

De novo Postoperative Postictal Psychosis Improved with Vagal Nerve Stimulation: A Case Report

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ABSTRACT

Post-ictal psychosis can manifest in patients with intractable temporal lobe epilepsy following temporal lobectomy, presenting with psychiatric symptoms such as hallucinations, depression, and aggressive behavior. This case study details a 43-year-old male with refractory focal epilepsy who developed de novo post-ictal psychosis after undergoing left anterior temporal lobectomy and hippocampal ablation. Initially, the patient achieved seizure control, but later experienced recurrent focal seizures and subsequent post-ictal psychosis, characterized by aggression, paranoia, and hallucinations. Despite multiple hospitalizations and trials of antipsychotic medications, no improvement was observed. Following the placement of a vagal nerve stimulator, the patient's psychotic episodes ceased, allowing for the discontinuation of antipsychotic medications. This case highlights the critical need for psychiatric monitoring in epilepsy patients post temporal lobectomy and underscores the potential of vagal nerve stimulator in managing peri-ictal psychotic episodes. Further research is essential to elucidate the mechanisms by which vagal nerve stimulator exerts its antipsychotic effects and to establish guidelines for its use in this patient population.

Introduction

Post-ictal psychosis (PIP) is a psychiatric disorder occurring in 6-10% of patients with intractable temporal lobe epilepsy [1]. It is characterized by psychiatric symptoms such as hallucinations, depression, delusions, aggressive behavior, and suicidal ideation. According to criteria defined by Logsdail and Toone, these symptoms typically occur within one week after a cluster of seizures, manifest after a lucid interval (24-72 hours), and can last up to two months [2]. De novo PIP can occur for the first time after temporal lobe resection and has recently been described as de novo postoperative post-ictal psychosis (PPP) [1]. Here, we report a case of de novo postoperative post-ictal psychosis which improved after vagal nerve stimulator (VNS) treatment.

Case Report

The patient is a 43-year-old right-handed male with a history of refractory focal epilepsy. He experienced the onset of focal

seizures at age 30, described as an olfactory aura followed by oro-alimentary automatisms and loss of awareness, sometimes progressing to bilateral tonic-clonic seizures. He had a history of febrile seizures as a child and prior head injury but no other epilepsy risk factors.

His evaluation with video electroencephalogram (vEEG) captured multiple typical seizures originating over the left temporal region. Magnetic resonance imaging (MRI) of the brain was unremarkable. He was trialed on multiple anti-seizure medications with continued focal seizures and ultimately underwent a phase II evaluation with subdural grid for recording and language mapping. The patient had a left anterior temporal lobectomy which was slightly smaller than usual due to his language area locating anterior to expected in 2015. He continued to have focal seizures although not as frequent after. He subsequently had left hippocampal ablation in 2017.

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After the lobectomy and hippocampal ablation, the patient remained seizure-free for approximately over 5 years. He then began experiencing recurrence of his typical focal seizures, occurring approximately once a month. Unlike his prior seizures, 48-72 hours after clusters of the seizures, he began displaying new post-ictal psychotic symptoms. During this post-ictal phase, he became aggressive, displayed interpersonal violence, suicidal ideations, paranoia, visual hallucinations, and auditory hallucinations. The post-ictal psychotic symptoms lasted on average 2-3 days before he returned to his neurological baseline, leading to multiple hospitalizations. He was trialed on anti-psychotic medications without improvement.

Due to the significant disruption in his life caused by these refractory focal seizures and recurrent post-ictal psychosis, he underwent another vEEG for consideration of further non-pharmacologic options. This epilepsy monitoring unit (EMU) admission captured left hemispheric electroclinical seizures, followed by post-ictal psychosis for several hours after the seizures. During the episodes of psychosis, there were no EEG changes from his normal background patterns. In 2024, he underwent VNS placement as another attempt to better control his breakthrough seizures. Despite VNS placement he continued to have breakthrough seizures. However, the episodes of post-ictal psychosis were halted, and anti-psychotic medications were able to be weaned and eventually discontinued. This effect was noted with VNS settings at a normal output current of 0.25–0.5 mA, auto stim output current of 0.625 mA, and magnet output current of 0.75 mA.

Discussion

Our patient developed PIP following resection of the left anterior temporal lobe for medically intractable left temporal lobe epilepsy (TLE). This case is particularly notable as the patient had no prior history of psychiatric comorbidities, use of antipsychotic medications, or any medications known to induce psychosis. Additionally, preoperative seizures captured in the epilepsy monitoring unit (EMU) and observed at home did not demonstrate any postictal psychiatric symptoms.

The development of PIP in this context raises important considerations regarding the neuropsychiatric outcomes following anterior temporal lobectomy (ATL). Psychiatric disorders are among the most common postoperative complications after ATL for intractable TLE [3]. This case underscores the need for thorough preoperative psychiatric evaluation and postoperative monitoring for psychiatric symptoms, even in patients without a prior psychiatric history.

There is emerging evidence in the literature suggesting an antipsychotic effect following VNS implantation. Lee et al. reported the first case of forced normalization after deactivating VNS in a patient with Lennox-Gastaut syndrome [4]. Alemany et al. have also suggested that VNS may modulate peri-ictal psychotic episodes [5]. These findings highlight the potential of VNS as a therapeutic option for managing psychiatric symptoms in epilepsy patients.

VNS has been utilized for decades as a treatment for seizures and, more recently, for depression. The mechanism of action

involves stimulating the vagus nerve with electrical impulses, which are then transmitted to the brain. Although the exact mechanism by which VNS alleviates depression is not fully understood, it appears to modulate brain circuits involved in mood regulation [6]. Neuroimaging studies suggest that VNS therapy acts by innervating the nucleus tractus solitarius, which projects to limbic and cortical structures involved in mood regulation. Brainstem regions containing serotonergic (raphe nucleus) and noradrenergic (locus ceruleus) perikarya project to the forebrain, potentially contributing to the antidepressant effect [7].

The U.S. Food and Drug Administration (FDA) approved VNS as an option for people with treatment-resistant depression in 2005 [8]. This approval was based on evidence demonstrating its efficacy in modulating mood and reducing depressive symptoms [8]. Previous reports, such as those by Lee et al. and Alemany et al., provide further support for the psychiatric benefits of VNS in epilepsy patients [5,6].

Conclusion

Our case highlights the occurrence of PIP following ATL in a patient without prior psychiatric history, emphasizing the need for vigilant psychiatric monitoring in such patients. The potential role of VNS in managing peri-ictal psychotic episodes and its established use in treating depression suggest that it may be a valuable adjunctive therapy in epilepsy patients with psychiatric comorbidities. Further research is needed to elucidate the mechanisms by which VNS exerts its antipsychotic effects and to establish guidelines for its use in this patient population.

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