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Letter to the Editor

Transient Epileptic Amnesia With Amygdala Enlargement Presenting to a Dementia Clinic

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Conflict of Interest
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Dear Editor,

Transient epileptic amnesia (TEA) is a distinct syndrome of late-onset limbic epilepsy of unknown cause, typically occurring in old age. It is an important cause of memory loss in older people because it could be treatable. However, it is often mistaken for neurodegenerative disease, transient global amnesia (TGA), cerebrovascular disease, and functional amnesia because amnesia is the only manifestation in some patients, unaccompanied by symptoms such as olfactory hallucination, motor automatisms, or brief unresponsiveness.[1] In some patients with TEA, suspected causative abnormalities are detected by magnetic resonance imaging (MRI). These most commonly involve the mesial temporal lobes,[2] which might provide some hints to clinicians in dementia clinics for diagnosing TEA. Here, we report the case of a patient with TEA accompanied by amygdala enlargement who presented to a dementia clinic.

A 62-year-old, right-handed, and 9-year-educated man presented with recurrent episodic memory loss. For the past 5 years, he had occasionally experienced short-duration memory loss. In the last 9 months, there were 4 transient amnestic episodes lasting from a few to thirty minutes, which were witnessed by his wife. In a representative episode, he experienced transient amnesia the day after his birthday party at his son's house. He was unable to remember sleeping at his son's house and kept asking, "Where am I?" "Why am I here?" or "What day is it today?" Otherwise, he was responsive and did not show any automatisms. He did not feel any aura or mood change. He was on medication for dyslipidemia and hypertension. His physical and neurological examinations were unremarkable. Differential diagnoses including TEA, TGA, transient ischemic attacks, and neurodegenerative diseases were considered. In the Korean Mini-Mental State Examination, he scored 29 out of 30. His performance in detailed neuropsychological evaluations was within the normal range, considering his age and education. His electroencephalogram (EEG) captured one event of electrographic seizure, which originated from the right temporal area (**Fig. 1A**), and several interictal sharp waves in bilateral temporal areas (**Fig. 1B and C**). His brain MRI (**Fig. 1D-F**) showed amygdala enlargement with hyperintensity on fluid-attenuated inversion recovery images, which was more remarkable on the right side. As differential diagnoses for etiologies of amygdala enlargement, we considered autoimmune/paraneoplastic encephalitis, tumor (glioma and astrocytoma), and others. Therefore, we performed further tests. The results of the chest and abdominal computed tomography, as well as autoimmune and a series

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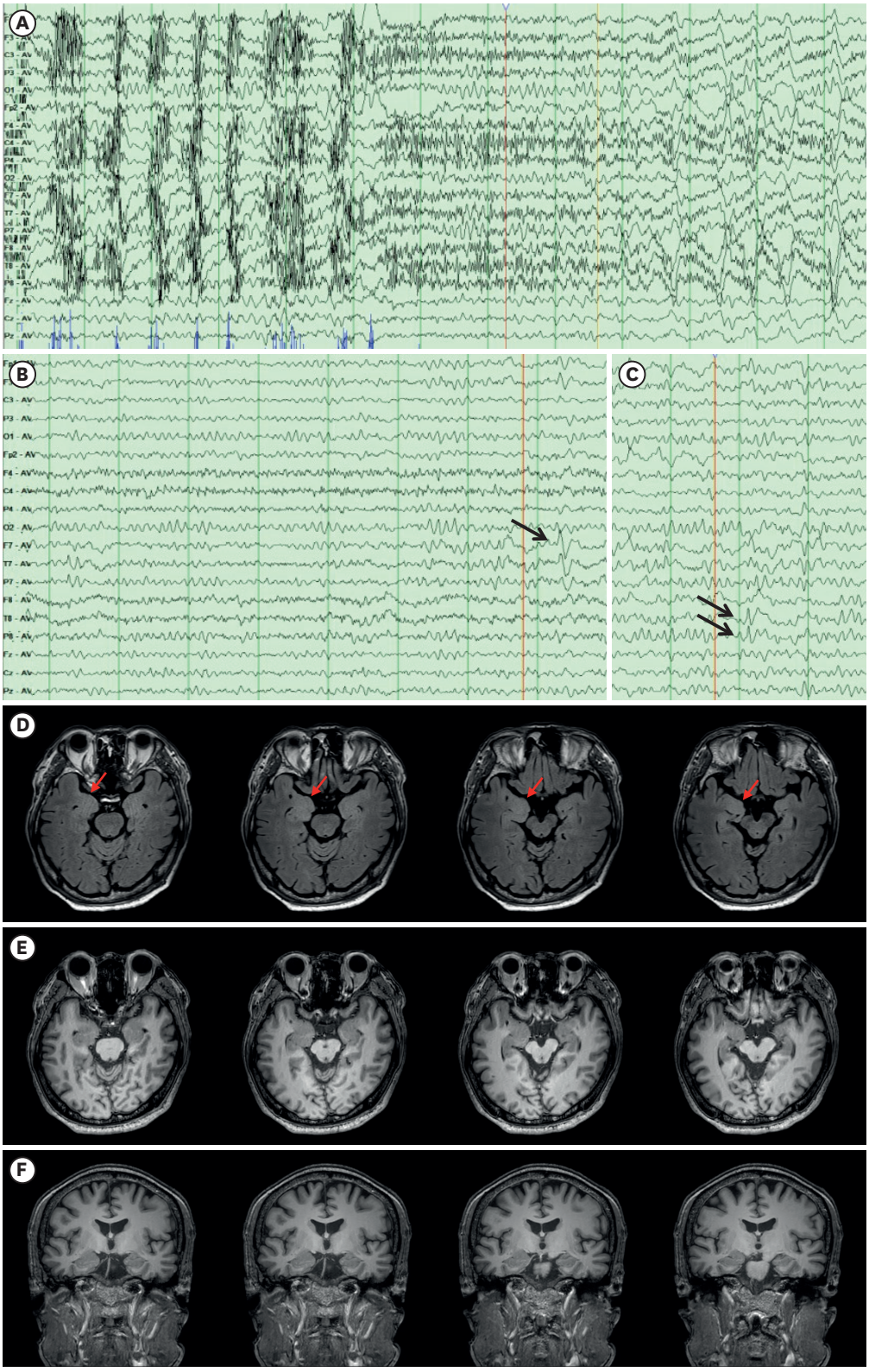

Fig. 1. Overnight EEG video and brain MRI findings of the patient. The EEG captured one event of electrographic seizure, which originated from the right temporal area (A) and several interictal sharp waves (black arrow) in bilateral temporal (T8 or P8; F7) areas (B, C). Axial FLAIR images (D), axial T1-weighted brain MRI (E), and coronal T1-weighted brain MRI (F) showed amygdala enlargement (red arrow) with high signal intensity in the FLAIR images.
EEG: electroencephalogram, MRI: magnetic resonance imaging, FLAIR: fluid-attenuated inversion recovery.

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of paraneoplastic antibody tests (lupus anticoagulant test, anti-nuclear antibody, anti-neutrophil autoantibodies, anti-Hu, anti-Ri, anti-Yo, anti-amphiphysin, anti-collapsing response-mediator protein-5, anti-paraneoplastic Ma antigen 2, anti-recoverin, anti-glial nuclear antibody, and anti-Titin) were unremarkable. Based on the criteria proposed by Zeman et al.,[3] the patient was diagnosed with TEA. He remained free from transient amnesic episodes after treatment with oxcarbazepine at 600 mg/day.

Our patient was diagnosed with TEA accompanied by amygdala enlargement and remarkable ictal EEG changes, which originated from the right temporal areas. Autoimmune or limbic encephalitis was excluded due to repetitive episodes over 5 years and no evidence of malignancy or related antibodies. TEA can be differentiated from TGA by the shorter duration of the episodes.[4] However, due to the lack of communication between clinicians and patients or caregivers, a diagnosis of TEA might be missed at first and only considered after seeing the suspected causative abnormalities on MRI such as mesial temporal lobe signal abnormalities, temporal cavernous hemangioma, a small hyperintense lesion in the hippocampus, and enlarged hippocampal volume with the loss of architecture and an increased hippocampal tail signal.[1] Furthermore, only a few case reports showed amygdala enlargement in patients with TEA, which was seen in our patient.[5-7] Among 3 reported patients with TEA with amygdala enlargement, 2[5,7] showed a decrease in the size of the amygdala after treatment with an anticonvulsant. Recently, increasing numbers of cases of temporal lobe epilepsy (TLE) with amygdala enlargement have been reported and attracted attention as a novel TLE subtype. According to the results of a systematic review, TLE with amygdala enlargement is characterized by later age at onset, a higher frequency of complex partial seizures compared to convulsive seizures, and excellent response to antiepileptic drugs.[8] Although the pathological characteristics of TLE with amygdala enlargement have not been well described, a few reports of associated neoplastic diseases such as glioma or astrocytoma have been published. Surgical treatment is considered if amygdala enlargement develops or if the response to antiepileptic drugs is poor.

In conclusion, this case suggests that clinicians need to consider the possibility of TEA when they encounter patients presenting with transient amnesia and that TEA may be accompanied by enlargement of the amygdala.

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