Case Study

Transient Global Amnesia Secondary to Atherosclerotic Stenosis of Accessory Posterior Cerebral Artery

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Key Words: Transient global amnesia—artery-artery embolism—accessory posterior cerebral artery—cerebral infarction—posterior circulation

Case Report

A 65-year-old woman was presented with a sudden onset of anterograde amnesia and iterative questioning after physical activity. Her past medical history included hypertension, diabetes, 2 symptomatic stroke events involving left temporal lobe infarction (Fig 1, A), and left basal ganglia infarction. There were no other positive neurological signs on this admission except for the previously right hemiparesis. At 3 days after onset, diffusion-weighted imaging (DWI) showed left hippocampal restricted diffusion foci (Fig 1, B). Magnetic resonance angiography indicated multiple intracranial vascular stenosis, and its novelty was a rare variation of left posterior cerebral artery (PCA), which was divided into 2 respective branches including the parietooccipital branch and temporal branch named accessory PCA.[1] Two branches originated directly from left internal carotid artery, and a severe focal stenosis was found in the accessory PCA (Fig 1, C). At the fourth day, we conducted transcranial Doppler (TCD) emboli monitoring, we firstly located the PCA monitoring area by repeatedly adjusting the monitoring depth and sampling volume combined with compression test of carotid artery. Fortunately, we detected small amount of abnormal signals in left accessory PCA after 1 hour of monitoring, which were further confirmed to be microemboli signals rather than signal artifacts by the spindle wave moving with depth of detection (Fig 1, D). Using the same procedure, any microemboli signals were not be detected in bilateral middle cerebral artery and contralateral PCA.

In order to clarify the source of the embolus, cardiac embolism and embolization from the ipsilateral posterior circulation were excluded by some relevant tests including 24-hour Holter, echocardiography, TCD foaming test, and transesophageal ultrasound with head and neck Computed Tomography Angiography (CTA). To rule out other causes of transient amnesia, the patient completed other relevant auxiliary examinations including long-term electroencephalogram monitoring, single-photon emission computed tomography cerebral blood flow perfusion imaging, Cerebrospinal Fluid (CSF) tests including autoimmune encephalitis antibodies. The above examinations had no any valuable positive findings. The patient received antiplatelet, lipid-lowering, antihypertensive, and hypoglycemic treatments, and her symptoms did not recur after half a year of follow-up.

Discussion

Acute transient amnesia may be the sole or main manifestation of some diseases, mainly including transient global amnesia (TGA), epileptic seizures (transient epileptic amnesia), migraine with aura (migrainous amnesia), limbic encephalitis, dissociative amnesia, and stroke in special site.[2] TGA refers to a neurological emergency for a sudden onset of antegrade amnesia with accompanying repetitive questioning that generally resolves within 24 hours.[3] Patients may have mild disorientations in time and space, accompanied by symptoms of autonomic nervous system such as headache, nausea, and dizziness, and no positive signs of nervous system in the examination. Punctate hippocampal DWI hyperintensities are described in up to 80% of patients, which

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Received November 1, 2018; revision received December 26, 2018; accepted December 30, 2018.

Source of supports/grants: Nil.

Conflict of interest: None.

Patient consent: Obtained.

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1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2018.12.046

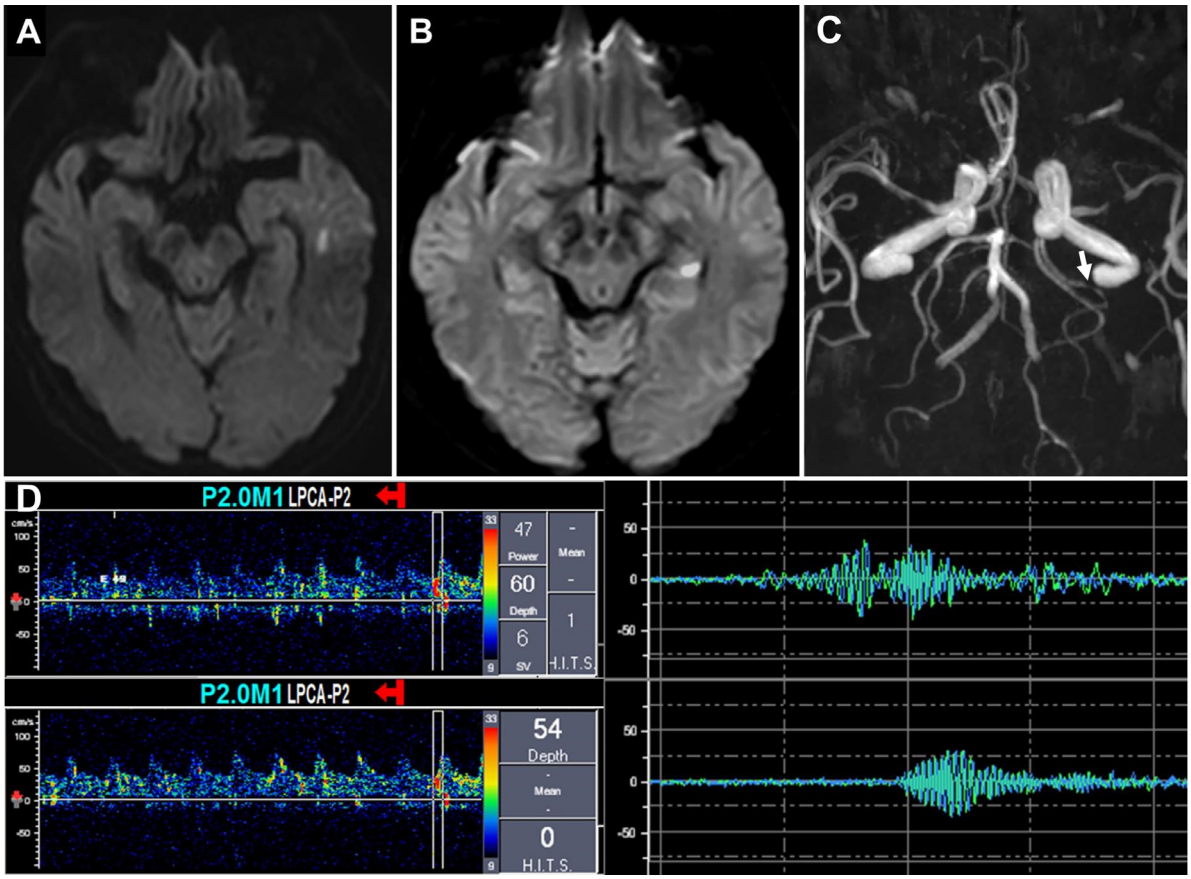

Figure 1. (A) *Diffusion-weighted imaging (DWI) before 1 year showed left temporal lobe infarction.* (B) *DWI after this admission showed left hippocampal punctate hyperintensity.* (C) *Magnetic resonance angiography revealed the severe stenosis of left accessory PCA (white arrow).* (D) *Transcranial Doppler emboli monitoring detected the microemboli signals on left accessory PCA.*

usually appear 24-72 hours after TGA onset.[4] The clinical manifestations and imaging characteristics of this patient were consistent with TGA, and extensive ancillary investigations excluded other possible causes.

Although the etiology of TGA is currently unclear, including vascular factors (venous congestion or arterial ischemia), epileptic seizure, and migraine,[5-8] the hippocampal punctate lesion was usually detected in DWI. The mechanisms of the hippocampal lesion were controversial, but focal arterial ischemia may be still one important mechanism.[8,9] Anatomically, accessory PCA is extremely rare, and there are only a few case reports in the literatures. In 2016, Uchino et al pointed out that the accessory PCA may be the hypoplastic anterior choroidal artery, mainly supplying the temporal branch of the PCA.[1] Interestingly, 2 ischemic lesions in left temporal lobe of the patient were located in the blood supply regions from the accessory PCA. In our case, there was severe arteriosclerosis stenosis with evidence of falling off embolus on left accessory PCA. Therefore, after excluding other causes, the cause of this TGA was most likely due to hippocampal artery-artery embolism secondary to atherosclerotic stenosis of accessory PCA.

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