The patient is a 47-year-old woman with no history of substance abuse and an unremarkable medical history who was diagnosed by her primary care physician 18 months prior to presentation at our hospital with major depression. She had initially been prescribed duloxetine and then fluoxetine. She then traveled to Mexico in an attempt to recover from her depression, but while there she decompensated and was admitted to a university psychiatric facility. Doctors there diagnosed her with conversion disorder and prescribed mirtazapine, paroxetine, and clonazepam. She returned home to the United States upon discharge but relapsed into depression, refused to eat, and became bedridden. Being concerned, her family brought her to an emergency department multiple times, but at these visits all laboratory tests, computerized tomography scans, and magnetic resonance imaging scans were normal, so she was never admitted. Finally, 2 months after her return to the US she was medically admitted for altered mental status in the setting of a urinary tract infection.

During her admission, psychiatry was consulted. Although she had been compliant with her psychiatric medications, an evaluation revealed the following: a 50-pound weight loss over the past 6 months, inability to care for basic needs, loss of energy with severe psychomotor retardation, unsteady gait with recent falls, dysphagia, alogia, grimacing, negativism, and echolalia. In light of these symptoms, a full neurologic workup was completed to rule out any organic causes such as a brain tumor or seizure activity; however, no organic causes were identified on electroencephalogram, computed tomography, magnetic resonance imaging, or lumbar puncture.

With an initial diagnosis of major depressive disorder with catatonia, psychiatry recommended electroconvulsive therapy (ECT) and prescribed venlafaxine and bupropion. She underwent eight ECT sessions with bilateral node placement and sustained seizures of no less than 28 seconds. By the completion of her sixth treatment, the patient’s mood was brighter, her psychomotor retardation was less, she was more spontaneous with language, and she had a more reactive affect. Prior to the eighth ECT treatment she was noted to be slightly restless and agitated. Further ECT treatments were postponed as she became more agitated, and her mental status examination was positive for both auditory and
visual hallucinations. There was clouding of consciousness, she was disoriented, and had an impaired sensorium. It was thought that she was experiencing ECT postictal delirium (PID) so treatment with a course of low-dose haloperidol was prescribed, but over the course of 3 days her psychotic symptoms did not improve. Olanzapine and injectable aripiprazole were prescribed next. Over the course of 2 weeks her psychotic symptoms slowly resolved, and her depressive and catatonia symptoms did not reoccur.

**DIAGNOSIS**

**Schizoaffective Disorder, Depressed Type with Catatonia**

After some discussion and debate it was decided to modify the patient’s differential diagnosis to schizoaffective disorder, depressed type with catatonia. At the time of this writing the patient had returned home, remained psychiatrically stable, continued taking olanzapine and venlafaxine, and continues to care for her basic needs independently.

**DISCUSSION**

ECT is a gold standard for treating catatonia in depression and other psychiatric conditions. Although reserved for more severe cases that are usually refractory to pharmacologic treatment, ECT is efficacious and results in improvement of symptoms. The main indications for ECT include psychotic depression, depression with risk of suicide, delirious mania, catatonic stupor, postpartum psychosis, cycloid psychosis, letha-tal catatonia, and neuroleptic malignant syndrome. Schizophrenia spectrum and depressive disorders have a catatonia specifier; however, limited research and case reports exist to support the effectiveness of ECT for catatonia in schizophrenia spectrum disorders.

The presentation of catatonic features can occur in both depressive and schizophrenic spectrum disorders. The presentation of catatonia is rare and can be characterized by bizarre behaviors such as echolalia, echopraxia, mutism, negativism, waxing flexibility, and posturing. The *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5) defines catatonia as a specifier for major depressive disorder, schizophrenic, and schizoaffective disorder. ECT was a valid treatment in this case as she presented in a catatonic state with progressively worsening symptoms and hallmark symptoms of catatonia. The predicted remission rate in case of major indication of psychotic depression is 92% to 95%, but only 55% to 84% in non-psychotic depression. As expected, her symptoms of depression and catatonia showed improvement with a course of ECT treatments. However, after the eighth treatment she developed symptoms of psychosis. Depression with psychosis is a major indication for ECT; however, there are no guidelines for psychosis that develops after ECT treatments. ECT has been used to treat psychosis in the form of psychotic depression, delirious mania, postpartum psychosis, and drug-induced psychosis. An extensive literature review done by our group found no reports regarding psychotic symptoms manifesting after ECT that were not already present.

In the case of our patient, she developed confusion, auditory and visual hallucinations, and psychomotor agitation after eight ECT sessions, and these symptoms seemed consistent with a diagnosis of PID. It should be noted that we did consider serotonin syndrome due to concomitant use of venlafaxine and bupropion and ECT delirium as part of the differential diagnosis. PID is defined by Kikuchi et al. as an “acute confusional state, [which] can often occur during the immediate post-ictal phase in patients receiving ECT.” Because ECT elicits seizures, patients may develop PID in about 10% of cases, although it has been reported in up to 36%. Patients present with altered consciousness, disorientation, behavioral disturbances, and motor agitation. The study published by Kikuchi et al. indicated that catatonic features may be an indicator for development of PID, because incidence of PID in these patients was as high as 88%. Catatonic features in our patient were a risk factor for developing PID. However, in this patient’s case, the psychotic symptoms did not resolve in the time frame usually observed in PID. She experienced prolonged psychosis that lasted more than 2 weeks and had to be controlled with antipsychotic medications, which is also uncommon.

A literature review revealed few similar cases of prolonged delir-
um. One case described a 41-year-old woman with depression who developed a delayed-onset delirium after her third ECT session. In this case, her confusional state resolved spontaneously within 48 hours. No antipsychotics were necessary. The other case described a 50-year-old man with depression who developed delirium 2 days after his sixth ECT session. His symptoms lasted longer, showing improvement after 1 week with administration of haloperidol, lorazepam, and donepezil. The cause of his symptoms was not clear, as he did not have any risk factors or cortical abnormalities; bilateral electrode placement and older age were thought to have contributed to his condition.

The chemical etiology and biological mechanism for ECT causing PID are unknown. Multiple theories describing variations on neurotransmitters causing changes in pituitary hormones and increased neural plasticity or synaptogenesis have been developed to explain the mechanism by which ECT works. The monoamine neurotransmitter theory postulates that ECT alters dopamine, serotonin, and adrenergic transmitters by augmenting receptor sensitivity or neurotransmitter availability. Animal studies demonstrate increased dopamine after ECT, but it has never been observed to cause long-lasting psychotic symptoms.

Patients who develop PID usually improve within minutes to days, and there are no reports of ECT delirium lasting as long as in the case of our patient. PID is mostly observed in patients with seizure disorders and it usually correlates with the severity and duration of the seizure episode. If cortical abnormalities or mental retardation are present, even longer periods of PID have been observed, lasting 4 to 10 days. Our patient had no organic abnormalities during neurologic testing and imaging. Moreover, cases of delirium that develop after ECT usually occur during the initial treatment, resolve spontaneously in less than 1 hour, and are more frequent after switching from unilateral to bilateral electrode placement. If medications are needed, these patients usually respond to benzodiazepines such as diazepam, barbiturates, or droperidol.

CONCLUSION

This case remains unique in its presentation of delayed onset and severe psychotic symptoms requiring prolonged treatment with high doses of antipsychotics. It is possible that the patient was experiencing prolonged PID caused by the direct effect of bilateral electrode placement during ECT, consequences of the anesthesia received during treatment, or a reaction to her antidepressants. The very presence of catatonic symptoms may also have placed the patient at higher risk for the development of PID. However, it is more likely that her initial diagnosis was only partially correct and that instead of being a depressive disorder her true diagnosis was in the schizophrenic spectrum. A diagnosis of schizoaffective disorder depressed type with catatonia explains fully the patient’s presentation and symptoms. This case highlights the need for more research regarding the use of ECT in patients with a diagnosis in the schizophrenia spectrum who are depressed or catatonic.

REFERENCES