

## Vertebral Artery Dissection Leading to Fornix Infarction: A Case Report

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*Background:* The subcallosal artery is a proximal branch of the anterior communicating artery and has been recognized as the vessel responsible for fornix infarction. Fornix infarction caused by vascular damage to the posterior circulation has not been reported previously. *Results:* A 26-year-old woman suffered from fornix infarction due to artery-to-artery embolism after vertebral artery dissection. Cerebral infarctions were also found in the left thalamus, body of the left caudate nucleus, and the left occipital lobe other than the fornix. *Conclusions:* Occlusion of the subcallosal artery results in cerebral infarction of fornix, anterior cingulate cortex, and genu of the corpus callosum. However, in our case, lesions were restricted to the territory of posterior circulation. In addition to subcallosal artery, lateral posterior choroidal artery, a perforating branch of the posterior cerebral artery, has been described to send branches to the fornix, so we speculated that the left lateral posterior choroidal artery was actually responsible for fornix infarction. **Key Words:** Vertebral artery dissection—fornix infarction—lateral posterior choroidal artery—cerebral embolism—posterior circulation—amnesia.

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Fornix infarction is a very rare form of cerebral infarction<sup>1</sup> caused by occlusion of a branch of the anterior communicating artery known as the subcallosal artery.<sup>2</sup> The subcallosal artery has a single dominant main trunk<sup>3</sup> that, when occluded, results in bilateral fornix infarction. The subcallosal artery also supplies blood flow to regions including the anterior cingulate cortex and genu of the corpus callosum. Therefore, cerebral infarctions in these regions are often associated with fornix infarction.<sup>1,2,4</sup> In this report, we describe a case of bilateral fornix

infarction due to occlusion of the left lateral posterior choroidal artery, which is a previously unreported culprit vessel, after artery-to-artery embolism triggered by dissection of the vertebral artery (VA).

### Case Report

A 26-year-old healthy woman developed rotatory vertigo and nausea after performing a 40-minute video-based exercise. The next day on waking, she experienced

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The authors have nothing to disclose.

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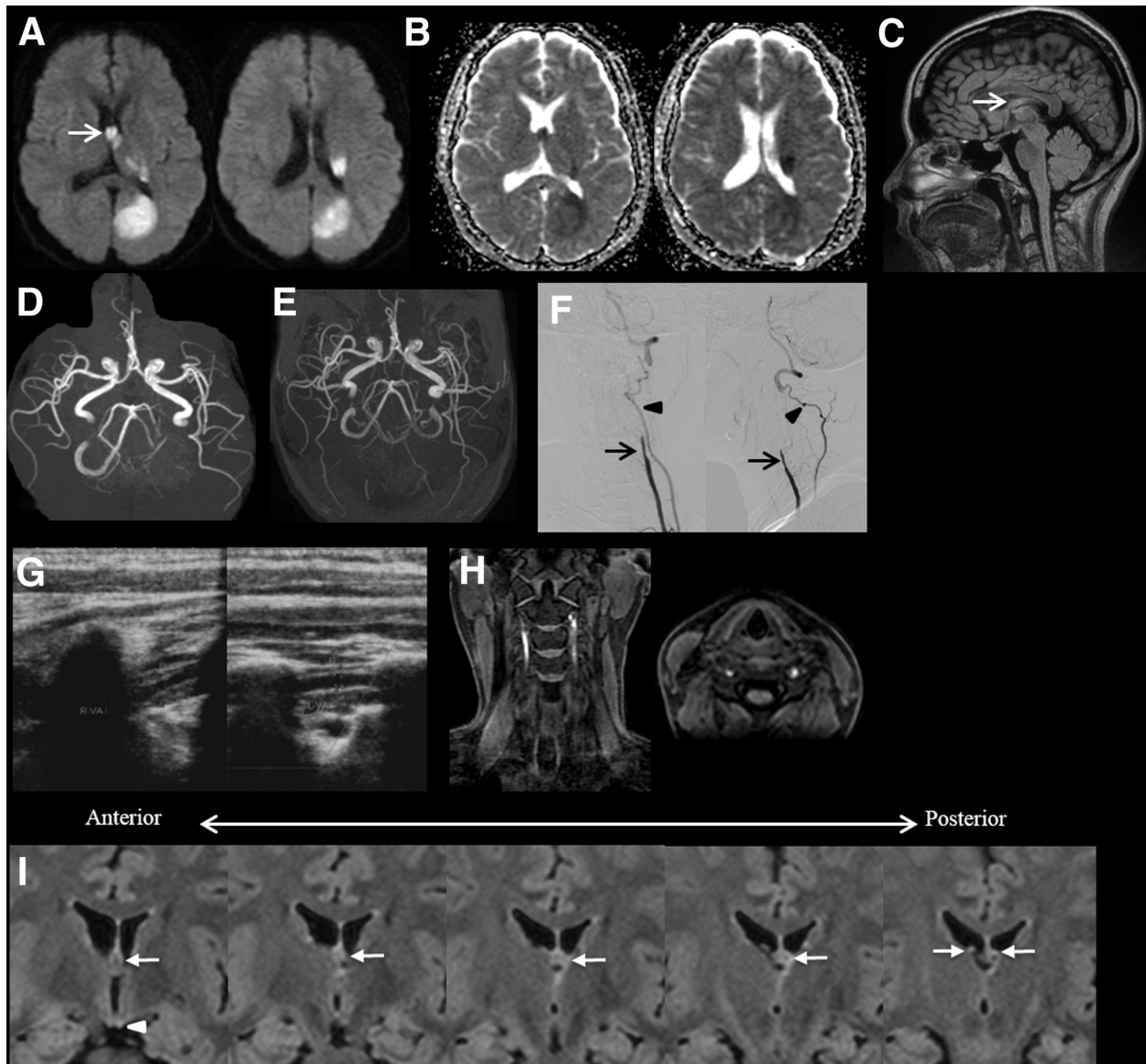
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rotatory vertigo, vomiting, and a limited field of vision. She also developed severe occipital headache, and subsequently presented to our hospital for examination. She did not smoke and only drank occasionally and was not on oral contraceptives. On arrival at the hospital, her blood pressure was 113/74 mm Hg, heart rate was 71 bpm (regular), and body temperature was 36.8°C. She did not have any physical findings suggestive of connective tissue disease, such as hyperextensibility of the skin and joints. On neurologic examination, she was lucid,

but exhibited both anterograde and retrograde amnesia, right homonymous hemianopsia, and sensory ataxia in the right upper and lower limbs.

Blood testing did not reveal any diabetes mellitus, dyslipidemia, endocrine disorders, or autoantibodies. Electrocardiography showed that she did not have arrhythmia, and findings from transthoracic cardiac ultrasonography were normal. Her brain magnetic resonance imaging (MRI) revealed acute cerebral ischemia of the bilateral fornix, left body of the caudate nucleus, of the bilateral fornix, left body of the caudate nucleus,



**Figure 1.** Axial diffusion-weighted (A) and apparent diffusion coefficient (B) magnetic resonance imaging (MRI) show acute cerebral infarction of bilateral fornix (arrow), left thalamus, left body of the caudate nucleus, and left occipital lobe. Sagittal fluid-attenuated inversion recovery MRI shows infarction of the body of the fornix (arrow) (C). Magnetic resonance angiography (MRA) shows occlusion of the second segment of the left posterior cerebral artery and poor visualization of the left vertebral artery (VA) (D). MRA at 4 weeks after the onset shows recanalization of the second segment of the left posterior cerebral artery, and fourth segment of the left VA. Bilateral posterior communicating arteries, which were not visualized at first scan, are identified (E). Anteroposterior and lateral left subclavian angiograms show complete occlusion of the second segment of the left VA (arrows), which is reconstituted by collateral flow from the left deep cervical artery (arrowheads) (F). Duplex Doppler ultrasonography reveals intramural hematoma in bilateral VA (G). Coronal and axial fat-suppression MRIs show hyperintense lesions in bilateral VA, a finding that represents dissection (H). Coronal fluid-attenuated inversion recovery MRI at 4 weeks after the onset shows infarction of the fornix with the left side and the posterior part predominance (arrows). Arrowhead indicates the mamillary body. Slice thickness is 3 mm (I).

**Table 1.** Wechsler Memory Scale Revised test index scores at 4 weeks after the onset

General memory	Verbal memory	Visual memory	Attention/concentration	Delayed recall
72	67	97	113	63

left thalamus (anterior pole and posterolateral part including pulvinar), and left occipital lobe (Fig 1, A-C). On magnetic resonance angiography, the left VA was narrowed and the left posterior cerebral artery (PCA) was occluded at the P2 segment (Fig 1, D). Cerebral angiography revealed an occlusion in the second segment (V2) of the left VA. Collateral blood circulation was seen from the left deep cervical artery to the third segment (V3) of the left VA, and the distal left VA was visible (Fig 1, F). Duplex Doppler ultrasonography of left VA indicated an occlusion pattern in the left VA and intramural hematoma in bilateral VA (Fig 1, G). In addition, fat-suppressed cervical MRI showed crescent-shaped high-intensity signals in bilateral VA, leading us to confirm the existence of intramural hematoma (Fig 1, H).

After hospitalization, artery-to-artery embolism caused by left VA dissection was suspected, and she was immediately started on a regimen of heparin, cilostazol, and rehabilitation. She demonstrated gradually improved sensory function. Two weeks after hospitalization, the retrograde and anterograde amnesia and sensory ataxia had resolved, but right homonymous hemianopsia remained. Wechsler Adult Intelligence Scale Third Edition scores were normal. Wechsler Memory Scale revised test results demonstrated that her verbal memory and delayed recall were still impaired (Table 1). Coronal fluid-attenuated inversion recovery MRI at 4 weeks after the onset showed bilateral fornix infarction with the left side and the posterior part predominance (Fig 1, I). Magnetic resonance angiography at 4 weeks after the onset showed recanalization of P2 segment of the left PCA and fourth segment of the left VA (Fig 1, E). Bilateral posterior communicating arteries, which were not visualized at first scan, were also identified. She was transferred to a hospital specializing in recovery-phase rehabilitation at 2 months after onset.

## Discussion

Our patient experienced cerebral infarction due to artery-to-artery embolism triggered by bilateral VA dissection after strenuous exercise. The cerebral infarction occurred in the fornix, left body of the caudate nucleus (lateral posterior choroidal artery territory), thalamus (thalamotuberal, thalamogeniculate, and lateral posterior choroidal artery territories<sup>5</sup>), and left occipital lobe. The thalamotuberal artery is a perforating branch of the pos-

terior communicating artery, and the thalamogeniculate and lateral posterior choroidal arteries are perforating branches of the P2 segment of the PCA. The acute ischemic lesions in the thalamus were therefore caused by embolus from the VA via the posterior communicating artery and the PCA (P2 segment). Cerebral angiography revealed that the left VA was occluded in the V2 segment due to artery dissection, and the distal artery (from the V3 segment) maintained blood flow by collateral circulation from the left deep cervical artery. VA has several anastomoses with the deep cervical artery and ascending cervical artery distal to the V2 segment. Previous literature has also reported that distal VA in VA dissection cases is supplied blood flow from collateral circulation.<sup>6,7</sup> We presume that anastomoses between the left VA and deep cervical artery in our patient had potentially functioned in a compensatory manner for the V2 segment proximal occlusion caused by the VA dissection.

To the best of our knowledge, the literature to date has not described fornix infarction caused by VA dissection. All previous reports of fornix infarction have identified occlusion of the subcallosal artery originating from the anterior communicating artery as the cause.<sup>1,2,4</sup> The subcallosal artery supplies blood flow not only to the fornix but also to regions containing the anterior cingulate cortex and genu of the corpus callosum, so occlusion of this artery often leads to the formation of cerebral infarctions in these regions. In our patient, however, embolus from the VA dissection would have had to move via the vertebral, basilar, posterior cerebral, posterior communicating, internal carotid, anterior cerebral, and anterior communicating arteries to reach the subcallosal artery. Furthermore, the patient did not have any accompanying infarctions in the anterior cingulate cortex, so fornix infarction seems unlikely to have been caused by occlusion of the subcallosal artery. The lateral posterior choroidal artery has also been described to send branches to the crus, body, and columns of the fornix.<sup>8,9</sup> In our patient, the left lateral posterior choroidal artery is likely to supply blood to the bilateral fornix owing to the unpaired variation of that artery. We concluded that the fornix infarction was caused by VA embolus that migrated to the left lateral posterior choroidal artery via the left PCA, which was extremely rare and unreported situation.

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