

Postictal Psychosis Treated Successfully With Olanzapine

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Postictal psychosis is a significant psychiatric complication of chronic epilepsy estimated to occur in 2%–7.8% of epilepsy patients.¹ Risk factors include bilateral seizure foci, bilateral limbic lesions, increase in seizure frequency, or a cluster of seizures preceding psychiatric symptoms.^{1–3} Seizure control can prevent postictal psychosis,^{2,4} which is often recurrent,^{2,4,5} but when present, immediate recognition of this disorder is critical. Treatment must be initiated promptly in order to avoid significant morbidity and possibility of mortality.⁴ Despite the estimated frequency of postictal psychosis, it is often not recognized, and literature regarding the diagnosis and treatment of this condition is scarce. We present a case of recurrent postictal psychosis successfully treated with olanzapine.

Case report. Ms A, a 36-year-old woman with a history of temporal lobe epilepsy with complex partial seizures and secondary generalized tonic clonic seizures since age 18, was admitted to the neurology floor for seizures. Two days prior to her hospital presentation, Ms A was visiting her parents and forgot to take her antiepileptic medications. Consequently, she suffered 6 seizures: 2 generalized tonic-clonic seizures and 4 complex partial seizures. Her father noted that, after the series of seizures, Ms A was uncharacteristically aggressive and argumentative and was using profane language toward her family. However, she did not have any psychotic symptoms initially. After what appeared to be a lucid interval of 24 hours, she began complaining that something in her head was about to explode and thought that different people were in danger of being hurt. She subsequently presented to the emergency department of a large teaching hospital. She was admitted to the neurology service and started on her antiseizure medications, levetiracetam 1,000 mg twice daily and zonisamide 400 mg twice daily. Lacosamide 100 mg twice daily was also added. Continuous electroencephalogram (EEG) monitoring was initiated, and a psychosomatic medicine consultation was ordered due to concerns about ongoing psychosis.

Upon interview, Ms A described various delusions, including paranoid, somatic, religious, and thought control. Her other symptoms included confusion, agitation, and auditory and olfactory hallucinations. Specifically, Ms A experienced the cenesthetic hallucination of the sensation that her brain was “burning and frying,” with the accompanying nihilistic somatic delusion that her brain was “dead” because of the EEG leads placed on her head. She also had the paranoid delusion that these leads were extracting information from her brain. Additionally, she assumed that the hospital staff must have placed these same leads on the heads of her family members in order to extract more information

from them as well. She had a persistent fear that the hospital physicians might harm her family and were “playing mind games” with her. Ms A also developed a Fregoli delusion in that she believed that the members of her family had taken on the form of the hospital nurses who were coming to her aid when she was agitated.

Furthermore, Ms A had delusions of reference. She noted that God and her deceased sister were communicating to her through the television. Ms A believed her sister was instructing her to look deep into the eyes of her youngest daughter in order to stop the seizures. Ms A also noted a specific and persistent somatic delusion of pseudocyesis. Despite 2 negative pregnancy tests, Ms A explained that she was sure she was pregnant and could even feel the baby moving inside of her. Additionally, when she saw a commercial for a pregnancy test the day prior, she noted that it was a message and a sign from God telling her that she was indeed pregnant. Throughout the interview Ms A had persistent auditory hallucinations of intermittently hearing a dump truck.

Anticonvulsant toxicity was ruled out (antiepileptic drug levels were within normal range, but lower than her usual therapeutic levels). Results from a urine drug screen, thyroid-stimulating hormone, routine blood chemistries, metabolic studies, and a head computerized tomography scan were within normal limits. Continuous EEG monitoring showed no epileptiform discharges or seizures. She was started on olanzapine 5 mg, which was increased to 10 mg on the tenth day of admission for continued psychosis. Within less than a week on this dose, her psychotic symptoms began to resolve.

Importantly, 1 year prior to this admission, Ms A had a similar episode of postictal psychosis, which was treated with olanzapine 5 mg and resolved after 2 weeks. She discontinued the olanzapine upon discharge and had no seizures or psychotic symptoms until her most recent admission. There were no other known episodes of treated or untreated postictal psychosis. At baseline, Ms A has no psychotic features, is functional, and is the mother of 3 children.

Many accounts of postictal psychosis cases describe similarities to positive symptoms seen in schizophrenia such as delusions, hallucinations, and disorganized speech. However, these descriptions are also notable for their absence of negative symptoms and better premorbid function compared to what is typically observed with schizophrenic patients.⁴

Based on findings from a series of similar cases, diagnostic criteria for postictal psychosis were proposed over 20 years ago.⁵ Ms A's symptoms meet these diagnostic criteria exactly.

Previous reports of postictal psychosis have alluded to the many treatment considerations for this condition. Many of the commonly used antipsychotic agents have proconvulsant properties; ie, they can lower the seizure threshold, and their use can increase the risk of having seizures and status epilepticus in patients with epilepsy.^{4,6,7} Olanzapine with and without concomitant medications or epilepsy has been associated with seizures and status epilepticus (including a case report⁸ of fatal status epilepticus) in the literature.^{9,10} Continuous EEG monitoring may therefore be necessary during initiation and dosage changes of antipsychotics in these patients in order to promptly detect and treat any such events.

After weighing risks and benefits of initiating antipsychotic treatment, olanzapine was started for our patient with continuous EEG monitoring, and she experienced no subsequent seizure activity. Furthermore, a previous case report¹¹ suggested that patients should remain on the neuroleptic medication for an indefinite period of time in order to prevent future episodes. Our patient demonstrated that she was able to discontinue her use of olanzapine after her initial episode 1 year ago and remained free of both seizures and psychotic symptoms.

Olanzapine was used in our patient because of a history of prior response to this drug in an episode of postictal psychosis and also because of its sedating properties; however, any antipsychotic could have been tried. It is unclear at this time because of a lack of comparative studies whether one antipsychotic is more beneficial or effective than another.

Our patient was successfully treated with olanzapine. On this regimen, she experienced no further seizure activity, and the sedative properties ameliorated her agitation, eliminating the need to consider other treatment options, such as benzodiazepines or antiepileptic agents, with mood stabilizing properties. Antipsychotics have been shown to be helpful in reducing symptom severity, as evidenced in our patient whose symptoms decreased in severity after a few doses of olanzapine 5 mg and were further reduced to minimal after olanzapine was increased to 10 mg. Although postictal psychosis is often self-limiting and does not necessarily always require hospitalization, neuroleptic agents, or both,¹² treatment may be necessary to reduce morbidity, distress, and the

danger to self and others. Since current studies and research on postictal psychosis are insufficient, it is unclear at this time whether antipsychotics are indicated and will help reduce the duration of symptoms or not.

In conclusion, additional randomized controlled trials are needed to compare treatment versus no treatment in postictal psychosis and also to determine effective treatment regimens and adequate length of treatment if treatment is indicated.

REFERENCES

1. Knowlton R. Can magnetoencephalography aid epilepsy surgery? *Epilepsy Curr.* 2008;8(1):1–5.
2. Devinsky O, Abramson H, Alper K, et al. Postictal psychosis: a case control series of 20 patients and 150 controls. *Epilepsy Res.* 1995; 20(3):247–253.
3. Toone BK. The psychoses of epilepsy. *J Neurol Neurosurg Psychiatry.* 2000;69(1):1–3.
4. Kanner AM. Psychosis of epilepsy: a neurologist's perspective. *Epilepsy Behav.* 2000;1(4):219–227.
5. Logsdail SJ, Toone BK. Post-ictal psychoses: a clinical and phenomenological description. *Br J Psychiatry.* 1988;152(2):246–252.
6. Devinsky O. Psychiatric comorbidity in patients with epilepsy: implications for diagnosis and treatment. *Epilepsy Behav.* 2003; 4(suppl 4):S2–S10.
7. Alper K, Schwartz KA, Kolts RL, et al. Seizure incidence in psychopharmacological clinical trials: an analysis of Food and Drug Administration (FDA) summary basis of approval reports. *Biol Psychiatry.* 2007;62(4):345–354.
8. Wyderski RJ, Starrett WG, Abou-Saif A. Fatal status epilepticus associated with olanzapine therapy. *Ann Pharmacother.* 1999;33(7–8):787–789.
9. Camacho A, Garcia-Navarro M, Martinez B, et al. Olanzapine-induced myoclonic status. *Clin Neuropharmacol.* 2005;28(3):145–147.
10. Spyridi S, Sokolaki S, Nimatoudis J, et al. Status epilepticus in a patient treated with olanzapine and mirtazapine. *Int J Clin Pharmacol Ther.* 2009; 47(2):120–123.
11. Marino J, Augsten A, Henry J. Postictal psychosis successfully treated with quetiapine: a case report. *Ann Pharmacother.* 2009;43(9):1544.
12. Lancman ME, Craven WJ, Asconape JJ, et al. Clinical management of recurrent post-ictal psychosis. *J Epilepsy.* 1994;7(1):47–51.

Drug names: lacosamide (Vimpat), levetiracetam (Keppra and others), olanzapine (Zyprexa and others), zonisamide (Zonegran and others).

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