weighted imaging (DWI) showed a multifocal infarction in the left MCA region (Figure 1-A), and perfusion MRI detected a moderate time delay in the left MCA region. Magnetic resonance angiography (MRA) indicated severe focal stenosis of the M1 segment. Transfemoral cerebral angiography was performed with a contrast medium and 3-dimensional rotational angiography system to visualize the stenosis of the left MCA (Figure 1-B, C). The conventional

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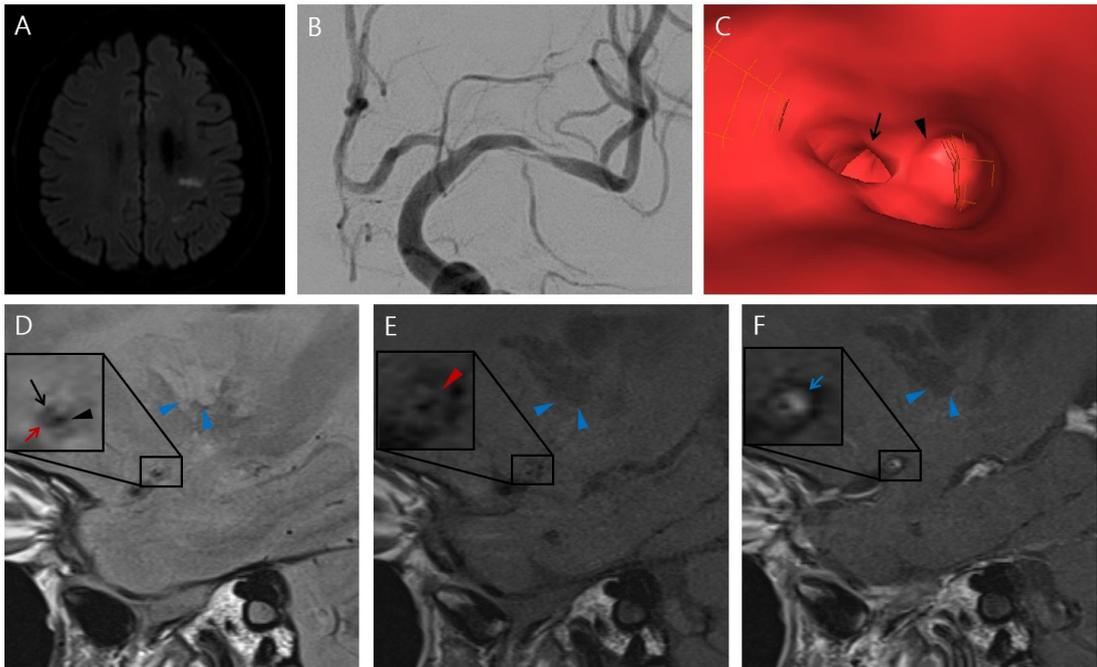


Figure 1. Initial radiographic images of the patient. (A) Axial diffusion-weighted MR image showing a typical ischemic lesion. (B) Digital subtraction angiography revealing the intimal flap and the double lumen. (C) Tridimensional virtual EndoView at the arterial dissection level of left mid M1 from the 3-dimensional reconstruction image presenting true lumen (black arrow) and false lumen (black arrowhead). (D) Proton density sequence showing the dissecting flap (red arrow) and the double lumen, consisting of true (black arrow) and false lumen (black arrowhead). (E) T1-weighted image of HR-MRI showing eccentric wall thickening and atherosclerotic plaques (red arrowhead) without intramural hematoma. (F) Contrast-enhanced T1-weighted image of HR-MRI showing strong enhancement in eccentric atherosclerotic plaques (blue arrow) and the old infarcted lesion (blue arrowheads) associated atherosclerosis.

angiography revealed a flame shaped intimal flap with a double lumen and diffuse narrowing of the mid-portion of the left MCA (Figure 1- B, C).

We performed a high resolution-MRI (HR-MRI) for evaluation of the MCA lesion. The HR-MRI protocol at our institution for MCA stenosis, as described subsequently, was followed for the scan. The 3D proton-density imaging with turbo spin-echo sequences was set with the following parameters: repetition time (TR)/echo time (TE)=2450/37ms, field of view (FOV)=100x100, matrix size=195x256. T1-weighted imaging with turbo spin echo sequences was obtained with the following parameters: TR/TE=670/9.1, FOV=100x100, matrix size=282x256. T1-enhanced imaging with turbo spin echo sequences was obtained with the following parameters: TR/TE=670/9.1, FOV=100x100, matrix size=256x256.

The HR-MRI images were assessed independently by 2 neurologists and 1 radiologist who were blind to the clinical data. Discrepancies were resolved at a consensus meeting.

Although there was no intraluminal hemorrhage on T1 images, the proton-density sequences showed the presence of an intimal flap with a tapered pseudolumen in the MCA and the contrast-enhanced T1-weighted imaging showed enhancing atherosclerotic plaque near the dissecting intimal flap (Figure 1-D, E, F). This atherosclerotic lesion was also the origin of the perforating artery associated with the old corona radiata and basal ganglia infarction lesion.

The diagnosis was extensive MCA dissection with an atherosclerotic plaque. The procedure-related risk from stent insertion was thought to be higher than that from a rupture or an embolic risk. Therefore, we decided on a conservative management with antiplatelet therapy. 40-days later, the patient revisited the hospital complaining of right sided weakness.

DWI showed multiple infarction in the left MCA region (Figure 2 - A). Perfusion MRI detected a large mismatched area in the left MCA region and MRA revealed a more advanced stenotic lesion in the M1 segment of the left MCA

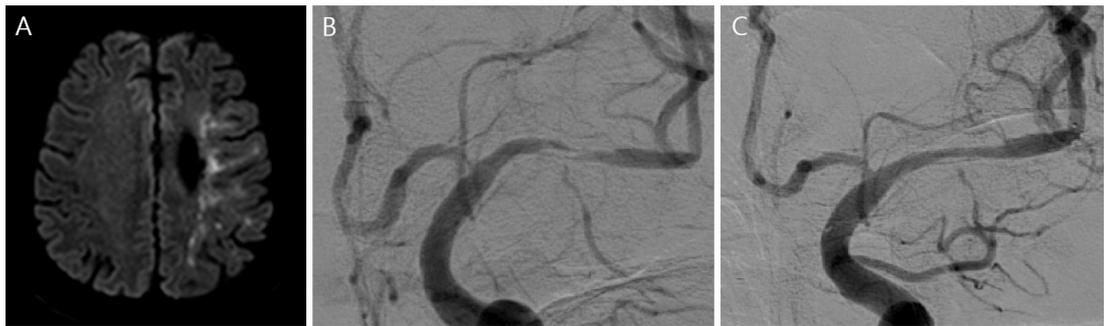


Figure 2. Follow-up radiographic images after 40 days. (A) Axial diffusion-weighted MR image showing a new multiple infarction in the left MCA territory. (B) Digital subtraction angiography representing a more advanced stenotic lesion and a bulging flap. (C) After the intracranial stenting, final angiography presenting widened lumen and narrow gap of flap.

compared to the previous MRA. The follow-up transfemoral cerebral angiography still showed the intimal flap of the left MCA, with increased luminal narrowing of the left MCA (Figure 2 - B).

We decided to perform an intracranial stenting procedure. Stents were positioned across the diseased segments so that they overlapped on each side of the dissected orifice. After the procedure, final angiographic images of the MCA and intracranial circulation revealed a widened lumen and improved flow across the dissection with no embolic sequelae in the distal intracranial circulation (Figure 2 - C). He remained on dual antiplatelet therapy, and was discharged without further complications.

DISCUSSION

Arterial dissections are delineated by sudden disruption of the endothelium, the intima, and the internal elastic lamina with subsequent influx of circulating blood into the media. Results from a previous study indicated that in patients with intracranial artery dissection, thromboembolism is an important cause of subsequent stroke.² Based on these results, antithrombotic drugs such as anticoagulants or antiplatelets were considered as the first-line treatment option. However, antithrombotic treatment might not be useful in some cases of cerebral infarction associated with hemodynamic compromise.³ Previous studies have reported the annual neurologic morbidity rates under long-term medical therapy to be 8–12%.³

There are many associated conditions that might predispose to MCA dissection (or dissections of other cerebral arteries), and some of them are associated with atherosclerosis.⁴ In cases of dissection with atherosclerosis, the intimal

flap might not attach adequately during the healing process, and can cause intimal irregularities. Eventually, a hemodynamic infarction might be induced.

When dissection and atherosclerosis coexist, it is difficult to distinguish the features on imaging. In characterizing dissection, the intimal flap and the double lumen are the most important imaging findings. On the other hand, plaque enhancement on contrast-enhanced T1-weighted imaging is considered the most important characteristic of intracranial atherosclerosis.⁵ In our case, we could confirm that the atherosclerotic plaque was accompanied by the dissecting intimal flap on HR-MRI and transfemoral cerebral angiography. Despite 40 days of antiplatelet therapy, the dissection did not heal at all. In a patient with persistent or progressive symptoms, the space occupied by the loose flap or the fluttering of the flap is an important reason for the aggravation of neurologic symptoms and progression of the hemodynamic brain ischemia. These patients might fail to respond to medical treatment.

In conclusion, in a case of dissection with atherosclerosis, early stage intracranial stenting rather than medical treatment might prevent cerebral infarction.

DISCLOSURE

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Conflict of Interest: Dr. SH Lee performs joint research with CGBio during the conduct of the study.

REFERENCES

1. Chien C, Chang FC, Huang HC, *et al*. Characteristics and Outcomes of Vertebrobasilar Artery Dissection with Accompanied Atherosclerosis. *Cerebrovasc Dis Extra* 2017;7:165-72.
2. Benninger DH, Georgiadis D, Kremer C, *et al*. Mechanism of ischemic infarct in spontaneous carotid dissection. *Stroke* 2004;35:482-5.
3. Xianjun H, Zhiming Z. A systematic review of endovascular management of internal carotid artery dissections. *Interv Neurol* 2013;1:164-70.
4. Asaithambi G, Saravanapavan P, Rastogi V, *et al*. Isolated middle cerebral artery dissection: a systematic review. *Int J Emerg Med* 2014;7:44.
5. Choi YJ, Jung SC, Lee DH. Vessel Wall Imaging of the Intracranial and Cervical Carotid Arteries. *J Stroke* 2015;17:238-55.