Seizure 31 (2015) 141–143

Contents lists available at ScienceDirect

Seizure

journal homepage: www.elsevier.com/locate/yseiz

Clinical letter

Transient global amnesia with a hippocampal lesion followed by transient epileptic amnesia

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ARTICLE INFO

Article history:
Received 14 June 2015
Received in revised form 24 July 2015
Accepted 24 July 2015

1. Introduction

Transient epileptic amnesia (TEA) is a seizure disorder characterized by brief, recurrent attacks of amnesia in middle-aged or elderly subjects, often occurring on waking, with favorable response to anticonvulsant medication [1]. While this syndrome is becoming increasingly recognized, its association with transient global amnesia (TGA) is not fully understood. It remains unknown whether TEA is a separate entity from recurrent TGA, or whether TGA can be a cause of TEA. We report a patient initially presenting with TGA and a hippocampal lesion on magnetic resonance imaging (MRI), followed by TEA episodes, suggesting that this damaged region associated with TGA may be an epileptic focus resulting in TEA.

2. Case report

A 75-year-old right-handed woman presented to our hospital with an abrupt onset of confusion and forgetfulness. Her past medical history included cataract and hyperlipidemia. She had previously not suffered from epileptic seizures. In the evening, she excitedly discussed about the neighborhood association with her husband and neighbors. After the neighbors left at 7 pm, she abruptly started asking repetitive questions, such as "What happened"? and "What are we doing here"?

A neurological examination demonstrated that she was awake and alert. Anterograde and retrograde amnesia were observed; she could neither remember her blood pressure being measured just before the examination nor current events from a couple of days prior. The remainder of the neurological examination was normal. She was suspected of having TGA and discharged. Her symptoms diminished at midnight. The following day, the neurological examination was normal, and the mini-mental state examination score was 30/30. A routine scalp electroencephalogram (EEG) was normal. Brain MRI 5 days after the episode revealed a small hyperintense lesion in the right hippocampus on diffusion-weighted imaging (DWI) (Fig. 1); thus, she was diagnosed with TGA.

Five months after the first episode, on waking, she again abruptly started asking repetitive questions, such as "What happened". She forgot that she had brought a towel to wash her face and asked her husband "Who brought this towel"? This episode lasted approximately 30 min. EEG taken 3 days after the episode showed spikes in the temporofrontal areas (Fig. 2), and carbamazepine (CBZ) (400 mg/day) was prescribed. After 8 months, she presented another amnesic episode that lasted 30 min in the early evening. During this episode, her husband reported that she presented a short episode of unresponsiveness while standing at the beginning of the amnesic episode. CBZ was increased to 600 mg/day. No amnesic episodes have occurred over the past 19 months. At the follow-up MRI (30 months after the first one), the right hippocampal hyperintense lesion on DWI

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http://dx.doi.org/10.1016/j.seizure.2015.07.020

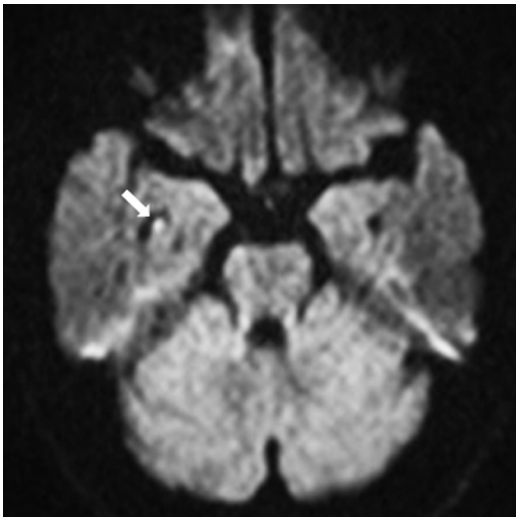

Fig. 1. Diffusion-weighted magnetic resonance imaging scan in the axial plane shows a punctate high-signal intensity lesion in the right hippocampus (white arrow).

disappeared, and no abnormal signals were observed in the hippocampus on the T2-weighted images.

3. Discussion

Our patient presented three transient amnesic episodes. The first episode was relatively long in duration (5 h), triggered by emotional stress, and not accompanied with other ictal manifestations, such as hallucinations, automatisms, and unresponsiveness. Brain MRI revealed a small hyperintense lesion in the hippocampus on DWI. Postictal EEG showed no epileptiform abnormalities. These characteristics coincided well with TGA [2].

On the other hand, the patient experienced two further episodes of transient amnesia; these episodes were of short duration (<30 min), occurred on waking, were accompanied by a brief episode of unresponsiveness on one occasion, and were successfully treated by anticonvulsant monotherapy. Interictal EEG showed right temporofrontal spikes. These transient amnesia attacks after the first episode satisfied the proposed criteria of TEA and coincided well with previously reported characteristics of TEA [1].

A brain MRI taken after the first episode showed a small hyperintense lesion in the hippocampus. In TGA, focal hyperintense lesions on DWI and T2-weighted images can be reliably

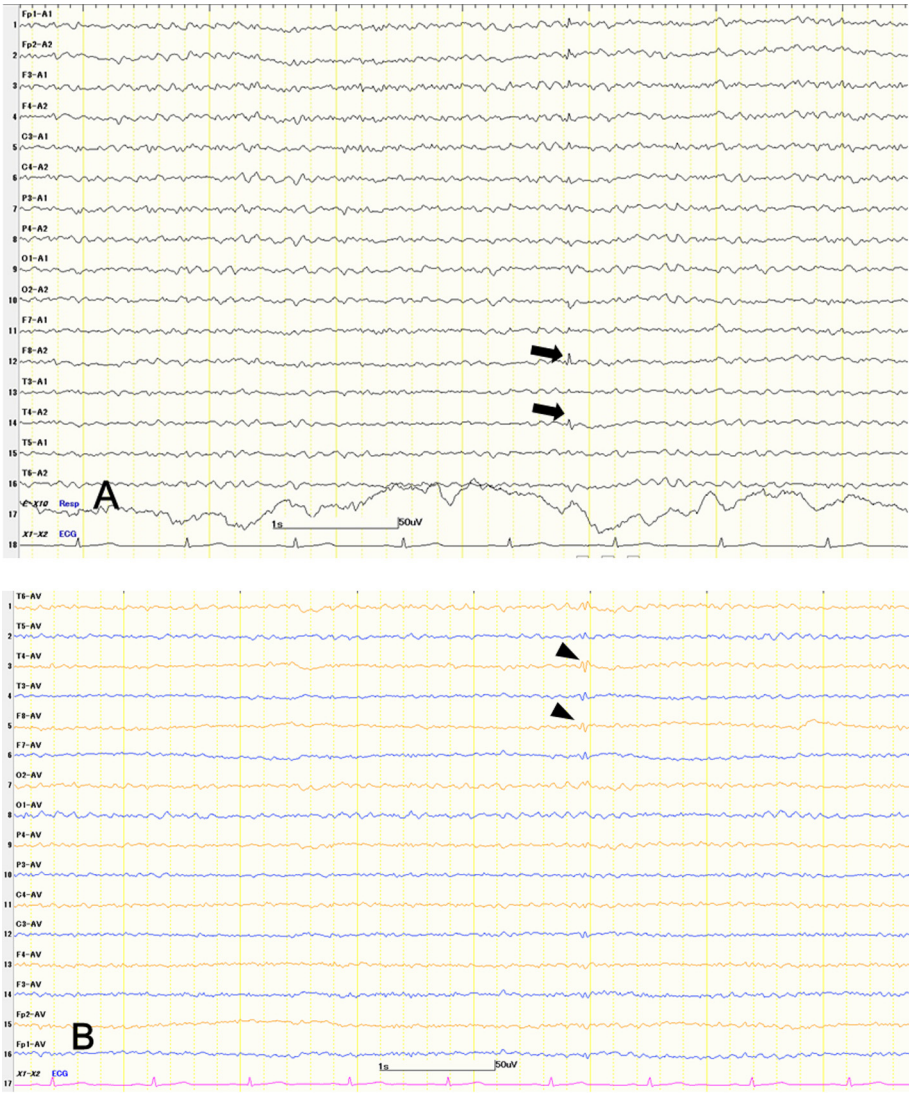

Fig. 2. Electroencephalogram taken 3 days after the second amnesic episode shows small spikes in the temporofrontal areas (A, black arrows and B, black arrowheads).

detected in the hippocampus [2]. A detailed analysis of the location of these lesions showed that almost all lesions were selectively found in the area corresponding to the CA1 section of the hippocampal cornu ammonis [3]. In TEA, although subtle hippocampal volume loss is identified by manual volumetry, clinical neuroimaging with brain MRI is usually unremarkable [1]. To the best of our knowledge, there has only been one case of TEA showing a hyperintense lesion in the hippocampus on DWI [4]; however, this lesion was more extensive than TGA lesions. The nature of hippocampal abnormality and the pathophysiological mechanisms of TGA remain unclear. Several mechanisms have been suggested, such as a migraine-related mechanism, hypoxic-ischemic events, and venous flow abnormalities that may lead to impaired cellular diffusion in the hippocampal lesion. Although this hippocampal abnormality is completely reversible, persistent functional impairment of this lesion has also been shown [3]. It seems that this damaged region of the hippocampus could be the seizure focus of TEA. There is also the possibility that TEA occurred with TGA by coincidence; however, this is unlikely because of low prevalence of both disorders.

We report a case of TEA with the first amnesic episode presenting as TGA with pathognomonic findings from the brain MRI. It is thought that the hippocampal lesion that was damaged during the TGA episode could be the seizure focus of TEA.

Conflict of interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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