Case Report
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Epilepsy Mimicking Affective Disorder in a Patient with Amygdala Enlargement

Jung-Ju Lee, MD, PhD, Kyusik Kang, MD, PhD, Jong-Moo Park, MD, PhD, Woong-Woo Lee, MD, PhD, Ohyun Kwon, MD, Byeong-Kun Kim, MD, PhD

Department of Neurology, Nowon Eulji Medical Center, Eulji University College of Medicine, Seoul, Korea

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Corresponding author: Jung-Ju Lee, MD, PhD
Department of Neurology, Nowon Eulji Medical Center, Eulji University College of Medicine, 68 Hangeulbiseok-ro, Nowon-gu, Seoul 01830, Korea
Tel. +82-2-970-8312
Fax. +82-2-970-8862
E-mail; sss331@eulji.ac.kr

Affective disorders are commonly associated with epilepsy. Affective symptoms rarely occur concomitantly with seizure occurrence, which can lead to misdiagnosis. Here, we describe a 69-year-old man who experiencedintermittent manifestations of unpleasant mood and aggressive behavior. He had temporal lobe epilepsy with amygdala enlargement. After successful treatment with an antiepileptic drug, hissymptoms resolved.Additionally, the amygdala enlargement decreased when checked at 5 years after treatment. We discuss the clinical characteristics and differential points of the case. **(2019;9: 83-86)**

Key words: Epilepsy, Amygdala

Introduction

Affective disorders, including depression, bipolar disorder, and anxiety disorder, are common in patients with epilepsy. Emotional symptoms associated with these disorders are typically found independent of seizure occurrence; thus, they are known asinterictal affective disorders. Patients may rarely experience symptoms of affective disorder exclusively around the time of seizure occurrence: either preceding the seizure (preictal); following the seizure (postictal); or as an expression of the seizure (ictal).[1] In such situations, symptoms of affective disorders comprise the presenting symptoms of epilepsy and should be differentiated from symptoms caused by affective disorders. Previous studies have shown that temporal lobe epilepsy (TLE) with amygdala enlargement (AE) (TLE-AE) is often associated with ictal and interictal emotional disturbances.[2-4] Herein, we describe a patient with TLE-AE who was initially misdiagnosed with affective disorder. The ensuing discussion addresses the clinical characteristics and differential points to distinguish emotional disturbances in TLE-AE from other psychiatric disorders.

Case

A 69-year-old man with no previous illness visited the department of psychiatry of the authors' hospital with intermittent development of an unpleasant feeling. This symptom was typically followed by aggressive behaviors, such as cursing and fighting with his acquaintances, which lasted for only a few minutes. He could partially recall the events after they occurred but did not understand why they had occurred. Notably, his symptoms had developed after a particularly stressful event: he had been blackmailed by an acquaintance regarding a financial issue. He was initiallydiagnosed with atypical depression and had been treated with antidepressants and antipsychotics (amisulpride 50 mg/day and escitalopram 10 mg/day) for 1 year; however, he did not exhibit improvement.

Subsequently, as the patient preached a sermon in church, he lost consciousness, which was preceded by the unpleasant feeling. He was then referred to the department of neurology. During meticulous history taking, his family explained that the aggressive behaviors were sometimes observed after a brief duration of behavioral arrest with a motionless stare. Electroencephalography (EEG) and magnetic resonance imaging (MRI) were performed. Epileptiform discharges were observed in the left temporal region on EEG (Fig. 1), and enlargement of the left amygdala was observed with slight hyperintensity on T2-weighted MRI (Fig. 2) without enhancement.

The symptoms completely disappeared after treatment with an antiepileptic drug (topiramate 100 mg/day). Antidepressants and antipsychotics were gradually tapered and discontinued 6 months later. The patient has been symptom-free for 5 years. Follow-up MRI revealed decreased left amygdala volume, compared with that in previous images (Fig. 3).

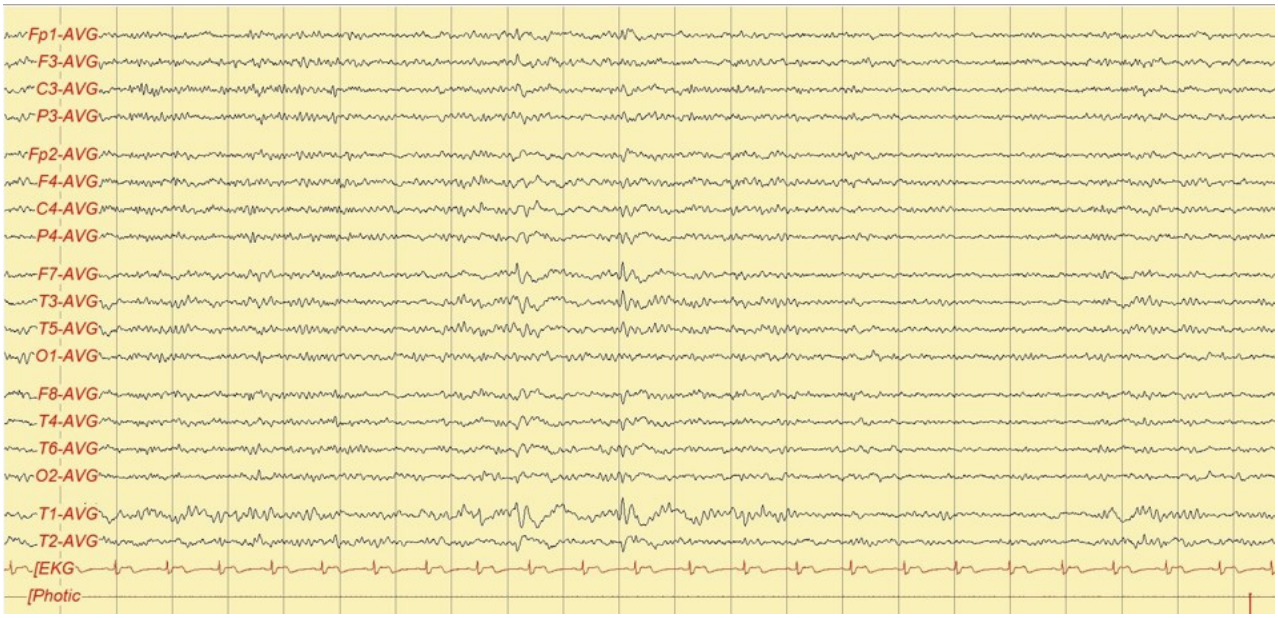

Figure 1. Electroencephalogram revealing epileptiform discharges in the left temporal leads.

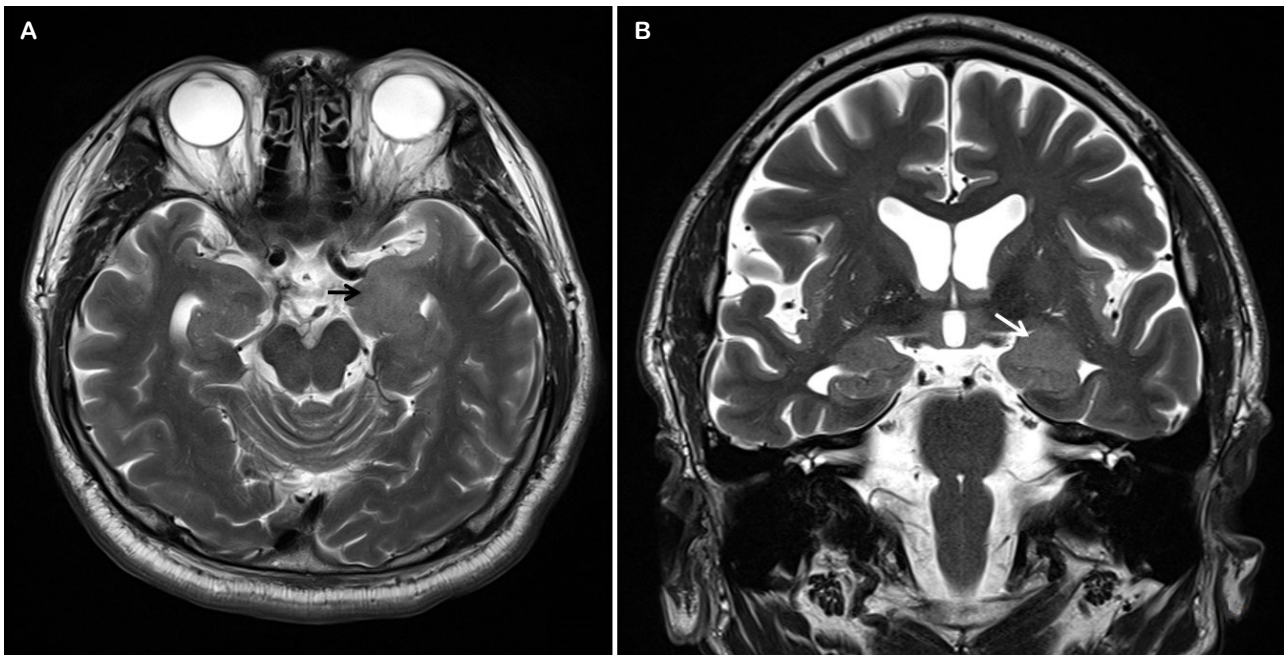

Figure 2. Axial (A) and coronal (B) T2-weighted magnetic resonance imagingrevealing increased signals in the left amygdala with enlargement (black and white arrow).

Discussion

Affective disorders, such as depression, are the most common disorders among patients visiting clinics.[5,6] Rarely, affective symptoms develop as a consequence of organic brain disorders. In the patient described, emotional seizures occurred after a stressful event, which led to a misdiagnosis of a psychiatric disorder. In patients with TLE, partial preservation of consciousness during seizure can occur.[7,8] Meticulous history-taking and adequate diagnostic work-up are both necessary to ensure correct diagnosis.

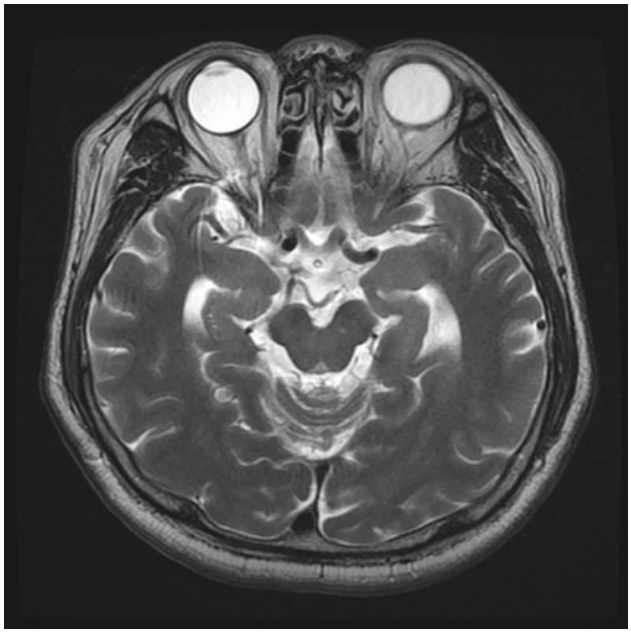

Figure 3. Follow-up magnetic resonance imagingdemonstratingthat the size of the amygdala has decreased compared with that depicted in previous images.

Epilepsy often can be misdiagnosed as a mood disorder in patients with emotional seizures without convulsive seizures. Emotional symptoms may occur as preictal, ictal, or postictal.[1] In the patient described in the present case, his unpleasant mood may have been a preictal or ictal symptom, while his aggressive behavior may have been a postictal symptom. Video-EEG monitoring during seizure events, however, would be necessary to confirm our hypothesis.

The amygdala is often the focus for the initiation of seizures, or the area responsible for their propagation.[2-4] Activation of the amygdala during seizures can generate various emotional symptoms. Moreover, the amygdala plays a role in interictal mood disorders.[3] A previous study reported that some patients with TLE-AE treated with antiepileptic drugs experienced recovery of emotional responses, as well as control of seizures.[2] In that study, TLE-AE was often associated with autoantibody-positive limbic encephalitis, as well as with focal cortical dysplasia or tumors of the amygdala. Although autoimmune pathology could be a possible etiology in our case, our patient did not exhibit any signs of autoimmune limbic encephalitis, such as hyponatremia, recurrent facio-brachial dystonic seizures, or autonomic dysfunction. Moreover, AE evanesces with anti-epileptic drug treatment rather than immunosuppressive therapy. Although the possibility of a tumor in the amygdala cannot be excluded, it is very low. However, it should be noted that some types of highly epileptogenic brain tumors occur frequently in the mesial temporal lobes, including the amygdala, and tend to lack radiological features of tumors that investigators attempt to screen for; as such, it cannot completely rule out the possibility of several neoplastic causes of AE.

The cause of AE is a matter of debate. Some reports have suggested that TLE-AE is a distinctive nosological and less homogeneous syndrome, and that it may be a subtype of TLE without hippocampal sclerosis. Focal cortical dysplasia and low-grade tumors or chronic inflammatory processes, including autoimmune mechanisms, can lead to AE.[2,9,10] Alternatively, AE may be caused by a seizure-related changes, such that chronic epileptic activity could lead to the development of AE.[11] AE can be reversed simply by suppressing seizures. Quantitative analysis, such as amygdala hippocampal volumetry, would have provided more objective numerical data. We did not perform quantitative analysis, which can be considered a limitation of the present case study.

In conclusion, emotional symptoms mimicking affective disorders can be a manifestation of seizure in patients with TLE-AE. AE is caused by diverse pathological disease entities and may be reversible when seizure control is achieved. Meticulous historytaking and diagnostic work-up arecritical to reach the correct diagnosis.

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