Case Report

Transient Hyperintensity On Diffusion-Weighted MRI in Transient Global Amnesia

Gülçin BENBİR¹, Sibel ERTAN¹, Sait ALBAYRAM², Batuhan KARA², Hakan SELÇUK², Turan ERTAN³

¹Istanbul University Cerrahpasa Faculty of Medicine, Departments of Neurology, Istanbul, Turkey ²Istanbul University Cerrahpasa Faculty of Medicine, Departments of Radiology, Istanbul, Turkey ³Istanbul University Cerrahpasa Faculty of Medicine, Departments of Psychiatry Istanbul, Turkey

Abstract

Objects: Different theories were suggested on the etiopathogenesis of transient global amnesia.

Methods: We demonstrate a patient with transient global amnesia, in whom, a transient hyperintense signal change on left hippocampus was detected in diffusion- and T2-weighted magnetic resonance images.

Conclusion: The absence of vascular risk factors or atherosclerotic changes in neuroimaging, in addition to transient lesion and antecedent migraine attack suggest the phenomenon of “spreading depression” secondary to migraine as underlying etiopathogenetic mechanism of transient global amnesia in this patient.

Keywords: Transient global amnesia, diffusion-weighted MRI, spreading depression

Geçici Global Amnezide Difüzyon-Ağırlıklı MRI'da Geçici Hiperintensite

Özet

Giriş: Geçici global amnezin etiyo-patogenezinde pek çok farklı hipotezler öne sürülmüştür.

Metot: Bu yazida, difüzyon ve T2 ağırlıklı kraniyal MRI incelemesinde sol hipokampusta geçici hiperintensite saptanan bir geçici global amnezi olgusu sunulmaktadır.

Sonuç: Hastada vasküler risk faktörlerinin olmaması, nöro-görüntülemede aterosklerotik değişikliklerin görülmemesi, ve geçici lezyon öncesinde migren atağının olması bu hastada geçici global amnezinin etiyo-patogenetik mekanizması olarak migrene sekonder gelişen “spreading depression” teorisini desteklemektedir.

Anahtar Kelimeler: Disferlin, disferlinopati, distal miyopati, limb-girdle musküler distrofi, distal anterior kompartman miyopati

INTRODUCTION

Transient global amnesia (TGA) is characterized by an abrupt onset of both anterograde and retrograde amnesia. Immediate recall ability is preserved, as is remote memory; however, patients experience striking loss of memory for recent events, and an impaired ability to retain new information. These memories are regained progressively as the patient recovers typically within less than 24 hours⁽¹⁾.

The precise pathophysiology of TGA is still not clear, while some hypothesis on localization and etiopathogenesis have been suggested on the basis of imaging studies. Studies using single photon
emission computed tomography (SPECT), positron emission tomography (PET) and magnetic resonance imagings (MRI) have suggested that TGA results from a transient decrease in blood flow to specific brain areas that involve memory\(^{(11)}\). The most tenable hypotheses that have been put forward so far to explain the pathogenesis of TGA are transient ischemic attack (TIA) in the inferomedial parts of the temporal lobes, epileptic discharge in the hippocampus, or recently, spreading depression of cortical electrical activity\(^{(14)}\). We here demonstrate a patient who experienced a migraine attack followed by TGA, and had a hyperintense signal change on left hippocampus in both diffusion weighted images (DWI) and T2-weighted images (T2WI), which disappeared in follow-up MRI studies.

**CASE PRESENTATION**

A 55-year-old right-handed woman is admitted to our neurology department complaining of memory loss. The patient, who was an academician, mentioned that during her speech in a meeting she had a three hours of blank-period, consisting about 2 hours of anterograde and posterograde amnesia. She had no memory of having such an conversation, and in video recordings of the meeting, authors watched that she tried to answer the same question for four times, which caused mockery and laughing. She stated that her lecture was far beyond her usual performance, with more pauses, and disarranged answers. She added that in the morning of the event, she had her typical migrinous headache, which diminished after 2 hours without any medication.

In her past medical history, she had fibromyalgia and migraine for 40 years. She was not on any medication. She was non-smoker, and non-drinker. Family history was unremarkable. Her physical and neurologic examination were normal. Routine laboratory investigations, including complete blood count, electrolytes, liver and thyroid function tests were within normal limits. Electrocardiography, transthoracic echocardiography, and extracranial carotid doppler examination were normal. Cranial MRI performed within the first 24 hours showed left hippocampal lesion hyperintense in DWI, T2WI, and flair-weighted cessions, and hypointense in apparent-diffusion coefficient (ADC) mapping (Figure 1). In the control MRI after 37 days, no abnormal intensity was observed (Figure 2).

*Figure 1:* There is a hyperintensity in the left temporal lobe on the DWI, associated with a diminished ADC.  
*Figure 2:* There is normalization of findings and no more changes on DWI.
DISCUSSION

Although many mechanisms have been proposed, no single cause can fully explain all features of TGA. Suggested mechanisms include TIA, temporal lobe seizure, and migraine variant. Many authors have accepted the hypothesis that TGA is cerebrovascular in origin, caused by a thromboembolic occlusion, vasospastic, vasomotor, or hemodynamic mechanisms, or a lacunar infarction. Transient ischemia in the territory of the posterior cerebral artery, or alternatively of the anterior choroidal artery, causing dysfunction in the deep limbic system involved in memory, has been suspected of being responsible for TGA. Studies controversial to support TIA hypothesis showed much less incidence of risk factors and much better prognosis in TGA. In our patient, cerebrovascular risk factors such as hypertension, diabetes mellitus, smoking were absent, and hyperlipidemia was under control with no treatment.

Some authors have suggested an epileptic origin on the basis of abrupt onset, brevity and reversibility of the attack, combined with electroencephalographic (EEG) abnormalities in some instances. However, the duration of the attack, the absence of impairment of consciousness, and lack of repetitiveness do not yield an epileptic origin. Furthermore, EEG recordings obtained during typical TGA attacks are usually normal except some rare case reports of epileptiform discharges.

Recently, many authors emphasized the association between TGA and migraine. Cerebral blood flow abnormalities after TGA episodes were found similar to those found in migraine, but different from those seen in patients with TIA. It is interesting to note that the phenomenon of spreading depression of cortical activity described by Leao produces similar changes in experimental animals. It has also been suggested that TGA and migraine share this particular common pathogenetic mechanism. As precipitated by intense sensory inputs to the hippocampus, a surge of excitotoxic neurotransmitter glutamate temporarily blocks the normal memory function in the hippocampus.

Imaging studies may also be helpful in explaining the underlying mechanisms of TGA. Although transient hypoperfusion in SPECT studies is a well-known phenomenon in TGA, MR studies are still questioned. There have been four different MRI patterns reported in patients with TGA: (i) no pathologic signal change in both conventional and DWI, (ii) presence of pathologic signal change in both conventional and DWI that disappears during follow-up, (iii) pathologic signal change DWI without any change in T2WI, and (iv) permanent pathologic signal change in both conventional and DWI. These findings have been studied to imply different pathophysiologic mechanisms underlying TGA. Table 1 summarizes the imaging findings observed in a scarce number of TGA cases. Two prospective MRI studies demonstrated the absence of any abnormal lesion in DWI at early stages of TGA. Four case reports were included in the literature, showing acute signal changes in both DWI and T2WI. Pathologic signal change DWI without any change in T2WI was reported by Strupp et al. Lastly, three case reports showed permanent DWI lesions in TGA patients.

In this case report, we demonstrated a hyperintense change on left hippocampus in DWI, which disappeared at 37th day following the event. Although the possibility of “invisible” sequela of small infarction could not totally be excluded, it is accepted that one month is rather early. The absence of vascular risk factors, the absence of atherosclerosis and ischemic changes in cranial MRI, and antecedent migraine attack before the amnestic event, in addition to transient DWI lesion support
the presence of “spreading depression” secondary to migraine as underlying etiopathogenetic mechanism of transient global amnesia in our patient.

Table 1. The summary of imaging studies in patients with TGA.

<table>
<thead>
<tr>
<th>Literature</th>
<th>Cases</th>
<th>Imaging</th>
<th>Lesion</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matsui M et al., 2002</td>
<td>1 case report</td>
<td>DWI + T2WI</td>
<td>Right hippocampal changes</td>
<td>Normal &gt;2 weeks</td>
</tr>
<tr>
<td>Jeong Y et al., 2003</td>
<td>1 case report</td>
<td>DWI + T2WI</td>
<td>Left hippocampal changes</td>
<td>Normal &gt;56 days</td>
</tr>
<tr>
<td>Huber R et al., 2002</td>
<td>10 patients</td>
<td>DWI (6-44 hour)</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Gass A et al., 1999</td>
<td>8 patients</td>
<td>DWI (1-8 hour)</td>
<td>Left hippocampal changes</td>
<td></td>
</tr>
<tr>
<td>Woolfenden AR et al., 1997</td>
<td>case report</td>
<td>DWI + T2WI</td>
<td>Right hippocampal changes</td>
<td>Normal &gt;2 months</td>
</tr>
<tr>
<td>Ay H et al., 1998</td>
<td>1 case report</td>
<td>DWI + T2WI</td>
<td>Left parahippocampal and splenial changes</td>
<td>Normal at control</td>
</tr>
<tr>
<td>Strupp M et al., 1998</td>
<td>10 patients</td>
<td>DWI</td>
<td>Left / bilateral hippocampal changes</td>
<td>Normal &gt;2 weeks</td>
</tr>
<tr>
<td>Greer DM et al., 2001</td>
<td>1 case report</td>
<td>T1WI + T2WI</td>
<td>Left mesial temporal lobe Permanent acute infarction (emboli)</td>
<td></td>
</tr>
<tr>
<td>Saito K et al., 2003</td>
<td>1 case report</td>
<td>DWI</td>
<td>Left retrosplenium acute infarction</td>
<td></td>
</tr>
<tr>
<td>Bartsch et al., 2006</td>
<td>41 TGA patients</td>
<td>DWI + T2WI</td>
<td>Hippocampal lesions</td>
<td>Normal &gt; 20 days</td>
</tr>
<tr>
<td>Presented case</td>
<td>1 case report</td>
<td>DWI + T2WI</td>
<td>Left hippocampal changes</td>
<td>Normal &gt;37 days</td>
</tr>
</tbody>
</table>

DWI = Diffusion-weighted imaging; T2WI = T2-weighted imaging; T1WI = T1-weighted imaging.

Correspondence to:
Gulcin Benbir
E-mail: drgulcinbenbir@yahoo.com

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