CASE REPORT

POSTICTAL FUGUE IN TEMPORAL LOBE EPILEPSY: A CASE REPORT

Cindy Prianto[1], Edeline Samudra[1], Tiara Julianti[1], Retno Jayantri Ketaren[1,2*]

[1] Faculty of Medicine, Pelita Harapan University, Tangerang, Banten, Indonesia
[2] Department of Neurology, Siloam Hospitals Lippo Village, Tangerang, Banten, Indonesia
***Correspondence:** retno.ketaren@uph.edu; ORCID ID: 0000-0003-0047-8752*

Abstract

Introduction: Temporal lobe epilepsy (TLE) is the most prevalent form of epilepsy. Postictal fugue, a state of altered consciousness and amnesia that can lead to unusual behaviors, is an unusual type of postictal phase in TLE. This case report describes a rare case of postictal fugue in adult-onset TLE.

Case Report: A 27-year-old man with complaints of recurrent seizures over the past six years, described as blank staring accompanied by purposeless action with each episode lasting 5-10 minutes on average. During the episodes, he can perform various activities without the ability to recall the events after regaining consciousness. There were neither aura nor specific triggers preceding the episodes. Some long-term memories are impaired. No other cognitive impairment is found. There was a long-standing history of substance use. Electroencephalogram examinations revealed abnormal spikes wave in bilateral temporal region.

Discussion: The patient's symptoms resemble dissociative fugue but is attributed to an organic etiology, which is mesial/limbic TLE. The postictal phase which manifests as postictal fugue is caused by the abnormal electrical activity in the hippocampus leading to disruption in memory formation and spatial navigation. The underlying mechanism causing epilepsy is thought to be related to the prolonged history of substance abuse.

Conclusion: The postictal phase in TLE can present in various forms and may be challenging to diagnose. Therefore, it is crucial to thoroughly investigate the causes of organic dissociative disorder, including postictal fugue in TLE.

Keywords: Postictal fugue, temporal lobe epilepsy, organic dissociative disorder

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Introduction

Temporal lobe epilepsy (TLE) is the most prevalent form of focal epilepsy. It is usually diagnosed in the first two decades of life. Approximately 60% of all epilepsy cases are focal, with the majority originating in the temporal lobe. About 48-56 percent of these occurrences occur bilaterally.[1] The postictal phase, which occurs after a seizure, is a period of impaired brain function that can last from minutes to days.[2] One of the less commonly documented phenomena during this phase is postictal fugue, a state of altered

consciousness and amnesia that can lead to unusual behaviors without the patient's awareness.

The underlying mechanism of the postictal fugue is not yet fully understood; however, it is associated with changes in cerebral blood flow, neurotransmitter function, and may be induced by the side effects of substances. The exact prevalence is difficult to determine due to its transient nature and challenge of capturing these episodes in clinical settings. This case report aims to describe a rare case of postictal fugue episodes in adult-onset TLE.

Case Presentation

A 27-year-old man presented with a six-year history of recurrent seizures, which had improved over the past year after receiving treatment from a neurologist. The seizures experienced by the patient involved a blank stare, similar to zoning out, followed by purposeless actions, such as adjusting an apron, with his eyes directed downward and occasionally with lip-smacking. The episodes typically lasted 5-10 minutes, with the longest duration lasting 30 minutes. During these episodes, he was able to ride a motorcycle for a considerable distance, squat, walk, or engage in other activities unconsciously. He reported falling off a motorcycle four times and hurting himself with a cigarette once, without feeling any pain. There were no specific triggers for the seizures, and he denied any auras beforehand, such as smelling odors, hearing sounds, or seeing flashes of light. No tonic- clonic seizures were reported, and the patient could not recall events during the episodes. He also denied any personality changes during the seizures. The seizure could be terminated by receiving a strong blow. After a seizure, he quickly regained consciousness without confusion, fatigue, or difficulty speaking.

The patient also complained of intermittent headaches around his eyes but denied experiencing any severe headaches. This was accompanied by the loss of some long-term memories, such as experiences from elementary school or the names of friends from the middle school, though his short-term memory remained intact. There was no history of head injury or severe head trauma. He denied any history of fainting, neck stiffness, brain infections, hemiparesis, facial weakness, speech difficulties, or childhood seizures. There were no traumatic experiences prior to the onset. His other cognitive functions remained intact, and he was able to carry out daily activities independently.

The patient reported a history of alcohol consumption and psychoactive substance use since he was 12 years old, specifically tramadol, heroin, and trihexyphenidyl for 6 years, along with marijuana for 7 years, methamphetamine for 8 years, and alcohol for 11 years. The patient has denied the use of any psychoactive substances since 2020 but still smokes 20 cigarettes per day. Family history of neurological disease was unremarkable, and the family denied any relatives with similar complaints.

On electroencephalogram (EEG) examination, one spike-and-wave discharge was observed in the bilateral anterotemporal region, along with one interictal spike in the bilateral temporal region. Based on the ILAE criteria of epilepsy and EEG findings, the patient has

been diagnosed with epilepsy for the past 4 years, specifically temporal lobe epilepsy. The patient has been prescribed several antiepileptic drugs, including sodium divalproex 500 mg twice daily, phenytoin 100 mg twice daily, and carbamazepine 200 mg twice daily.

Discussion

Postictal fugue in temporal lobe epilepsy refers to a fugue or confusional state that occurs after the ictal phase, characterized by altered consciousness with varying degrees of motor activity and amnesia during the event.[3] We present a rare case of postictal fugue in temporal lobe epilepsy (TLE) with a history of substance abuse. The patient fulfilled the criteria for epilepsy shown in Table 1. The patient reported experiencing seizures four times a month. EEG findings indicate that the patient experienced focal epilepsy, specifically mesial temporal lobe epilepsy as the seizures were accompanied with impaired consciousness. The motoric action following the non-motoric seizure was thought to be a manifestation of postictal epilepsy, specifically a postictal fugue.

Table 1. Criteria of Epilepsy based on International League Against Epilepsy (ILAE)[4]

Criteria
1. At least two unprovoked (or reflex) seizures occurring >24 h apart
2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years
3. Diagnosis of an epilepsy syndrome

The disorder experienced by the patient resembles dissociative fugue (Table 2 and Table 3), but it is attributable to an organic etiology. The distinguishing characteristics in postictal fugue typically include a history of epilepsy, the absence of stress-inducing events, and less purposeful and more fragmented activities and travel patterns observed in epilepsy patients. Therefore, the patient is thought to have organic dissociative disorder, a condition that meets the criteria for one of the disorders within Dissociative Disorders (F44.-) and also fulfills the general criteria for an organic cause.

Table 2. Criteria of Dissociative Amnesia (F44.0) based on DSM-V-TR[5]

Criteria
a. An inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is inconsistent with ordinary forgetting.
b. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
c. The disturbance is not attributable to the physiological effects of a substance (e.g., alcohol or other drug of abuse, a medication) or a neurological or other medical condition (e.g., partial complex seizures, transient global amnesia, sequelae of a closed head injury/traumatic brain injury, other neurological condition).
d. The disturbance is not better explained by dissociative identity disorder, posttraumatic stress disorder, acute stress disorder, somatic symptom disorder, or major or mild neurocognitive disorder.

Table 3. Criteria of Dissociative Fugue (F44.1) based on ICD-10[6]

Criteria
a. The features of dissociative amnesia (F44.0);
b. Purposeful travel beyond the usual everyday range (the differentiation between travel and wandering must be made by those with local knowledge); and
c. Maintenance of basic self-care (eating, washing, etc.) and simple social interaction with strangers (such as buying tickets or petrol, asking directions, ordering meals)

Based on the location of neuronal activity, epilepsy can be categorized into focal and generalized types. Focal epilepsy is related to specific regions of the brain, while generalized epilepsy involves the entire cerebral hemisphere simultaneously. Focal (or partial) epilepsy can be further divided into simple (aware), complex (with impaired awareness), and secondarily generalized types. Simple partial seizures are characterized by jerking movements in one extremity or one part of the body without loss of consciousness. Complex partial seizures involve impaired consciousness, with or without motor activity. If the seizure affects areas of the brain responsible for motor control, it may present with motor manifestations. Conversely, disruptions in other brain regions, such as the temporal lobe, are more likely to manifest non-motor symptoms. EEG findings in simple partial seizures show focal spikes in the contralateral hemisphere, while EEG findings in complex partial seizures reveal focal spikes in the frontal or temporal lobes. Secondary generalized seizures are marked by focal epilepsy that can spread from one focus to other areas of the brain, also known as focal-to-generalized, where the initial onset involves abnormalities in a focal brain structure.[7]

Temporal lobe epilepsy (TLE) is the most common form of focal epilepsy. In TLE, mossy fiber sprouting occurs, where axons from the dentate gyrus not only project to the cornu ammonis (CA3) in the hippocampus but also form new and abnormal synaptic connections in the molecular layer of the dentate gyrus. This sprouting creates excitatory feedback loops, leading to the formation of epileptogenic foci. According to the ILAE classification, TLE can be divided into mesial/limbic TLE (mTLE) and neocortical/lateral TLE (nTLE) shown in Table 4.[8,9]

In temporal lobe epilepsy (TLE), non-motor areas of the brain are often involved, including the thalamus and limbic system. The thalamus plays a crucial role in sensory information processing before it reaches the cortex, which results in impaired awareness of the environment.[10] Limbic system is a group of brain structures that located lateral to the thalamus, between cerebral cortex and hypothalamus, above the brainstem. Connected with other parts of the brain, the limbic system is involved in emotional, motivation, behavior processes, and also important to memory.[11] Memory, an essential cognitive process, can be divided into short-term (working memory) and long-term memory, which includes explicit (declarative) and implicit (procedural) memory.[12,13]

Table 4. Comparison of mTLE and nTLE[8,9]

	mTLE	nTLE
Region	Hippocampus, entorhinal cortex, amygdala, parahippocampal gyrus, and dentate gyrus	Temporal neocortex, specifically the superior, medial, and inferior temporal regions, as well as the temporo-occipital and temporo-parietal junctions and associative areas for auditory, visual, and language functions
Onset	10-year-old, particularly in individuals with a history of febrile seizures	23-year-old
Aura	Visceral sensations (such as epigastric discomfort) and déjà vu	Auditory or visual hallucinations, somatosensory symptoms
Ictal	Loss of consciousness, all activity ceases, blank stare, pupil dilation, and automatisms like chewing, smacking, or swallowing. Automatisms in the ipsilateral hand (e,g,. touching and picking) and dystonic posturing of the contralateral arm may also be observed	Motor manifestations such as jerking movements or sensory disturbances
Postictal	Present in 85% of cases, characterized by confusion, language disturbances, or psychiatric symptoms	Motor weakness (Todd's paresis) or sensory deficits may also occur. The postictal phase tends to be shorter than in mTLE
EEG	During the ictal phase, there is rhythmic focal activity between 5-9 Hz. In the interictal phase, unilateral spike-wave activity is observed in the anterior, mesial, and posterior temporal regions, which may become bilateral if the limbic system is involved	During the ictal phase, there is more prominent focal activity compared to mTLE. In the interictal phase, sharp waves, spikes, or spike-wave activity may be observed in the lateral temporal lobe

Explicit memory, which is stored in the hippocampus and medial temporal lobe, involves conscious recall of information such as events and facts. Implicit memory, stored in areas like the cerebellum and amygdala, includes skills and conditioned responses.[12,13] The Papez circuit, which begins and ends in the hippocampus and connects with the hypothalamus, thalamus, and other regions, is key in processing both memory and emotion. Disruptions in this system, particularly in the dominant hemisphere, can impair memory, language, and orientation, while disruptions in the non-dominant hemisphere may affect visual memory, such

as facial and spatial recognition.[14,15]

The postictal phase is a temporary state that occurs after a seizure has ended, lasting from several minutes to days. Postictal fugue in epilepsy can be observed in absence seizures, non-convulsive complex partial seizures, or generalized seizures. During this phase, patients may experience drowsiness, confusion, impaired consciousness, focal neurological deficits, cognitive disturbances, or psychiatric issues. The underlying mechanisms of the postictal state are not yet fully understood, but changes in brain blood flow, neurotransmitter function, and medication side effects are believed to play a role. In temporal lobe epilepsy (TLE), abnormal electrical activity in the hippocampus and surrounding structures can disrupt memory formation and spatial navigation. Cognitive function can be compromised due to reduced blood flow to the brain during the postictal phase decreases oxygen and glucose supply, which can worsen cognitive function in postictal fugue. Additionally, neurotransmitter imbalances persist during the postictal phase, with GABA reducing normal cognitive function. Lastly, consciousness is also impaired as the thalamus and the brain's ability to process sensory information are disrupted. As a result, individuals in postictal TLE exhibit purposeful motor behavior that occurs automatically, yet unaware of their surroundings, making them appear outwardly normal while being unresponsive to external stimuli.[3,16]

As happened in this patient, no motor areas were involved, rather, the thalamus and limbic system were affected. Consequently, the patient was able to move and maintain consciousness but was unaware and unresponsive to external stimuli because the thalamus could not relay sensory information to the cortex. Disruption in the limbic system, particularly the dentate gyrus and entorhinal cortex of the hippocampus, impaired the patient's ability to remember the events during the seizure, the postictal phase, as well as long-term memory.

The underlying mechanism behind the incidence of epilepsy in this patient is thought to be related to the prolonged history of substance abuse. Particularly the use of alcohol which has been associated with seizures upon withdrawal. The chronic use of alcohol can disrupt the neurotransmitter systems, especially the gamma-aminobutyric acid (GABA) and glutamate as the major inhibitory and excitatory neurotransmitters, respectively. Evidence has suggested that alcohol potentiates the effects of GABA, which accounts for the sedation effect upon initial ingestion. Over time, chronic and excessive consumption of alcohol reduces the number of GABA receptors. Another effect is the increase in glutamate receptors in the hippocampus, an area in the brain that is responsible for memory and involved in epileptic seizures. During alcohol withdrawal, alteration in glutamate and GABA receptors occur. Glutamate receptors that have adapted to the long-term presence of alcohol become upregulated. This effect combined with the deficiency of GABA receptors lead to the overexcitation throughout the brain and subsequently causing seizures within 1 - 2 days upon stopping ingestion of alcohol.[17]

Other drugs commonly associated with seizures are cocaine, amphetamine and other stimulants, cannabinoids and psychedelic agents through their ability to produce excitation of the central nervous system.[18] Among the drugs frequently associated with seizures, the patient had a history of abusing methamphetamine and cannabinoids. Amphetamines-induced-seizures occur with use at high doses and are mediated by the stimulation of N-methyl-D-aspartate (NMDA) receptors and inhibition of GABA receptors. Cannabinoids were thought to reduce the GABA turnover at low doses but increase it at high doses.[18] Other study revealed that cannabis and heroin, a type of opioid, were associated with a lower seizure incidence compared to other drugs.[19] Tramadol, another opioid used by the patient, has been proposed to lower the seizure threshold by inhibition of serotonin and norepinephrine reuptake pathways while also exerting an inhibitory effect on GABA. Consequently, the patient's long-standing history of substance abuse may increase patient's risk of seizures.

Based on the diagnosis of postictal fugue in temporal lobe epilepsy, the patient was prescribed phenytoin as the first-line therapy for focal seizures. The patient has been on this treatment for four years, but seizures have persisted, so additional therapy was gradually introduced with two other anti-epileptic drugs (AEDs), carbamazepine and sodium divalproex.[20,21] These three medications function as anticonvulsants by blocking voltage-sensitive sodium and calcium channels, and by inhibiting GABA transaminase, thereby increasing GABA concentrations, which reduce neuronal excitability.[22]

Conclusion

This case report highlights a rare case of postictal fugue in adult patient with TLE. The characterized symptoms in this patient are purposeless actions, amnesia, and impaired consciousness following seizures. Despite optimized medical therapy, the patient's episodes of postictal fugue still occur. Although postictal fugue shares similarities with dissociative fugue, the patient is thought to have organic dissociative disorder due to history of TLE and the absence of stress-induced triggers. Recognizing this phenomenon is important for accurate diagnosis and effective treatment of epilepsy- related presentations. This case report also emphasizes the need for further research into the mechanisms underlying postictal fugue in TLE and the impact on cognitive and behavioral function.

Conflict of Interest

The authors declared no conflict of interest.

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