

Bilateral Hippocampal Abnormalities on Diffusion-weighted MRI in Transient Global Amnesia: Report of a Case

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Abstract- The pathophysiology of transient global amnesia (TGA) is still speculative. Recently, diffusion-weighted image (DWI) of magnetic resonance imaging (MRI) documented tiny lesions in the hippocampus of patients with TGA in the acute stage. Most studies reported unilateral lesions on MRI. We present one patient of TGA with high signal-intensity lesions in bilateral hippocampus on DWI at the acute stage. The serial findings of brain MRI support the ischemic nature of TGA. Related mechanism about TGA is discussed.

Key Words: Transient global amnesia, Hippocampus, DWI, Cerebral ischemia

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INTRODUCTION

Transient global amnesia (TGA) is a benign clinical entity that is characterized by a sudden-onset transient memory disturbances with preserved alertness, attention, and personal identity⁽¹⁾. It is a topic of interest considering its recent discovery in imaging findings and pathophysiology. Cases of focal magnetic resonance (MR)-signal abnormalities on diffusion-weighted imaging (DWI) at the lateral aspect of the hippocampus have been reported since 1998⁽²⁾. Of these reports, most patients showed unilateral signal changes. We document a TGA patient with hyperintensity lesions on DWI and T2 weighted image (T2WI) in bilateral hippocampus during the acute stage. The lesions on DWI disappeared 6 weeks later but the high signal-intensity lesions on T2WI persisted at this time-point.

CASE REPORT

A 59-years-old male, without underlying diseases other than hypertension under regular medication control, presented to the emergency room owing to repeating same questions after defecation. The neurological examinations on arrival revealed marked memory impairment. He had retrograde amnesia up to 3 months ago, anterograde amnesia and disorientation to time but normal immediate-recall. Other cognitive functions, including language, calculation and abstract thinking remained intact. There was no limb weakness or aphasia. The blood pressure was 150/88 mmHg. An immediate non-contrast brain computed tomography did not show any abnormality. Laboratory data including biochemistry, hemogram, and electrocardiogram were normal. A brain MR study during hospitalization (approx-

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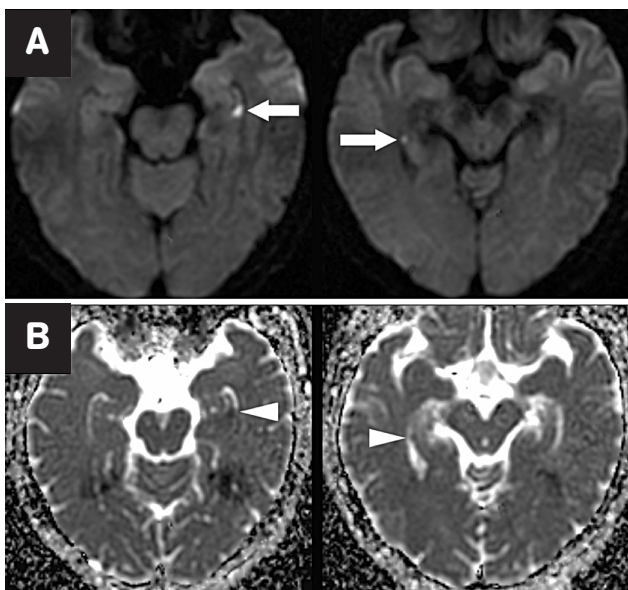


Figure 1. (A) The initial axial MRI diffusion weighted imaging shows lesions of high signal-intensity in bilateral hippocampus (arrows). (B) ADC imagings show hypointensity lesions in the corresponding areas of bilateral hippocampus (arrowheads).

mately 45 hours after the onset of symptoms) disclosed two restricted diffusion lesions (increased signal) in the hippocampus on DWI and T2WI: one at the posteriolateral aspect of the right hippocampus with a size of 3.5×3 mm, and the other at the anterior aspect of the left hippocampus. The apparent diffusion coefficient (ADC) mapping of the corresponding areas showed lesions of low signal-intensity (Fig. 1). MR angiography did not show stenosis of major intracranial arteries. Electroencephalogram and color-coded carotid and transcranial duplex sonographies revealed normal results. There was no abnormality of bilateral internal jugular venous flow. He regained most of his memory and could memorize new events about 24 hours after symptom onset. However, he was still unable to recall what had happened on the day of symptom onset. He received anti-platelet treatment after admission. He had normal appearance and memory functions at the follow-up examinations 6 weeks later. Eventually, he still had no memory about the day of symptom onset. The follow-up brain MR taken 5 weeks after the symptom-onset showed persistent hyperintensity of the previous left hip-

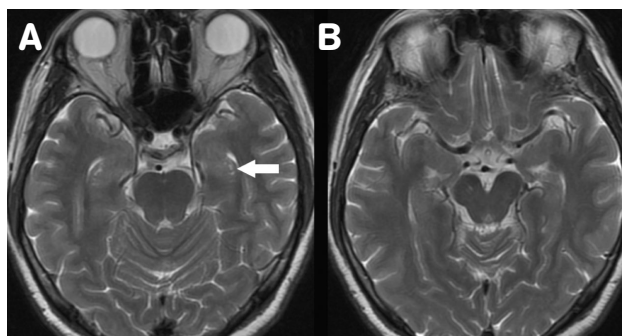


Figure 2. (A) A follow-up brain MR 5 weeks later demonstrates the persistence of high intensity lesion in the left hippocampal area (arrow). (B) The right hippocampal lesion disappeared on T2WI.

pocampal lesion with the disappearance of the right lesion on T2WI. The previous hyperintensity lesions on DWI also disappeared (Fig. 2).

DISCUSSION

The patient reported here presented as a clinical feature of TGA. Diagnosis of TGA is further supported by bilateral hippocampal hyperintense lesions on DWI of the brain MR. No epileptic event could be traced from the history and electroencephalogram. The impaired memory improved gradually without any anticonvulsant treatment. No abnormal internal jugular venous flow was detected by carotid duplex sonography. We categorize it into a small vessel ischemic stroke with a risk factor of chronic hypertension. Long-term antiplatelet agent was given for stroke prevention.

Transient ischemic changes and epileptic events are two major mechanisms proposed to be the etiologies of TGA⁽²⁻⁷⁾. The hypothesis of transient ischemia of brain tissues, especially in the hippocampus, has been supported through modern imaging studies^(2,4-7). Jia et al using PET to display low metabolism in the hippocampus related to memory although no abnormality was found on the MR study⁽⁸⁾. Recent studies reported that DWI is helpful to demonstrate tiny lesions at the early stage (24 to 72 hours after time of onset) of patients with TGA^(6,7). The typical findings of the lesions by MR studies are hyperintensity at the hippocampal area on DWI and T2WI with corresponding hypointensity on ADC. These

MR findings support the ischemic etiology of TGA.

The etiology of ischemia selectively over hippocampal area is speculative. Hypothesis has postulated that CA-1 of the hippocampus (Sommer sector) may be more vulnerable to ischemic insult due to its low collateral circulation as a watershed area and unique membrane receptors^(5,6). Noting a prevalence of trigger events such as exertion, emotional stress, or sexual intercourse, another hypothesis of venous congestion, mediated by intracranial venous reflux, has recently developed. The intracranial venous reflux may be caused by the valve incompetence of the internal jugular vein or the left brachiocephalic vein occlusion after a precipitating factor such as Valsalva maneuver^(9,10). Chung et al. detected intracranial venous reflux in patients with TGA⁽¹⁰⁾. Currently, there is no publication of correlation between DWI abnormality and intracranial venous reflux.

In three large series of studies on TGA, the DWI abnormalities were detected in 55 of 93 (54.5%) patients^(5,7,11). Besides, only 13 patients (16.3%) in 80 reported TGA patients from four case series are bilateral hippocampal involvement^(5-7,11). No analysis was made between patients with unilateral and bilateral hippocampal involvement. There is sparse evidence to explain the phenomenon of bilateral hippocampal involvement. A potential hypothesis is that lesions caused by hemodynamic factor rather than certain arterial ischemia. In addition, some bilateral cases may be attributed to small vessels disease secondary to chronic hypertension or diabetes mellitus. However, we propose that there might be more bilateral hippocampal involvement than the current report for following reasons. Firstly, one side of the lesions may be too tiny to be demonstrated by brain MRI or be under-recognized by the radiologist. Some tiny high lesions on DWI should be reviewed carefully with the ADC mapping to determine their nature. Secondly, there are differences in the timing of imaging. Since not every serial MR study was performed day-by-day, one side lesion may be disclosed before the appearance of another side^(5,6). The patient reported here had bilateral hippocampal lesions on DWI of MR. However, the right hippocampal lesion is extreme tiny and is easily to be overlooked.

In conclusion, current results of MR imaging suggest a considerable proportion of TGA to be ischemic in

nature. Unilateral involvement of hippocampus is still the majority of the abnormal finding. Detail MR studies probably help to find bilateral hippocampal lesions. The precise mechanism of the hippocampal ischemia deserves further evaluation, such as the correlation between DWI abnormality and venous congestion, and the difference between unilateral and bilateral hippocampal involvement.

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