Atherosclerosis-Like Spontaneous Middle Cerebral Artery Dissection

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Abstract

Keywords
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► middle cerebral artery occlusion
► vessel wall imaging

The standard of care for treating acute large vessel occlusion is endovascular therapy. The most frequent cause of occlusion is either embolic occlusion or in situ thrombotic occlusion. However, occlusion resulting from intracranial dissection is extremely rare, especially in the middle cerebral artery. Prior to a thrombectomy or endovascular therapy, understanding and interpreting the angiographic findings is crucial for planning the appropriate treatment and preventing complications.

Introduction

Endovascular treatment for acute large cerebral arterial occlusion is proven and is becoming the standard treatment in which cardioembolic occlusion, or in situ thrombotic atherosclerosis occlusion, is the most common cause of occlusion. Uniquely in Asia, occlusion from intracranial atherosclerosis has a higher incidence than in many other regions whereas occlusion from spontaneous intracranial dissection has been less frequently reported, even less at the middle cerebral artery (MCA). It is not easy to distinguish intracranial dissection from more common etiology, atherosclerosis, particularly in an acute setting where decision-making is based on initial, less complex imaging and under time constraints. Acknowledging the cause of the occlusion beforehand may help in planning the procedure and avoiding complications. We present angiographic features during the endovascular treatment as well as magnetic resonance imaging (MRI) of the brain together with MR angiography (MRA) and intracranial vessel wall MRI after the procedure in acute spontaneous MCA dissection (MCAD) to help better recognize these uncommon etiologies from more common atherosclerosis.

Case Presentation

A 62-year-old woman with a history of dyslipidemia and hypothyroidism presented to an outside hospital with a sudden onset of speech disturbance and right-sided weakness while talking with her family. There is no record of any recent infection or head trauma. She was brought to a nearby hospital, and an initial neurological examination revealed global

aphasia, left-eyed deviation, right facial weakness, and right hemiplegia. An initial computed tomography (CT) brain scan showed no intracerebral hemorrhage. An acute ischemic stroke was diagnosed, and intravenous thrombolysis was administered 1 hour after the onset. She was transferred to our institute after no improvement was observed. On physical examination upon arrival 10 hours after the onset, blood pressure was 164/78 mm Hg and the heart rate was 54 beats per minute with normal systemic examination. A neurological examination revealed alertness but with global aphasia, left eye deviation, and right hemiplegia; the National Institutes of Health Stroke Scale was 14. MRI and MRA brain scans were performed which showed diffusion restriction at the caudate, putamen, insular, corona radiata, and parietal cortex of the left cerebral hemisphere. The Alberta Stroke Program Early CT Score from MRI brain was 6/10. The MRA showed occlusion at the proximal left M1 segment of the MCA.

Endovascular treatment under general anesthesia proceeded after discussing with and receiving consent from the family. The first left internal carotid artery angiogram showed severe stenosis at the left proximal MCA with decreased anterograde flow (►Fig. 1). Acute MCA occlusion from atherosclerosis was then first considered. A stent retrieval technique was first attempted with no ability to recanalize the stenotic vessel, and then an uneventful balloon angioplasty was performed, resulting in more anterograde flow to the distal vessel (not shown). A repeated left internal carotid angiogram showed no occlusion or impaired anterograde flow after which the procedure was stopped. However, we were unsure of the pathologic vessel, whether it was atherosclerotic occlusion or other pathologic condition; therefore, an MRI and MRA including vessel wall imaging technique of the brain were done the next day to investigate the suspected vessel wall of the left MCA and found an intimal flap at the left proximal MCA (►Fig. 2). The diagnosis of spontaneous MCAD was then made. The laboratory stroke panel, which included a thyroid function test, revealed


Fig. 1 Left internal carotid artery (ICA) cerebral angiogram in anteroposterior (AP) view of our patient demonstrated short segment occlusion at the middle part of the left M1 middle cerebral artery (MCA). Abnormal linear contrast filling just superior to the occlusive point is suspected false lumen of the left MCA dissection (arrow).

elevated levels of low-density lipoprotein at 3.18 mg/cm^3, as well as decreased levels of free T3 and free T4, and elevated levels of thyroid-stimulating hormone at 43.46 μIU/cm^3. Although the clinical picture was stabilized throughout the 5 days in the stroke unit, but from the arterial spin labeling MR perfusion, which showed decreased perfusion through the left MCA territory (►Fig. 3), we thought antiplatelet therapy may not effectively prevent further stroke events, with severe narrowing of the proximal MCA; therefore, permanent stenting was discussed with the family and the procedure was then done with the deployment of a Solitaire AB stent 3 × 30 mm across the stenotic part from the proximal M1segment of the MCA to proximal part of the inferior division of the MCA (►Fig. 4). The procedure result was
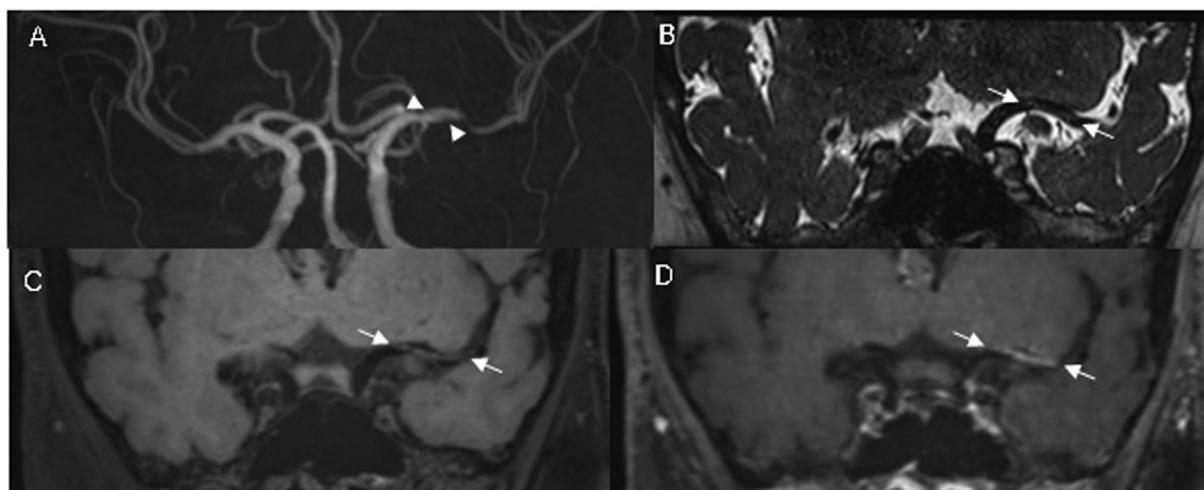
Fig. 2 Time-of-flight magnetic resonance angiography (MRA) of the brain (**A**) revealed focal occlusion of the left M1 middle cerebral artery (MCA) with subtle flap (arrowhead) proximal to the occlusive point. Intracranial vessel wall imaging with coronal T2-weighted vessel wall (VW)-magnetic resonance imaging (MRI) (**B**), coronal T1-weighted VW-MRI (**C**), and coronal contrast-enhanced T1-weighted VW-MRI (**D**) are clearly defined long enhancing intimal flap (arrow) along the left M1 MCA.
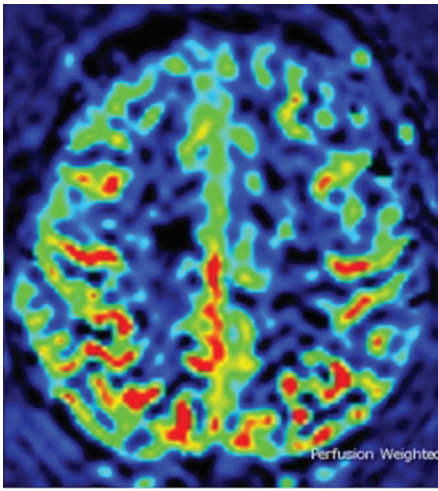
Fig. 3 Axial images of the three-dimensional (3D) arterial spin labeling (ASL) magnetic resonance (MR) perfusion of our patient found hypoperfusion of the left frontoparietal region in the left middle cerebral artery (MCA) territory.

uneventful and there were no any immediate and delayed complications, and she has been on double antiplatelet therapy for 3 months followed by a single antiplatelet since then. One month after the procedure, her global aphasia improved to motor aphasia and right-side motor power was persistent at Medical Research Council's scale of muscle power grade I.
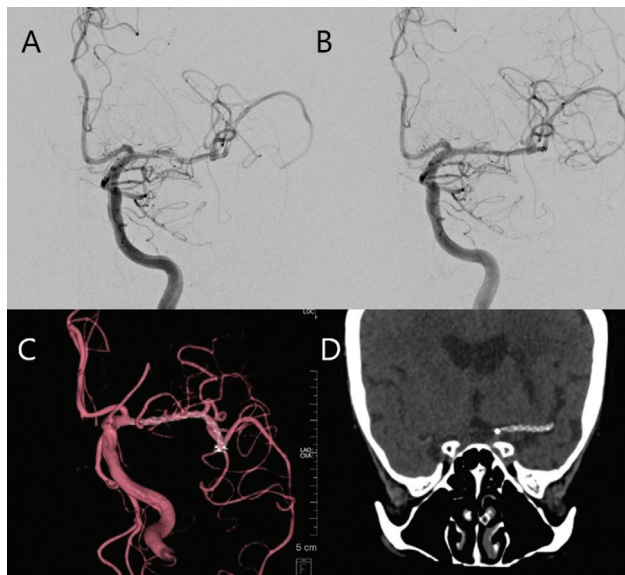
Fig. 4 Comparison between prestenting (**A**) and poststenting (**B**) of the left internal carotid artery (ICA) cerebral angiogram in antero-posterior (AP) view showed significant increase in caliber of the left M1 middle cerebral artery (MCA) with disappearance of the suspected false lumen and rapid flow to the left cortical MCA branches after stent placement. Poststenting three-dimensional (3D) cerebral angiogram in caudal view (**C**) and noncontrasted computed tomography (CT) study in coronal view (**D**) demonstrated location of the stent which covers the entire left M1 MCA and the proximal part of inferior division of left M2 MCA.

Discussion

Spontaneous intracranial dissection is extremely rare. Most cases of spontaneous dissection involve extracranial structures, such as the internal carotid artery. There are a small number of intracranial dissection cases reported in which most case reports are from the intracranial vertebral artery and even fewer from the anterior cerebral artery or MCA.

Asaithambi et al[1] conducted a systematic study in 2014 that was the most up to date and extensive, encompassing case series and case reports. Since then, very few cases have been reported.

There were 61 cases reported from the Asaithambi et al review, with a mean age of 44.16 years with majority of them (62.3%) being men. Surprisingly, 78.7% of reported patients were Asian. What is more intriguing is that most of the case reports following the Asaithambi et al's study originated from Asia.

The intracranial dissection can be manifestations of either nonfocal symptoms in which headache is the most common followed by seizure and tinnitus or focal symptoms such as stroke. In addition, 19.7% of cases may be asymptomatic.

Ischemic strokes account for 68.9% of stroke presentations, while hemorrhagic strokes are less common. It can occasionally manifest in both hemorrhagic and ischemic stroke concurrently.

Hemorrhagic stroke may manifest as subarachnoid hemorrhage or parenchymal bleeding. Additionally, in approximately 37.7% of cases,[1] a dissecting aneurysm was discovered, and more than half of those cases resulted in a hemorrhagic stroke.

Most of the pathogenesis behind intracranial dissection is still unclear. It could be linked to various connective tissue disorders, systemic infection, inflammation, atherosclerosis, or previous head trauma. Several studies[2–4] proposed that trauma may account for the MCA's M1 segment, given its proximity to the sphenoid wing's posterior boundary. Even though there was no prior record or evidence of any related conditions in our patient.

According to a report by Park et al,[5] the incidence of MCAD presenting with acute large vessel occlusion is approximately 2.4%. Technically, in acute large vessel occlusion, selecting candidate for mechanical thrombectomy is based on CT or MRI of the brain to determine the extent of ischemic brain and/or CT angiography (CTA) or MRA to confirm the location of the large vessel occlusion. During the planning stage prior to endovascular treatment, the cause of occlusion is typically determined to be embolic occlusion or atherosclerosis based on the mode of stroke presentation, physical examination, past medical history, and findings from CT or MRI and CTA or MRA. The etiology of the occlusion influences the planning stages of the procedure, including the selection of the device. However, the underlying cause of occlusion is not always known at this stage. Frequently, it is discovered after the initial angiographic scan or even after the endovascular procedure. Specific MRI technique as three-dimensional magnetization-prepared rapid acquisition gradient-echo sequences mentioned by Kwak et al[6] or Kato el al[7] use standard T2-weighted MRI sequencing
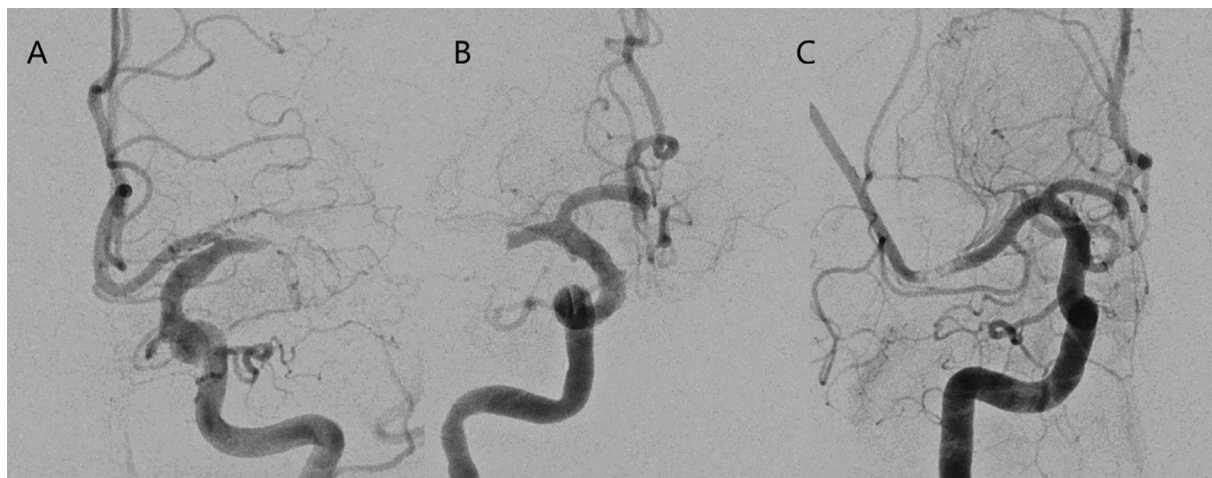
Fig. 5 Cerebral angiogram in anteroposterior (AP) view of the other patients with other causes of arterial occlusion (**A–C**); (**A**) Left internal carotid artery (ICA) angiogram demonstrated taper contrast filling until the point of obstruction, which is characteristic of occlusive plaque; (**B**) Right ICA angiogram demonstrated abrupt occlusion of the right middle cerebral artery (MCA), which is characteristic of occlusive thrombus; (**C**) Right ICA angiogram demonstrated occlusion of the right MCA with "tram-track" appearance, which is characteristic of nonocclusive thrombus.

in the coronal plane parallel to the sylvian vallecula with grayscale reversal during postprocessing which would enhance the sensitivity and specificity for detecting the intracranial dissection. However, this method could not be performed routinely and might be missed if routine CTA or MRA were not highly suspicious.

The angiogram suggestive characteristic of in situ thrombotic occlusion or atherosclerosis appears tapered or truncal-type occlusion or there is atherosclerotic feature in other intra- or extracranial vessels, whereas the angiogram suggestive characteristic of embolic occlusion appears as cutoff, clot meniscus, or tram-track sign (►**Fig. 5**). However, even these findings found differentiating the etiology difficult.

For intracranial dissection angiography, specifically the MCA, there are only a scattering of reports describing the angiographic findings. Most cases reported with advanced CTA, vessel wall MRI, and MRA, which cannot be extensively performed in an acute setting as we mentioned before, in addition, some of them were retrospectively reviewed.

Intracranial dissection is characterized by angiographic findings such as the intimal flap, pearl-and-string sign, and double lumen, with the pearl-and-string sign being the most prevalent[8] and most of them not being total occlusion. However, these angiographic characteristics are not always seen and also if there is complete occlusion, early detection of this dissection is extremely challenging.

Upon careful examination of the angiography in our case, we saw a distinct double lumen characteristic in the proximal region of the left MCA. This feature is distinguishable from the narrowing pattern often associated with atherosclerotic stenosis. Furthermore, atherosclerotic characteristics have not been observed in other cerebral arteries either.

Therefore, attempting to recognize different angiographic findings in dissection from atherosclerosis is essential. Bond et al[9] have recently suggested imaging criteria that can assist in diagnosing intracranial dissection.

If endovascular treatment is planned, endovascular treatment may necessitate passing a microcatheter or microguidewire blindly across the occlusion; this could be dangerous if these devices enter a false lumen and could result in devastating outcomes such as vessel perforation or rupture. Therefore, procedural step planning, device selection, and navigation of all devices are crucial for achieving the desired outcome and avoiding complications.

No established protocol or conventional treatment guidelines are available for this rare illness. The MCAD management must make decisions for each manifestation.[10] When the MCAD presents as ischemic stroke, antithrombotics or anticoagulants have been used for secondary prevention, as shown by Asaithambi et al's review with no randomized controlled studies. However, there is a significant probability of developing a recurrent ischemic stroke. In cases of acute occlusion, intravenous thrombolysis may be used. However, like large vessel occlusion caused by other factors, endovascular treatment is more effective in restoring blood flow and likely leads to improved clinical outcomes. Additionally, permanent stenting is often necessary in these cases.

Conclusion

The occurrence of acute occlusion caused by MCAD is infrequent but crucial to differentiate from in situ thrombotic occlusion or atherosclerosis, which is more prevalent, especially among Asian individuals. It is crucial to distinguish the various angiographic characteristics of these two disorders to prevent complications and achieve favorable clinical results for endovascular treatment, as the appropriate therapeutic approach is likely to differ.

Conflict of Interest
None declared.

References

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