Evidence of Acute Ischemic Tissue Change in Transient Global Amnesia in Magnetic Resonance Imaging: Case Report and Literature Review


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ABSTRACT

Transient global amnesia is a benign syndrome of sudden onset alteration of behavior with temporary dysfunction of anterograde and recent retrograde memory. Its neural substrates remain uncertain. Possible causes include ischemia, migraine, and epilepsy. The authors report a case of a 62-year-old man with a transient attack of memory disturbance, suggestive of transient global amnesia, in which magnetic resonance imaging performed 48 hours after onset showed left mesial temporal lobe signal changes on diffusion-weighted imaging and fluid-attenuated inversion recovery images. The findings and a literature review lend further support to the ischemic pathogenesis of transient global amnesia as a possible etiology, and underscore the role of diffusion-weighted imaging in the diagnosis of this condition.

Key words: Magnetic resonance imaging (MRI), amnesia, ischemic, diffusion-weighted magnetic resonance imaging (DWI).

Case Presentation

A 62-year-old man complained of an episode of memory loss the previous day, during which he appeared confused but remained alert and oriented, asking the same questions repeatedly, not able to retain new information. No motor weakness, uncoordination, speech slurring, or other neurological symptoms were noted. After 4 hours, the patient’s behavior returned to normal.

Thirty hours after the event, the patient was alert and attentive, with normal language and visuospatial functions. The patient encoded 6 words and retrieved them without difficulty after 30 minutes but remained completely amnestic of the previous morning’s events. He suffers from mild hypertension with satisfactory control by treatment, and his clinical neurological examination was unremarkable.

MRI 48 hours after the episode (1.5T GE Signa Advantage), including DWI and FLAIR sequences, revealed a small region of signal hyperintensity in the left hippocampus (Fig 1). MR angiography of extracranial and intracranial vessels, an awake and sleep electroencephalogram (EEG), a transthoracic echocardiogram, and routine lab tests were normal. A follow-up MRI after 3 weeks did not show signal abnormalities in any sequences, despite devote thin acquisition in axial and coronal planes on DWI and FLAIR acquisitions (Fig 2).

Discussion

Fisher and Adams3 coined the term transient global amnesia in 1958, reporting 17 cases of a transient memory disturbance, and discussed the possible causes. Others have described similar cases.2 The diagnosis of TGA is established on a clinical basis, as...

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proposed by Caplan et al, later modified and validated. TGA incidence ranges from 3 to 32 per 100,000 per year. Incidence peaks above age 50, without gender preference.

Clinical features consist of sudden-onset anterograde episodic memory impairment, not accompanied by focal neurological findings, lasting less than 12 hours. The patient classically repeats the same questions and is disoriented to time and sometimes location, with preserved self-awareness, attention, and working memory. Occasionally, mild impairment in retrograde memory is noted. There is a permanent memory gap for the period of the attack.

There is still limited knowledge regarding the site and etiology of TGA. There is evidence that severe anterograde amnesia is related to bilateral dysfunction of mesial temporal lobe structures or diencephalon or basal forebrain dysfunction. The role of unilateral lesions causing amnesia remains uncertain. A recently published large TGA case series showed a unilateral lesion in the hippocampus in the majority of the cases, with a marked predominance of left-side involvement. The etiology of TGA is probably heterogeneous, and the involvement of cerebrovascular disease is controversial. Similarities between TGA episodes and temporal lobe seizures led to an interpretation of TGA as seizures. EEGs performed during and after TGA episodes failed to disclose epileptiform discharges, arguing against this possibility. Other studies suggest a high prevalence of migraine in TGA patients, suggesting a connection between TGA and migraine. Our patient did not have a history of migraine or evidence of seizure.

DWI has been recently employed in the investigation of TGA. This technique has proved to be an important tool and a very sensitive method to demonstrate cytotoxic edema induced by cerebral infarct. Diffusion signal changes are not specific of infarction, and apparent diffusion coefficient (ADC) map analysis is crucial to differentiate restricted diffusion and T2 shine-through effect. Decreased diffusion appears as hyperintense signal changes on DW images and hypointense signal on ADC maps. ADC values return to baseline in 1 to 4 weeks.

Experimental models also show transient ADC reduction in spreading depression and epilepsy, but this finding has never been observed in humans.

MR examination of our patient was obtained 48 hours after symptom onset. DWI and FLAIR images showed focal hyperintensity in the left hippocampus, with corresponding low signal on ADC maps, indicating restricted water diffusion. DWI signal hyperintensity in medial temporal structures during or immediately after TGA attacks has been reported in the literature and is in line with our findings.

Strupp et al considered DWI as a sensitive method able to evaluate early stages of TGA. MRI findings are therefore not specific for ischemia. Likewise, others reported reversible increased signal intensity in DWI and T2WI in the right hippocampus of a patient who suffered a spontaneous TGA attack 44 hours earlier.

Woollenden et al described a patient with a TGA-like syndrome after cerebral angiography. MRI DWI and T2WI showed areas of high signal intensity, including the right hippocampus and bilateral occipital lobes, which resolved in 2 weeks. It was considered an indication of ischemia and speculated that the transitory nature of the signal alteration was due to the small dimension of the infarct area, rendering it impossible to detect in a follow-up MRI. Our case also showed resolution of the lesions in the left hippocampus on follow-up MRI performed 3 weeks later, even using thinner (3-mm) slices without gaps on DWI and FLAIR sequences.

Other studies have not detected signal abnormalities in MRI DWI obtained from patients with TGA. This can be due to heterogeneity of the underlying mechanism of TGA or the time of MRI acquisition.

Sedlaczek et al showed punctate lesions on lateral aspect of the hippocampal formation (pes and fimbria) in the majority of their cases. DWI changes were noted only in a 24- to 48-hour period after the TGA attack. The authors suggested a delayed ischemic mechanism, with high metabolic rates leading to relative hypoperfusion in the hippocampal vascular border zone. Similarly, we were able to demonstrate lesions in the same site on the late MR acquisition (48 hour) using DWI and FLAIR sequences.
Our findings and previous reports lend further support to the ischemic etiology in a subgroup of patients with TGA. We emphasize that DWI should be used routinely in the appropriate timing in TGA, with devoted hippocampal acquisitions to detect characteristic findings suggestive of ischemia. The sensitivity and specificity of DWI changes in the diagnosis and the pathogenesis of TGA currently remain incompletely understood and should be the object of further studies.

References