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# A Tiny Hippocampal Ischemic Lesion Associated with Transient Global Amnesia

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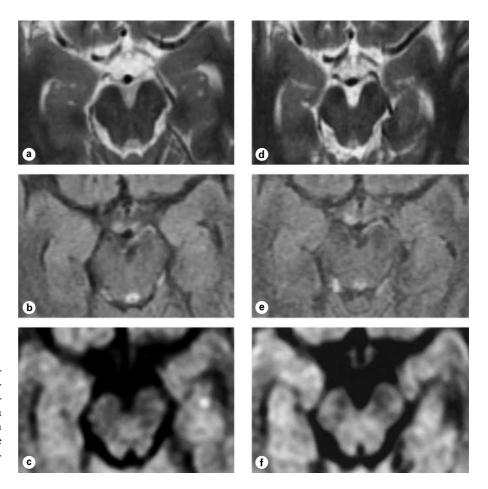
Transient global amnesia (TGA) is a transient, benign neurological syndrome, characterized by temporary loss of anterograde and recent retrograde memory with preserved consciousness and selfawareness, usually accompanied by repetitive questioning and temporal disorientation [1, 2]. Proposed etiologies include seizure, migraine, focal vasospasm, increased cerebral venous pressure and cerebral infarction. Recently, it has been reported that patients with TGA can show abnormalities on diffusion-weighted magnetic resonance imaging (DWI) [3–5], and these findings may help understand the pathophysiology of TGA. We report a case of TGA associated with a tiny left hippocampal ischemic lesion on DWI which was not accompanied by abnormalities on the T<sub>2</sub>-weighted image and resolved on follow-up images.

# Case Report

A 50-year-old right-handed man was admitted because of a transient memory disturbance that had developed 3 days previously during a trip with his family. After dinner on that day, he suddenly complained of dizziness and then asked his wife the same questions over and over such as 'Why am I here?' 'How did I get here?' 'Where have I been this morning?' About 15 min later, these symptoms disappeared, and the patient's condition appeared to be back to normal except that he vomited once on the way to a near hospital. There, brain CT showed no specific lesion. The patient visited our clinic for further evaluation. He reported that he could faintly remember what had happened during the attack.

His past medical history was only significant for gout and smoking (2 packs/day for about 30 years). Physical and neurological examinations were all unremarkable. Laboratory tests including coagulation profiles were all within normal limits except for an increased uric acid level. ECG, echocardiography and surface EEG were all normal. On the third day after the attack, brain MRI was carried out, which revealed a tiny high-signal lesion in the left hippocampus only on DWI with no changes on T<sub>2</sub>-weighted, fluid-attenuated inversion recovery, T<sub>1</sub>-weighted and T<sub>1</sub>-weighted enhanced images (fig. 1). The apparent diffusion coefficient value was decreased, being 83% of the value of the corresponding area of the contralateral side. Brain magnetic resonance angiography showed no diagnostic abnormalities. The scores of a neuropsychological battery, performed on the next day, which included attention, language, visuospatial, memory and frontal lobe functions were within normal limits. Tc-99mHMPAO SPECT carried out 7 days after the attack showed mild hypoperfusion in the left temporal area (fig. 2). Repeat MRI with the thin-slice technique around the temporal lobe performed 56 days after the onset showed no signal abnormalities on conventional magnetic resonance sequences and DWI.

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**Fig. 1.** Axial  $T_2$ -weighted (**a**), fluid-attenuated inversion recovery (**b**) and diffusion-weighted (**c**) magnetic resonance images taken 3 days after the TGA attack showed a high-signal lesion only on DWI. The lesion resolved on follow-up images (**d**-**f**) with the same magnetic resonance sequences performed 56 days after onset.

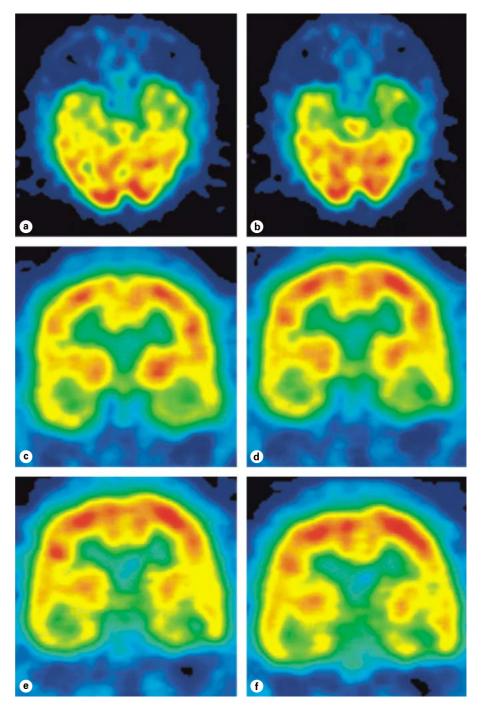
# Discussion

Our patient had experienced transient amnesia with no evidence of seizure or head trauma, which resolved within 24 h. Thus, these findings fulfilled the criteria of TGA [1, 2], although, because of the short duration (minutes rather than hours) of the amnesia, we cannot completely exclude the possibility of an amnestic seizure. Unexpectedly, DWI showed a tiny high-signal intensity in the left hippocampus. This lesion represents an acute ischemic lesion rather than an artifact or a  $T_2$ -weighted shine-through effect because the apparent diffusion coefficient value was decreased. Thus, among the proposed etiologies of TGA including focal vasospasm, seizure, migraine, increased cerebral venous pressure and cerebral infarction, our case supports the ischemic cause.

A study in which a group of TGA patients were examined with DWI demonstrated a diffuse high signal in the hippocampus in 7 out of 10 patients. This abnormality, however, has been interpreted as cellular edema rather than ischemic lesions [6]. Only 3 TGA cases, so far, have been reported, which had an acute infarction demonstrated by DWI. A patient developed TGA during posterior cerebral artery angiography. DWI showed acute infarction in the right and left hippocampus, and the right thalamus. The abnormalities, although they were subtle, were also seen on T<sub>2</sub>-weighted imaging [3]. Another patient had an infarction in the left parahippocampal gyrus on DWI

and an additional lesion in the splenium of the corpus callosum seen on DWI as well as T<sub>2</sub>-weighted imaging [4]. The third case showed a left hippocampal infarction similar to ours, which became a permanent lesion on follow-up T2-weighted MRI [5]. Our case may differ from these cases in that the lesion was restricted to the hippocampus and in that the diffusion abnormality was not accompanied by T<sub>2</sub>weighted abnormalities nor did it leave any permanent damage as seen by follow-up MRI. Our case may have an important clinical implication as follows. It has been widely known that classic TGA patients do not show abnormal findings in CT or MRI. We cannot exclude the possibility, however, that some of these patients might have had small infarctions in memory-related structures such as the hippocampus. This may be especially true when MRI was performed after the acute phase. In such situations, the patient, as having a benign condition, is considered not to need any stroke prevention. Therefore, DWI in the acute phase of TGA may play an important role in the clinical management of some TGA patients.

We are uncertain whether such a small lesion, even if it is located in the hippocampus, can cause amnesia even for a transient period. However, hypoperfusion in the left hippocampal area detected by SPECT 1 week later may indicate that there might have been a widespread dysfunction during the attack period, leaving only a small ischemic lesion. Previous TGA cases studied with SPECT during the



**Fig. 2.** Axial (**a**, **b**) and coronal (**c**-**f**) images of Tc-99mHMPAO SPECT taken 7 days after the TGA attack, demonstrating mild hypoperfusion in the left temporal area.

acute phase also demonstrated hypoperfusion or rarely hyperperfusion in the unilateral or bilateral temporal lobe [7–11]. Among the reported TGA patients with apparent ischemic changes found by MRI, our patient had the smallest lesion. This may explain the short duration of TGA in our patient, although this must be confirmed with more patients in the future. References

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# PET Study in Bilateral Internal Carotid Artery Occlusion

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#### Introduction

As opposed to ipsilateral internal carotid artery (ICA) occlusion, little information is available on the bilateral ICA occlusion [1–5]. We experienced a patient with bilateral ICA occlusion, and describe the positron emission tomography (PET) findings and discuss the prognosis of this patient.

### Case Presentation

A previously healthy 52-year-old Japanese businessman without histories of hypertension and diabetes mellitus was admitted to our hospital complaining of recurrent transient ischemic attacks (TIAs) on February 14, 2001. The initial attack was a transient numbness of his left third finger on December 8, 1999. The second attack was a transient paresthesia of his left upper and lower extremities with no abnormalities on computed tomography (CT) on July 14, 2000. Five days later, he suffered a third attack of transient weakness of his left hand. The last attack of transient left hemiplegia of 5 min duration occurred on January 3, 2001.

The past medical history and family history were unremarkable. On admission, blood pressure was 128/80 mm Hg, and heart rate was 85/min with a regular rhythm. The patient was alert and cranial nerves were intact. He exhibited no motor or sensory deficits in any extremity. Cerebellar function and bilateral deep tendon reflexes were normal, and Babinski signs were negative. He was completely independent in his activities of daily living. CT and magnetic resonance imaging on admission showed no abnormalities.

Laboratory examinations on admission yielded the following results: erythrocyte sedimentation rate, 11 mm/h; C-reactive protein, <0.1 mg/dl; red blood cell count,  $481 \times 10^4$ /mm<sup>3</sup>; white blood cell count, 7,900/mm<sup>3</sup>, and platelet count, 29.5 × 10<sup>4</sup>/mm<sup>3</sup>. Urinalysis was normal. Blood chemistry values relating to hepatic and renal function were normal, as were serum electrolyte concentrations. Serum concentrations of total cholesterol, high-density lipoprotein cholesterol, lipoprotein(a), protein S, and protein C were normal, except for triglyceride (251 mg/dl). The blood sugar concentration was 161 mg/dl on admission, and the serum HbA1c level was 5.6% (normal <5.8%). Coagulation tests showed normal bleeding and coagulation times. Electrocardiography, echocardiography, and chest roentgenogram showed no abnormalities.

Immunologic studies showed no abnormalities involving antinuclear factor, lupus anticoagulant, IgG or IgM cardiolipin antibody, antibody to native DNA, antibody to ribonucleoprotein, antibody to smooth muscle, antibody to SSA, antibody to SSB, serum IgG, IgA, IgM, C3 and C4 concentration, serum immunocomplex, a latex agglutination test for rheumatoid arthritis, anti-myeloperoxidase antibody, or anti-neutrophil cytoplasmic antibody. Neither anti-human T-cell lymphotropic virus type 1 nor anti-human immunodeficiency virus antibodies were detected.

Cerebral angiography showed that the ICA were occluded bilaterally at their origin (fig. 1a), and most of the blood flow to the whole brain was supplied through the vertebrobasilar system. Both vertebral angiograms (Towne view) showed that the circulation of the right middle cerebral artery (MCA) territory was weaker than that of the left MCA region (fig. 1b), but the lateral views of both vertebral angiograms seemed to reveal normal circulation in the supra- and infra-tentorial regions (fig. 1c, d).

The right carotid angiogram (lateral view) showed that the leptomeningeal collateral supply via external carotid system to the right hemisphere was weak, and retrograde flow through the ophthalmic artery was also faint (fig. 1e). The left carotid angiogram (lateral view) showed that these collateral circulations were extremely poor (fig. 1f).

Magnetic resonance angiography showed that both the posterior communicating (post.com.) arteries were thick enough, but the A1 segment of right anterior cerebral artery was faint (fig. 1g).

PET revealed that cerebral blood flow decreased and oxygen extraction fraction (OEF) increased in the whole brain except for the cerebellum (table 1). These findings were remarkable in the right frontal and parietal lobes (fig. 2). After the administration of ticlopidine 200 mg and warfarin 1 mg daily, the patient has not developed TIAs for 23 months.

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