

An Acute Tiny Left Putamen Lesion Presenting With Transient Global Amnesia

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Introduction: Transient global amnesia (TGA) is one of the most striking syndromes in clinical neurology. Brain lesions producing TGA have been reported in the hippocampus, amygdala, thalamus, caudate nucleus, and neocortex.

Case Report: We report a 63-year-old woman presenting with TGA associated with a localized lesion in the left putamen.

Conclusions: This case suggests a potential role of the putamen in human memory processing.

Key Words: transient global amnesia, diffusion-weighted image, putamen

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Transient global amnesia (TGA), a sudden onset of anterograde and retrograde amnesia, has been associated with ischemia, migraine, epileptic seizure, venous congestion, and psychological disturbances.¹ The underlying mechanisms and localization of lesions causing such memory loss have been subject of debate. Magnetic resonance imaging (MRI) has been used to study brain abnormalities in TGA. In the acute and postacute stages (within 24 h) of TGA onset, diffusion-weighted imaging (DWI), when positive, has generally revealed restricted diffusion in the temporal lobes, the hippocampal region, and in thalamic, striatal structure, and neocortex.² We report a case of TGA with a highly restricted lesion in the left putamen revealed by DWI; the lesion was associated with a reduced apparent diffusion coefficient (ADC) in the same area and an increased blood flow in the left temporal lobe.

CASE REPORT

Clinical History

A 63-year-old right-handed housewife presented to the emergency department complaining of memory lapse after severe emotional trauma. Her previously healthy husband had been admitted to hospital with an acute stroke 3 days before her visit to the emergency depart-

ment. The evening before she suddenly appeared perplexed and confused and repeatedly asked her sister where she was and why she was there. In addition to agitation she was unable to remember what she had been doing since the previous evening and could not remember her husband's admission to hospital. She was also unable to keep track of time. There was no alteration in her level of consciousness reported at any time during the episode. There was no history of any previous episode. She did not recall any form of recent head trauma, and there was no history of hypertension, diabetes, transient ischemic attacks, stroke, epilepsy, migraine, psychiatric illness, or alcohol abuse. She was not receiving any medication and did not smoke. The patient gradually recovered from the period of amnesia after 7 hours, but this time window and the events therein remained obscure for her. The memory deficit completely recovered over the next 10 hours. However, she could not recover her memory lapse during the first 7 hours of the amnesic attack.

Assessments

The patient was alert, oriented, and conversant. Her blood pressure was 111/80 mm Hg, respiratory rate 80/min, and body temperature 36.7°C. Physical examinations revealed no abnormalities in the chest, abdomen, and extremities. Neurological examination was normal except for retrograde and anterograde amnesia. She did not exhibit deficits in long-term memory, judgment, attention, language, or emotional lability on repeated neuropsychological assessments 48 hours after her initial symptoms. Laboratory studies including blood cell counts, hepatic and renal function, serum cholesterol, blood glucose, arterial blood gases, and blood coagulation were all normal. There were no abnormal findings on electrocardiography, chest x-ray, transthoracic echocardiography, carotid ultrasonography, or transcranial color sonography.

Initial and follow-up MRI investigations were performed using a GE Signa EXITE HD 3.0T (General Electronics) with echoplanar (EP) capability. Imaging was performed both immediately after the amnesic attack and approximately 48 hours after the onset of symptoms. A single-shot EP diffusion-weighted sequence (EP spin echo repetition time 1200 ms/ echo time 77.8 ms; a 192 by 192 matrix with number of signal averages of 1; slice thickness 3.5 mm with no gap; b value 0 to 2000 s/mm², and scan time 144 s) was used. Initial MRI revealed no abnormalities, but subsequent DWI demonstrated a focus of markedly elevated signal intensity in the left putamen, with decreased ADC in the corresponding area, suggestive of acute infarction (Figs. 1A, B). FLAIR sequence showed a slightly high signal intensity, whereas T1-weighted and T2-weighted images demonstrated no abnormal findings in the same region, which suggested that the responsible lesion was most likely due to acute ischemic insult. Brain single-photon emission computed tomography analysis with ^{99m}Tc-hexamethyl propyleneamine oxime disclosed mildly increased perfusion in the left temporal cortex and basal ganglia (Fig. 2).

DISCUSSION

The present case meets the clinical criteria for TGA proposed by Hodges and Warlow.³ Although the initial manifestation of TGA is severe amnesia, the condition is typically benign and self-limiting, and recurrence is uncommon. Brain lesions associated with TGA are most commonly located in the vicinity of the hippocampus. More rarely, lesions in the thalamus, corpus callosum, and the right caudate nucleus have

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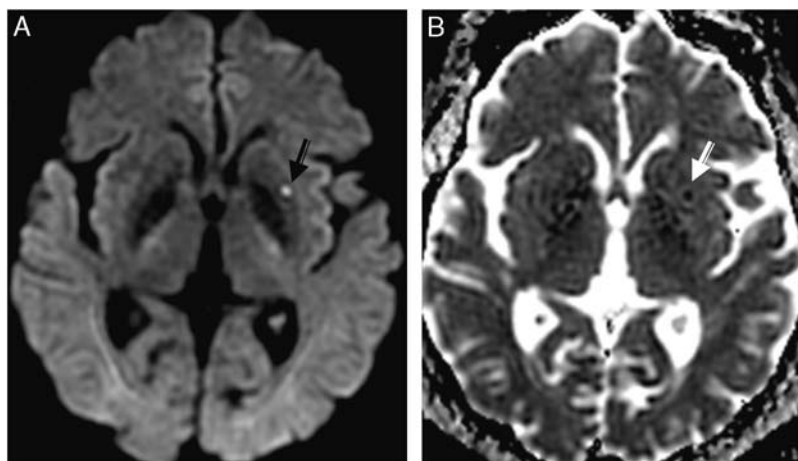


FIGURE 1. Diffusion-weighted imaging revealed a discrete and intense signal in the left putamen (A, black arrow). Apparent diffusion coefficient reduction was observed in the corresponding area (B, white arrow).

been associated with TGA.² To the best of our knowledge, lesions in the putamen have previously not been reported to be associated with TGA. In the present case, no hippocampal or thalamic abnormalities were noted, but a discrete lesion was seen in the left putamen. The presence of a highly localized and intense signal increase in the left putamen revealed by DWI, with concomitant reduction of ADC, suggests that the lesion is an acute microinfarct.

Pure ischemic stroke restricted to the putamen has only rarely been reported.⁴ Infarction limited to the putamen is

reported to affect specific cognitive operations, as revealed by conditions such as nonfluent aphasia or hemineglect.⁵ However, acute short-term memory deficit has been reported in cases of putaminal infarction, and here the left putamen is more commonly involved than the right.⁴ It was proposed that the short-term memory disturbances associated with putaminal lesions could indicate that the putamen is involved in the activation of memory processes.⁶ The basal ganglia and related structures have been implicated in specific cognitive processes including working memory, and lesions can give rise to diverse behavioral

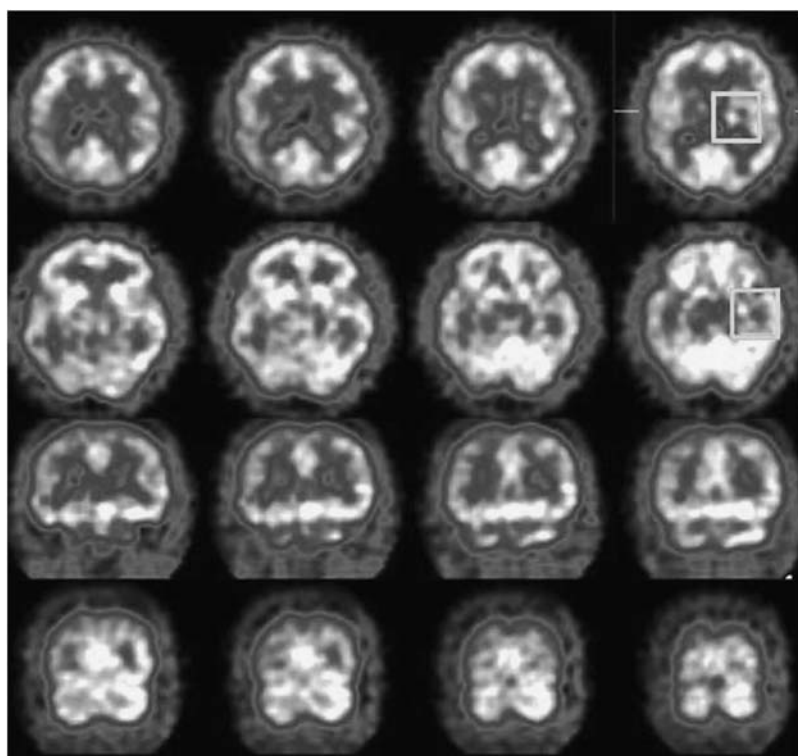


FIGURE 2. ^{99m}Tc-hexamethyl propyleneamine oxime brain single-photon emission computed tomography showing relatively increased perfusion in the left basal ganglia and temporal cortex (indicated by white rectangles).

symptoms. It is possible that such cognitive deficits reflect interruption of the dorsolateral prefrontal circuit projecting from Brodmann areas 9 and 10 (responsible for aphasia) and Walker area 46 (responsible for neglect) to the dorsolateral head of the caudate nucleus, with interconnections with the posterior parietal cortex (Brodmann area 7).⁷ Our case reinforces the contention that the putamen could also play a role in the processing of episodic memory.

Several studies argue that there is no clear relationship between TGA etiology and ischemic changes; however, the pathogenic role of an ischemic event is still in debate.^{8,9} Therefore, we conducted brain single-photon emission computed tomography imaging with ^{99m}Tc-hexamethyl propyleneamine oxime to evaluate the perfusion state of the brain 48 hours after the onset of symptoms. This revealed mildly increased perfusion in the left temporal cortex and basal ganglia. Findings of functional studies in the literature have not been consistent, and diverse changes in regional blood flow in the temporal lobe, thalamus, and frontal lobe have been reported during or after TGA.¹⁰ However, it remains uncertain in our case whether increased perfusion in these areas could reflect functional compensation for the acute ischemic change in the left putamen. We do not exclude the possibility that such perfusion changes taking place after TGA could contribute to the unique cognitive symptoms of TGA.

The lesion in this case was clearly identified with the delay in second imaging at 48 hours. This phenomenon has been well known in TGA, and it suggested the possibility that pathology was already present at the first MRI but was identified only once the signal change was prominent enough in the delayed sequence.¹¹ Since Strupp and colleagues first detected hyperintense lesions in the hippocampus in TGA using DWI, the frequency of lesions detected on DWI has been reported in a range of 0% to 84%.^{2,11–15} In addition, a recent study reported that TGA patients with a DWI lesion have a vascular risk profile and concomitant changes on brain MRI compared with those without DWI lesions.¹⁶

In the present case, there was an incontrovertible temporal association between the amnesic episode and acute lesion in the left putamen, raising the possibility that the putamen could play a role in episodic memory processing. Prospective, systemic investigation of several similar cases will be required to establish a possible role of the putamen in higher cognitive function.

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