

Simultaneous Onset of Anterior and Middle Cerebral Artery Dissections with an Old Vertebral Artery Dissection

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Multiple arterial dissections in the anterior circulation with simultaneous onset are extremely rare. We report a patient with infarctions caused by simultaneous arterial dissections in the right anterior cerebral artery and the left middle cerebral artery and discuss the characteristic feature of this vascular disorder. A 53-year-old woman presented with a severe headache and a mild aphasia. Magnetic resonance imaging revealed multiple acute cerebral infarctions in the left temporal and right frontal lobes. The initial angiographic findings revealed arterial dissections of the anterior cerebral, left middle cerebral, and right vertebral arteries. The follow-up angiographic examination found improvement of the stenosis in both the anterior cerebral and middle cerebral arteries. We have concluded that the lesion of the vertebral artery was not in an acute stage, because no interval change was seen during the radiologic evaluation. She underwent conservative therapy, and her symptoms disappeared. Multiple arterial dissections are rare, especially those developing simultaneously in different arteries. This is the first case of multiple arterial dissections of the different arteries in the anterior circulation manifesting cerebral infarction simultaneously. **Key Words:** Anterior cerebral artery—cerebral infarction—intracranial arterial dissection—middle cerebral artery—simultaneous onset.

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Spontaneous intracranial arterial dissection (IAD) predominantly occurs in the posterior circulation and to men in the fourth or fifth decade of life, and it causes subarachnoid hemorrhage and/or cerebellar or brainstem infarction with severe pain in the neck and occipital area.^{1,2} Multiple lesions constitute 10% of IADs, which are mainly located in the bilateral vertebral arteries (VAs). Only 6 cases of intracranial multiple arterial dissections involving the anterior circulation have

been reported.⁴⁻⁹ Among them, 3 cases presented with dissections in the vertebral artery and anterior circulation⁴⁻⁹; therefore, lesions within the anterior circulation are quite rare. In addition, there have only been 2 cases with simultaneous onset^{7,8}; however, both of them presented with dissections in the adjacent arteries. Therefore, the simultaneous onset of multiple arterial dissections in completely separate arteries in the anterior circulation has not been reported.

We report a patient with infarctions caused by simultaneous arterial dissections in the right anterior cerebral artery (ACA) and the left middle cerebral artery (MCA), and discuss the characteristic features of this vascular disorder.

Case Report

A 53-year-old woman with a history of hypertension who had been taking an angiotensin-receptor antagonist

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for 5 years was sitting in a conference when she suffered a sudden onset of severe headache, mainly in the left temporal region, that she had never experienced. She was referred to our hospital 5 days later with a persistent dull but not acute headache. On admission, she presented with mild motor aphasia without muscle weakness or sensory impairment. Diffusion-weighted magnetic resonance (MR) imaging scans revealed infarction in the right medial frontal lobe and the left temporal lobe (Fig 1, A and B). Time of flight (TOF) MR angiography revealed dilatation and stenosis, so-called pearl and string sign, of the lower trunk of the left M2 segment and in the right A2 segment. In addition, the lower trunk of the left M2 was shown as double lumens on the source images of TOF (Fig 1, C and D). In addition, a fusiform dilatation with intimal flap was found in the right VA (Fig 2, A and B). Internal carotid artery angiography revealed narrowing of the initial part of the right A2 segment (Fig 3, A), stenosis and dilatation of the lower trunk of the left M2 segment, and occlusion of its branch (M3) that appeared on the following angiographic examination (Fig 4, A and B). Right vertebral angiography revealed dilatation of the right VA (Fig 2, A, B). A biochemical analysis including the erythrocyte sedimentation rate and autoantibodies found no abnormalities. Together with the history and neuroradiographic findings, we diagnosed the patient with multiple arterial dissections resulting in cerebral infarction. She underwent conservative treatment with antihypertensive drugs but not anticoagulation, antiplatelets, or corticosteroids. Progression of the narrowing of the A2 portion of the right ACA and recanalization of the M3 portion of the left MCA were shown on the angiograms obtained 7 days after the onset (Fig 3, B and Fig 4, B). Complete remission of the lesions was revealed on angiogram taken 3 months later (Fig 3, C and Fig 4, C). Through the clinical

course of the patient, no change was seen in the dilatation of the right VA (Fig 2, A-D); therefore, we conclude that the lesion of VA was not in an acute stage and was incidentally found. Taken together, we diagnosed multiple arterial dissections of the right ACA and left MCA causing multiple cerebral infarctions in the corresponding territories. Her symptoms disappeared and she returned to normal functioning.

Discussion

Multiple spontaneous IADs involving anterior circulation are extremely rare, with only 6 reported cases.⁴⁻⁹ Among them, 4 cases were diagnosed as separate lesions with certain interval ranging from 62 days to 27 months between the onsets, and there have been only 2 cases with simultaneous onset of different dissections. However, the arterial lesions in these 2 cases were located closely at the adjacent regions of the same artery—such as A1 and accessory MCA and A2 and A3—and the possibility of the continuous single lesion cannot be excluded. The present case is apparently multiple dissections of the right ACA and the left MCA with simultaneous occurrence supported by not only the single clinical history of the headache but also the neuroradiologic findings of acute cerebral ischemia/infarction in the both arterial territories together with dynamic radiologic changes, although she did not show and neurologic symptoms caused by the ACA dissection. In addition, we diagnosed the right VA dissection as being chronic, because this lesion had not changed through the clinical course and there was no evidence of acute stage on diverse examinations. Therefore, the present case is the first report of multiple

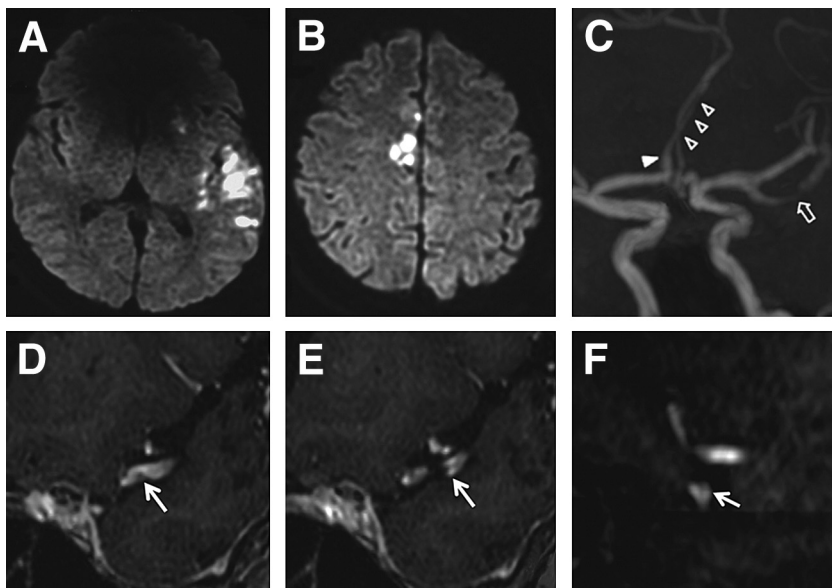


Figure 1. Diffusion-weighted magnetic resonance imaging scans on admission (A, B) revealing multiple acute brain infarction as high-intensity areas in the territories of the left middle cerebral artery (MCA) and right anterior cerebral artery (ACA). A 3-dimensional reconstructed magnetic resonance angiographic scan (C) revealed irregular signal attenuation at the lower trunk of the M2 portion of the left MCA (empty arrow) and dilatation (arrowhead) and stenosis of the A2 portion of the right ACA (empty arrowheads). The partition image of the time of flight angiogram (D; E, coronal view; F, sagittal view) revealed double lumen and intimal flap in the lower trunk of the left M2 (arrows).

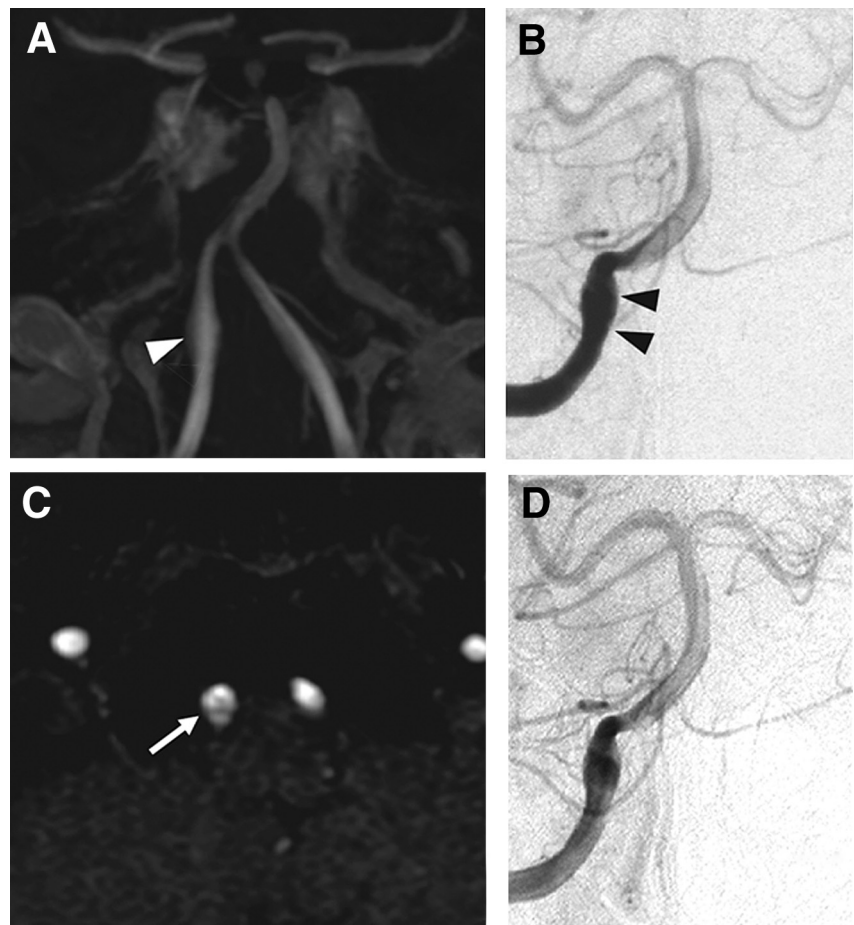


Figure 2. Magnetic resonance angiographic scan revealing the fusiform dilatation (arrowhead, A) of the V4 segment of the right vertebral artery; an intimal flap (arrow, C) was found of the same region on the source time of flight images. Anteroposterior views (B, D) of the right vertebral angiograms revealed aneurysmal dilatation of the vertebral arteries (arrowheads) on admission (B), which did not show any interval changes through the 3-month follow-up period (D).

spontaneous IADs of the completely separate arteries in the anterior circulation with simultaneous onset.

Arterial dissection is defined as a dissection of the sub-intimal-medial or medial-adventitial wall of the vessel accompanied by an intimal tear. The etiology of arterial dissection includes atherosclerosis, trauma, iatrogenic, pregnancy, fibromuscular dysplasia, inflammatory diseases, connective tissue diseases, and other congenital

disorders of the vascular wall. It mostly occurs in the carotid and renal arteries, rarely in the coronary and visceral arteries, and may or may not include aortic dissections.³ Our patient did not have any underlying diseases, such as connective tissue disease or congenital disorders of the vascular wall. However, the simultaneous occurrence of arterial dissections and the incidentally found VA dissection may imply a latent vulnerability of arteries in

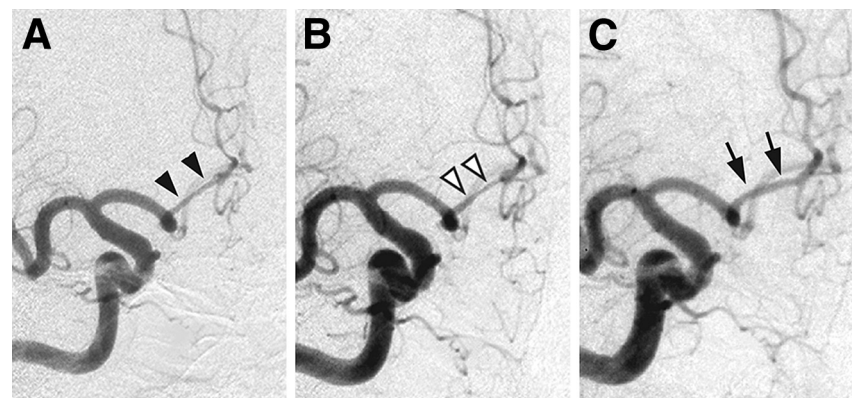


Figure 3. Oblique views (A-C) of the right internal carotid angiogram. The angiogram on admission (A) revealed narrowing of the right A2 segment (arrowheads). The angiogram 1 week later (B) revealed progression of the narrowing of the lesion (empty arrowheads), and the images 3 months after onset (C) show resolution of the narrowing (arrows).

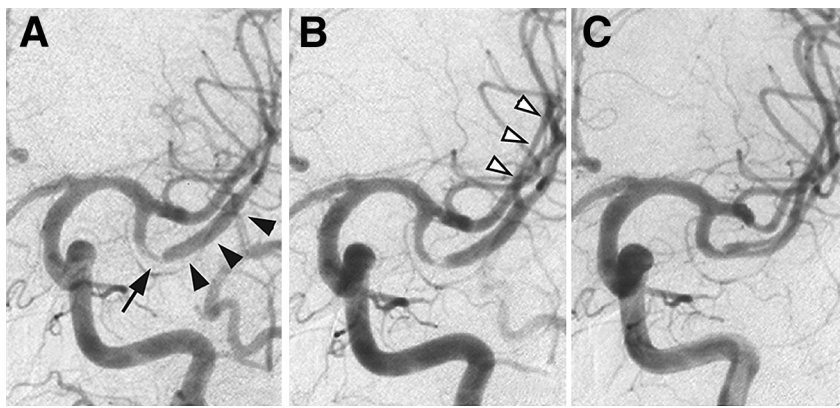


Figure 4. Anteroposterior views (A-C) of the left internal carotid angiograms on admission (A) showing a narrowing (arrow) and dilatation (arrowheads) of the left M2 segment. The angiograms 1 week later (B) reveal recanalization of the branch, which is probably responsible for the left temporal ischemia/infarction (open arrowheads). Angiograms 3 months after onset (C) show resolution of the narrowing and dilatation of M2 segment.

this patient. The underlying mechanism of the IAD is still unknown; however, the sudden disruption of the internal elastic lamina and/or intramural hemorrhage caused by the rupture of the vasa vasorum or vessels formed in response to medial necrosis has been proposed.¹⁰ In our patient, an abrupt change in blood pressure might have provided the opportunity for simultaneous arterial dissections in the separate arteries, but such evidence was not proven. Primary angiitis of the central nerve system and reversible cerebral vasoconstriction syndrome should also be taken into account as a differential diagnosis for this case. Because MR angiography showed the double lumen of left M2 segment, and because her symptoms improved without immunosuppressive therapy (such as corticosteroids or cyclophosphamides), we considered these diseases unlikely.

With regard to treatment strategy, arterial dissections occurring at the major arterial trunk, such as the internal carotid artery, M1, or A1, have the potential to cause severe hemorrhagic or ischemic complications⁵; therefore, these dissections should be treated to prevent catastrophic events. On the other hand, arterial dissections emerging in the distal part of the intracranial vessels that cause ischemic symptoms—as was observed in this case—usually take a relatively benign clinical course with or without spontaneous resolution, and can be treated conservatively.⁶ If a dissection occurs at a noneloquent area that gives rise to trivial clinical symptoms and that resolves spontaneously, there might be a possibility of overlooking the arterial dissection. However, clinicians should be aware that the existence of a dissecting lesion may imply the subsequent dissection of other arteries, because the multiplicity of this disorder based on the vulnerability of vessels is not negligible.

In conclusion, this is the first case report of the definitive multiple simultaneous arterial dissections involving the anterior circulation. This case provides a clue to explore the

nature of the onset and natural history of the disease, which has recently attracted attention in the medical community.

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