

Short communication

Two cases of spontaneous middle cerebral arterial dissection causing ischemic stroke

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Abstract

Spontaneous middle cerebral arterial dissection (MCAD) is a rare cause of ischemic stroke. We report two cases of isolated spontaneous MCAD causing ischemic stroke. MCAD should be considered when a young patient has a middle cerebral artery territory infarct with stenosis at the origin of the middle cerebral artery. We discuss noninvasive radiological techniques for the diagnosis of MCAD. © 2006 Elsevier B.V. All rights reserved.

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1. Introduction

Cervicocephalic arterial dissection must be considered in the differential diagnosis of ischemic stroke in young patients. Cervicocephalic arterial dissections usually involve the internal carotid and vertebral arteries, especially the extracranial portions. Middle cerebral arterial dissection (MCAD) is rare [1–3], and isolated spontaneous cases of MCAD are even seldom reported [2]. The clinical and radiological aspects of MCAD are poorly understood.

While digital subtraction angiography (DSA) has been the gold standard for identifying cervicocephalic arterial dissections [4,5], there is no consensus as to which angiographic tool is most useful in the diagnosis of MCAD. DSA is an invasive procedure with possible complications, so alternative noninvasive tools must be sought. We report two cases of isolated spontaneous MCAD causing ischemic stroke and discuss their clinical features and radiological characteristics, focusing on noninvasive angiographic tools such as magnetic resonance angiography (MRA) and computed tomographic angiography (CTA).

2. Case report

2.1. Patient 1

A 24-year-old male developed aphasia 3 weeks before admission and was referred to our department for further evaluation. There, he experienced motor-type aphasia and mild weakness in his right arm and leg. Six hours before the onset of the focal symptoms, he felt a pain in the left ocular area. His previous history was unremarkable, with no history of migraine, head trauma, or vigorous neck movement. He had fallen several times while skiing about 1 week before the onset of his headache, but he denied any direct head or neck trauma. On neurological examination, except for his difficulty in finding words, the other neurological deficits had disappeared. The homocysteine and C-reactive protein levels were 7.5 μmol/L (normal range, 5.0 to 15.0 μmol/L) and 0.03 mg/dl (normal range, 0.02 to 0.80 mg/dl), respectively. The results of other laboratory tests and cardiology evaluations were unremarkable. Warfarin was prescribed to prevent thromboembolism.

The T2-weighted images, which were taken on admission at 3 weeks after symptom onset, showed multiple scattered infarcts of various sizes in the left middle cerebral artery

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(MCA) territory (Fig. 1A). There was no evidence of intramural hematoma on the T1- and T2-weighted images. DSA showed an intimal flap and double lumen in the left proximal MCA (Fig. 1B). A double lumen was also visible in the three-dimensional time-of-flight (3-D TOF) MRA images (Fig. 1C), while only smooth narrowing of the artery was observed in the 3-D surface-rendered CTA images (Fig. 1D). The thin-sectioned axial source MRA images showed an intimal flap (Fig. 1E), whereas only arterial narrowing was seen in the maximum intensity projection (MIP) images from CTA (Fig. 1F). The MCA dissection began 9.10 mm from the posterior margin of the sphenoid wing on sagittal computed tomography using the cross-link technique (Fig. 3A). All other intracranial and extracranial cervicocephalic arteries were intact in these angiographic studies.

2.2. Patient 2

A 50-year-old female experienced right-side hemiplegia and aphasia 2 months previously. Two days before the onset

of the hemiplegia, she suffered an abrupt, continuous, pulsatile headache in the left temporoparietal area, which she had never experienced previously. She was taken to a local hospital and was managed for acute cerebral infarction, which was demonstrated by brain magnetic resonance imaging (MRI). She was referred to our department for further evaluation. Her previous history included a 2-year history of diabetes mellitus but was negative for migraine, head trauma, or vigorous neck movement. On neurological examination, her comprehension and ability to repeat were good, but she showed paraphasic naming and had difficulty finding words. Weakness and sensory change were found on the right side, including the face. Other cranial and cerebellar functions were intact. The homocysteine and C-reactive protein levels were 9.1 $\mu\text{mol/L}$ and 0.71 mg/dl, respectively. The results of other laboratory studies and the cardiology work-up were unremarkable. Aspirin was prescribed, and her symptoms did not worsen over a 2-year follow-up period.

A large subcortical infarct with a small cortical infarct was seen on diffusion-weighted images (DWI), which were

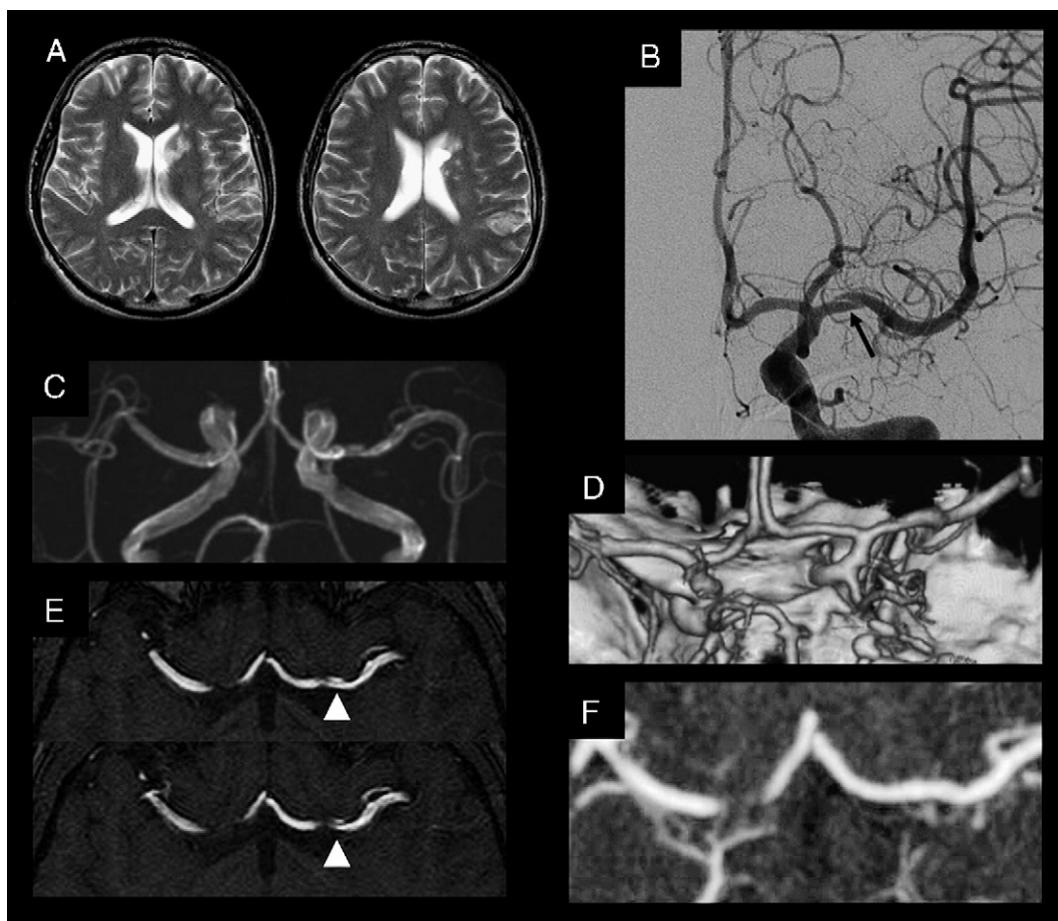


Fig. 1. A. The T2-weighted image shows multiple scattered infarcts of various sizes in the left cortex and subcortex. B. DSA reveals an intimal flap and double lumen in the left proximal MCA (arrow). C. The 3-D time-of-flight MRA shows a double lumen in the left proximal MCA. D. The 3-D CTA image shows smooth narrowing of the MCA. E. The thin-sectioned axial source MRA images document an intimal flap (arrowhead). F. The MIP images from CTA show mild MCA stenosis. DSA: digital subtraction angiography; MCA: middle cerebral artery; MRA: magnetic resonance angiography; CTA: computed tomographic angiography; MIP: maximum intensity projection.

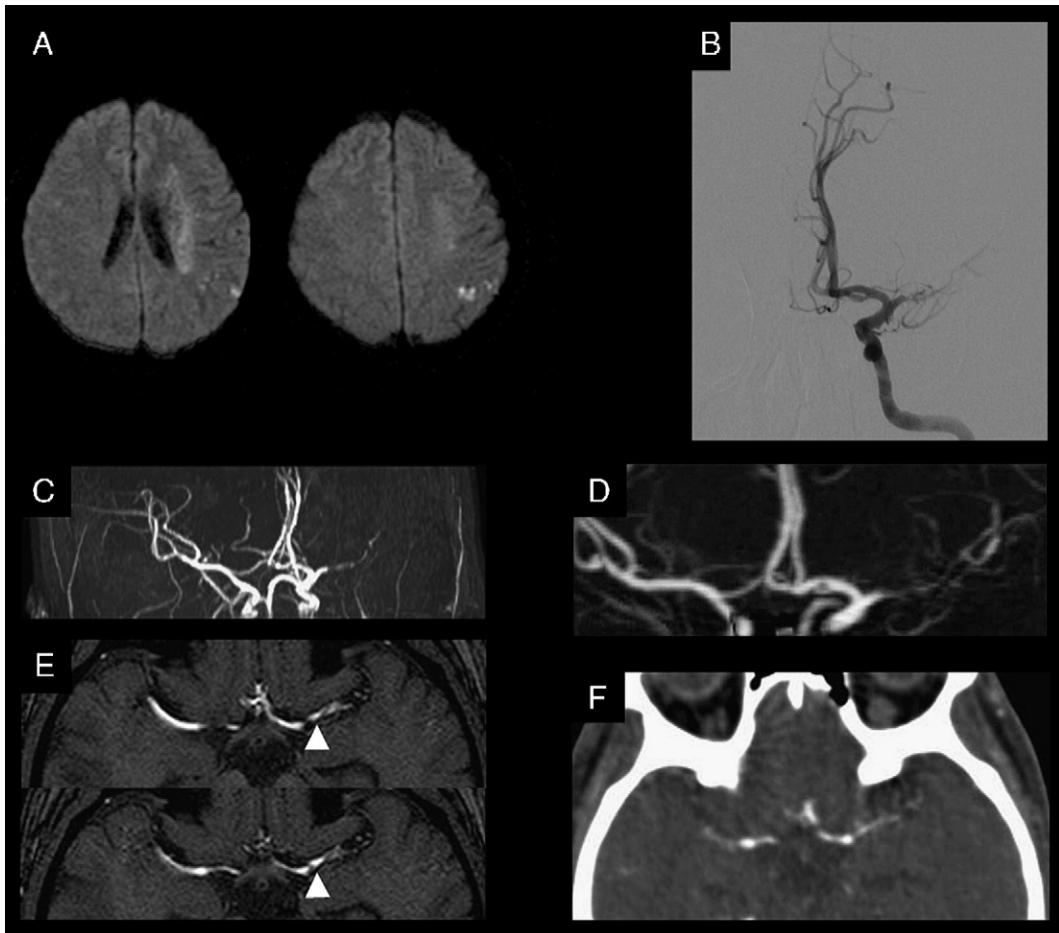


Fig. 2. A. A diffusion-weighted image shows the development of an internal border-zone infarct with an intra-infarct hemorrhage and additional small cortical infarcts. B. DSA shows near occlusion and a suspicious double lumen in the left proximal MCA. C. The 3-D time-of-flight MRA images reveal irregular stenosis. D. The 3-D CTA images show irregular stenosis. E. Thin-sectioned axial source MRA images document an intimal flap and small intramural hematoma at the origin of the MCA (arrowhead). F. The MIP images from CTA show irregular stenosis. DSA: digital subtraction angiography; MCA: middle cerebral artery; MRA: magnetic resonance angiography; CTA: computed tomographic angiography; MIP: maximum intensity projection.

taken at the time of onset (Fig. 2A). The T1- and T2-weighted images did not show any definite intramural hematoma in the left proximal MCA. DSA showed near occlusion and a suspicious double lumen in the left proximal MCA (Fig. 2B). Irregular stenosis was visible in 3-D TOF MRA and 3-D CTA (Fig. 2C and D). The thin-section axial source MRA images showed an intimal flap and a small intramural hematoma at the origin of the MCA (Fig. 2E), whereas the MIP images from CTA showed only irregular stenosis (Fig. 2F). The MCA dissection began 4.13 mm from the posterior margin of the sphenoid wing on sagittal computed tomography conducted using the cross-link technique (Fig. 3B). All other intracranial and extracranial cervicocephalic arteries were intact in these angiographic studies.

3. Discussion

MCA territory infarctions have several etiologies. Atherosclerosis or embolisms are the main causes, but hematologic disorders and hereditary or other vascular problems, including

arterial dissection, must be considered in stroke in younger people. The clinical features, stroke mechanism, and radiological characteristics of MCAD are still not fully understood. Spontaneous MCAD causing ischemic stroke is seldom reported. To our knowledge, there have been fewer than 20 cases, and these are often associated with internal carotid arterial dissection or trauma [1–3]. MCAD might be under-diagnosed; in our stroke database, of 940 patients with cerebral infarcts within the MCA distribution on diffusion-weighted images, two patients (0.21%) were diagnosed as having MCAD. The prevalence was even higher among the patients who underwent DSA (1.3% of 150 patients).

Here, we report two cases of spontaneous isolated MCAD causing ischemic stroke. Neither patient had any clinical, laboratory, or radiological evidence of other well-known factors, such as migraine, fibromuscular dysplasia, cystic medial necrosis, intimal fibroelastic aberration, atherosclerosis, Marfan's syndrome, or Ehlers–Danlos syndrome, that may cause intracranial arterial dissection. They also had no history of head trauma or vigorous neck movement. Most



Fig. 3. The relationship between the dissected MCA segment and the neighboring bony structure. The origin of the MCAD is located (A) 9.10 and (B) 4.13 mm from the posterior margin of the sphenoid wing on sagittal computed tomography using the cross-link technique, in patient 1 and 2, respectively. MCA: middle cerebral artery; MCAD: middle cerebral arterial dissection.

reported dissections involve the extracranial portion of the carotid and vertebral arteries, which are much more vulnerable to dissection than are other sites. This may be owing to the greater mobility of the extracranial internal carotid and vertebral arteries and to the potential for them to be damaged by contact with bony structures, such as the cervical vertebrae or styloid process. Although our cases occurred spontaneously, trauma might play a role in the development of arterial dissection. In both patients, the MCAD began at the site nearest the posterior margin of the sphenoid wing, with a distance of less than 1 cm, suggesting that MCAD is associated with contact between the MCA and the bony edge. This is supported by a recent report of traumatic MCA occlusion [6]. Mobbs and Chandran [6] reviewed the pathogenesis based on the anatomy of the proximal MCA segment and suggested that the part of the MCA most vulnerable to minor trauma is the initial segment of the MCA, as seen in our cases. Therefore, isolated MCA stenosis at the origin of the MCA should undergo further vascular studies to rule out the possibility of arterial dissection.

In this study, we confirmed MCAD using DSA because it is still considered the gold standard by which to diagnose

arterial dissection [4,5]. In our patients, DSA showed a double lumen, which is one of the pathognomonic findings in arterial dissection. The demonstration of a surrounding hematoma by MRI [7] or CTA [8] is another pathognomonic finding of arterial dissection. In our patients, no intramural hematoma was detected on the T1- or T2-weighted MRI images, which were taken 3 weeks and 2 months after the onset of the neurologic problems, respectively. The failure to detect a hematoma in our cases might be caused by the small size of the lumen and surrounding hematoma in the MCA, as well as the delay before evaluation.

By contrast, in our cases, an intimal flap and double lumen were documented by MRA despite the time since symptom onset. Although MRA is regarded as a good alternative and noninvasive method of imaging for cervicocephalic arterial dissection [9,10], its role is still unknown in MCAD. In our cases, MRA, especially axial source imaging, was a sensitive tool capable of detecting an intimal flap and double lumen, a definitive finding in dissection, whereas the MIP images from CTA failed to provide any additional diagnostic findings. The lower sensitivity of CTA axial thin reconstructed images compared with the

sensitivity of axial source MRA images may be caused by the low resolution of the CTA technique. The MRA source images may be particularly important for detecting MCAD for two reasons. First, because the MCA and its intimal flap run horizontally, the axial source MRA images may provide the optimal cut of view to delineate the course of the MCA. Second, while the pathognomonic features of dissection, such as an intimal flap or a double lumen, are detected in fewer than 10% of dissected arteries by using DSA [11], MRA may directly visualize an intramural thrombus or neighboring tissue [10]. Although it has not been determined which form of angiography is more helpful for the diagnosis of MCAD, as it is very rare, our cases suggest that axial source TOF MRA images are a sensitive alternative as a noninvasive procedure.

The infarct pattern on DWI may be associated with the pathogenic mechanism of stroke. Large subcortical infarcts and multiple cortical infarcts were found in our patients. However, it is difficult to suggest a mechanism of MCAD now. Further perfusion and transcranial Doppler studies designed to detect microemboli are needed in more patients.

In summary, we report two cases of spontaneous MCAD causing ischemic stroke. MCAD should be considered if a patient has an MCA territory infarct with stenosis at the origin of the MCA following a sudden ipsilateral headache. An axial source MRA image may be useful for detecting the intimal flap in MCA dissection.

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References

- [1] Chaves C, Estol C, Esnaola MM, Gorson K, O'Donoghue M, De Witt LD, et al. Spontaneous intracranial internal carotid artery dissection: report of 10 patients. *Arch Neurol* 2002;59:977–81.
- [2] Ohkuma H, Suzuki S, Shimamura N, Nakano T. Dissecting aneurysms of the middle cerebral artery: neuroradiological and clinical features. *Neuroradiology* 2003;45:143–8.
- [3] Sharif AA, Remley KB, Clark HB. Middle cerebral artery dissection: a clinicopathologic study. *Neurology* 1995;45:1929–31.
- [4] Guillou B, Levy C, Bousser MG. Internal carotid artery dissection: an update. *J Neurol Sci* 1998;153:146–58.
- [5] Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. *N Engl J Med* 2001;344:898–906.
- [6] Mobbs RJ, Chandran KN. Traumatic middle cerebral artery occlusion: case report and review of pathogenesis. *Neurol India* 2001;49:158–61.
- [7] Oelerich M, Stogbauer F, Kurlemann G, Schul C, Schuierer G. Craniocervical artery dissection: MR imaging and MR angiographic findings. *Eur Radiol* 1999;9:1385–91.
- [8] Kurokawa Y, Yonemasu Y, Kano H, Sasaki T, Inaba K. The usefulness of 3D-CT angiography for the diagnosis of spontaneous vertebral artery dissection—report of two cases. *Comput Med Imaging Graph* 2000;24:115–9.
- [9] Levy C, Laissy JP, Raveau V, Amarenco P, Servois V, Bousser MG, et al. Carotid and vertebral artery dissections: three-dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. *Radiology* 1994;190:97–103.
- [10] Provenzale JM. Dissection of the internal carotid and vertebral arteries: imaging features. *Am J Roentgenol* 1995;165:1099–104.
- [11] Houser OW, Mokri B, Sundt Jr TM, Baker Jr HL, Reese DF. Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. *Am J Neuroradiol* 1984;5:27–34.