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# Recurrent attacks of transient global amnesia with intracranial vertebral artery dissection: Case report

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***Abstract***

Whether Transient global amnesia (TGA) is related to ischemic pathogenesis remains unclear. The recurrence of TGA is rare. We report a rare case of recurrent attacks of TGA with intracranial vertebral artery dissection in young men without other triggers and any associated factor. A 39-year-old man was hospitalized for amnesia. Diffusion-weighted imaging (DWI) revealed a tiny focus of high-intensity signals in the left. High-resolution MRI showed the dissecting aneurysm of the V4 portion of the right vertebral artery. Two years later, the patient experienced sudden memory loss again. On DWI, a new punctate restricted diffusion lesion was observed in the left hippocampus. Considering the recurrence in young men with no other triggers, this case raises the hypothesis that a pure TGA in a young patient could potentially be a vertebrobasilar TIA. Therefore, when a patient presents with TGA, without any associated factors, it is important to investigate and exclude sinister causes of global amnesia and we recommend that a vascular examination be conducted.

*Keywords:* Transient global amnesia, intracranial vertebral artery dissection, stroke

## INTRODUCTION

Transient global amnesia (TGA) is characterized by sudden onset of altered behavior with temporary dysfunction of anterograde memory for a brief duration with gradual resolution.[1] However, there is no consensus regarding the cause of this phenomenon.[2] Whether TGA is related to ischemic pathogenesis caused by an embolic or hemodynamic arterial mechanism or a consequence of venous ischemia remains unclear.[3] The recurrence of TGA is rare and most patients with TGA experience no recurrence and show a benign natural history. Here, we report a rare case of recurrent attacks of TGA with intracranial vertebral artery dissection in young men without other triggers and any associated factor.

## CASE REPORT

A 39-year-old man was hospitalized for repeatedly asking the same questions and forgetfulness. The patient was mildly confused and disoriented in terms of time and place. The symptoms resolved within 4 hours. Immediately after the emergency room visit, the patient's symptoms had resolved. There were no lapses of consciousness, weakness, tingling, dysarthria, and vertigo. There was no history of similar episodes, and the patient had no underlying diseases. On physical examination, his blood pressure was 158/113 mmHg, pulse was 72 beats per minute, body temperature was 36.0 °C, and respiratory rate was 18 breaths per minute. Cardiovascular examination revealed a regular heart rate and rhythm without murmurs. Neurological examinations revealed no specific findings. Laboratory analyses and electrocardiography findings were within the normal range. MRI of the brain was performed at 4 h (Figure 1 A) and 57 h after the onset of symptoms revealing a tiny focus of high-intensity signals in the left hippocampus on diffusion-weighted imaging (DWI) (4 mm thickness, b-value = 1000), with a corresponding apparent diffusion coefficient (ADC). T2-FLAIR images at the level of the DWI lesions were unremarkable. Time-of-flight magnetic resonance angiography (MRA) and contrast-enhanced MRA showed localized irregular stenosis and enlargement of the V4 portion of the right vertebral artery (VA) (Figure 2 A).

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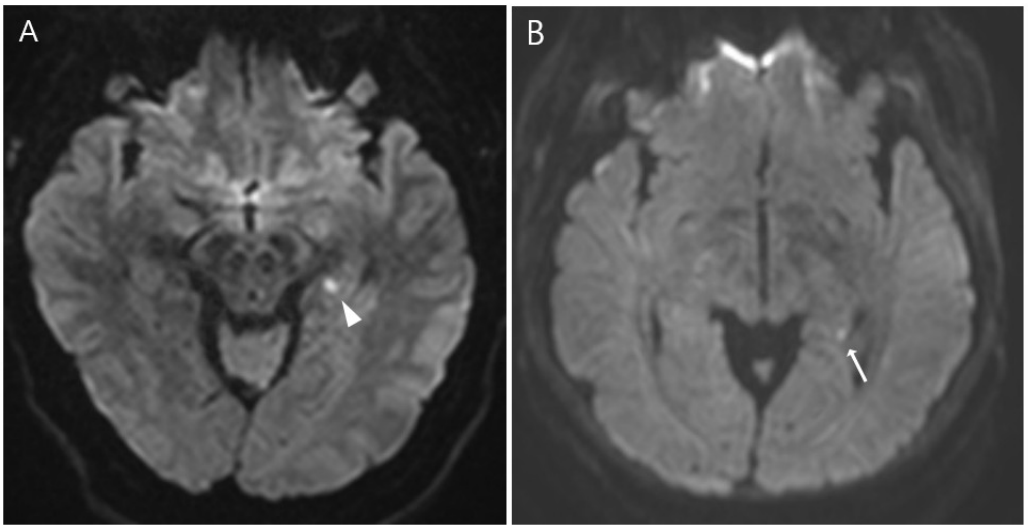


Figure 1: Magnetic resonance imaging findings. (A) Diffusion-weighted imaging shows punctate hyperintense signals in the left hippocampus, 4 hours after onset of symptoms (white arrowhead); (B) 2-years later, a new punctate restricted-diffusion lesion was revealed in the left posterior hippocampus

High-resolution MRI (HRMRI) was performed to accurately assess the inner wall of the cerebral artery and evaluate the possibility of a VA dissecting aneurysm. HRMRI was performed using a 3-Tesla (3T) scanner with a 20-channel head coil (MAGNETOM Skyra, Siemens, Germany). Two-dimensional sequences and blood-flow suppression images were acquired using turbo spin echo (TSE) with variable-flip-angle refocusing radio frequency pulses (sampling perfection with application-optimized contrast using different angle evolutions (SPACE). The number of slices was 15 and the acquisition time was 202s. The scanning orientation was transversal, and the 3D images were reconstructed into coronal, axial, and sagittal images. The HRMRI images were independently assessed by two neurologists and one radiologist. The HRMRI showed intimal flap and double-lumen at the location of local dilatation in the proton-density imaging (Figure 2 B). Crescentic intramural hematoma that displayed an eccentric signal void surrounded by a semilunar hyperintensity was observed on T1-weighted imaging (Figure 2 C). Contrast-enhanced T1-weighted imaging of HRMRI showed no significant enhancement of the intramural hematoma (Figure 2 D).

The patient's Korean version of the Mini-Mental Status Examination (K-MMSE) score was 30/30. He underwent transthoracic echocardiography, 24-hour Holter monitoring, and transcranial Doppler monitoring with the saline agitation test. All results were normal. We decided on conservative management using antiplatelet therapy for a year.

Two years later, the patient experienced sudden memory loss lasting an hour which had resolved at the time of hospital visit. Physical and neurological examinations revealed no specific findings. MRI was performed again. On DWI and ADC maps, a new punctate restricted diffusion lesion was observed in the left hippocampus (Figure 1 B). MRA of the extracranial and intracranial vessels showed that the right vertebral artery dissection seen earlier was unchanged after 2 years. Additional transesophageal echocardiography (TEE) showed no abnormal findings, such as PFO. The patient continued the antiplatelet therapy.

## DISCUSSION

Although many factors including venous congestion, migraine, hypoxic-ischemia, and epilepsy contribute to the dysfunction of the CA 1 region, whether these are of major significance have yet to be determined.[4] In TGA patients, brain MRI may reveal a punctate focal diffusion restriction lesion of 2–3 mm in the mesiotemporal area. However, vigorous evidence supporting an ischemic etiology remains lacking.

In this case, it can be estimated that the vertebral artery dissection is in the acute stage as an intramural hematoma was distinctly observed on HRMRI, suggesting that TGA symptoms were highly likely at the time of the VA dissection. There were no other triggers; only the VA dissection was observed. This further supports its association with TGA. In addition, most patients with TGA do not

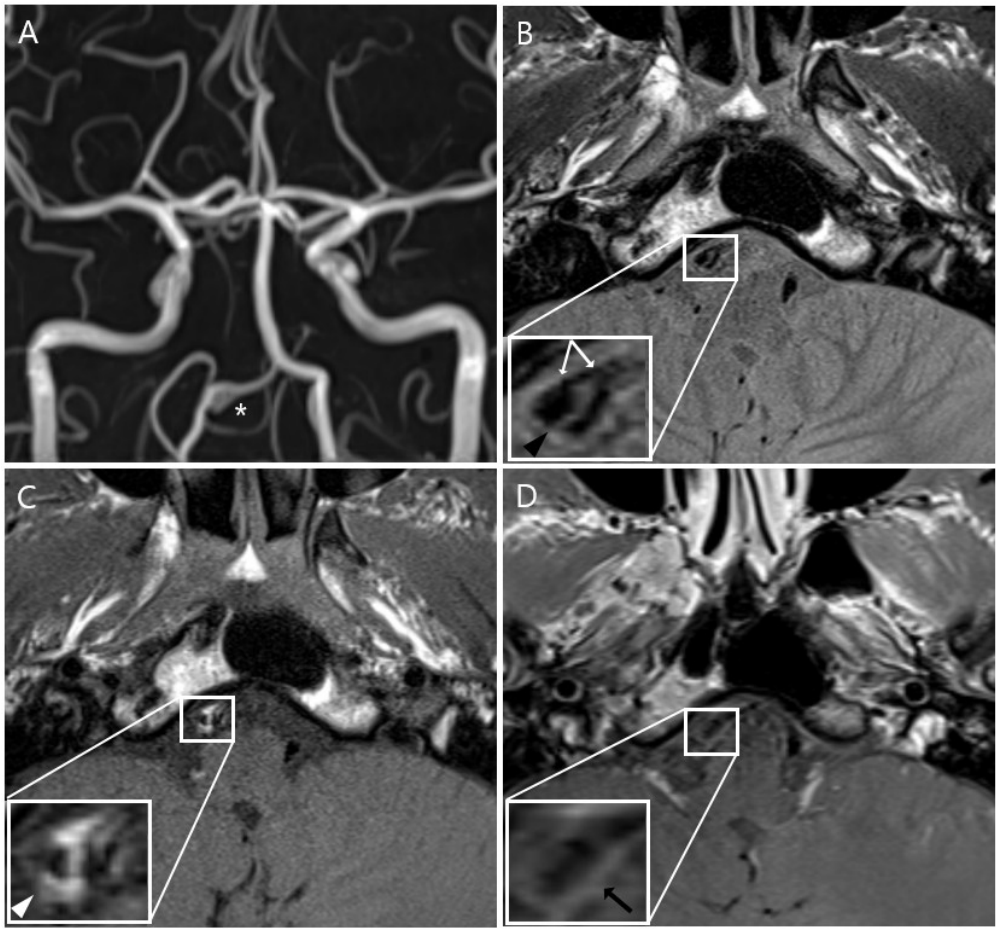


Figure 2: HRMRI images of the patient (A) Time-of-flight magnetic resonance angiograph showing dissecting aneurysm in the right vertebral artery (white star); (B) Proton density sequence showing the dissecting flap (black arrowhead) and the double lumen, (white arrow); (C) T1-weighted image of HRMRI showing eccentric intramural hematoma (white arrowhead); (D) Contrast-enhanced T1-weighted image of HRMRI showing no significant enhancement of intramural hematoma.

experience recurrence and have a benign natural history.[3] Some reports suggest that familial cases of TGA, depression, previous head injury and family history of dementia may be associated with higher recurrences of TGA.[5] However, no known recurrence characteristics were observed in this patient. The patient experienced recurrent TGA symptoms after 2 years, and the recurrent lesions were observed at similar locations as before.

Some previous studies reported that TGA is less to be a factor associated with stroke or transient ischaemic attack (TIA).[1,2] However, VA dissection is a common cause of ischemia in young adults. The hippocampus is mainly supplied by the longitudinal terminal segments of the hippocampal arteries from the posterior cerebral artery.[4] In this case, it is possible that an embolus originating from the dissecting aneurysm of the right vertebral artery caused the initial ischemia, and later, an embolus from the previously partially occluded hippocampal artery induced the recurrent ischemic change. Considering the recurrence in young men with no other triggers, this case raises the hypothesis that a pure TGA in a young patient could potentially be a TIA. Therefore, when a patient presents with TGA, without any associated factors, it is important to investigate and exclude sinister causes of global amnesia and we recommend that a vascular examination be conducted.

## DISCLOSURE

Ethics approval: This study was approved by our institutional ethical review board and was conducted in accordance with the ethical standards and with the 1964 Declaration of Helsinki and its later amendments. Written informed patient consent was obtained.

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Conflict of interests: None

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