Catatonia After Neuroleptic Malignant Syndrome Successfully Treated With Electroconvulsive Therapy

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We present a case of a patient who developed catatonia after resolution of neuroleptic malignant syndrome (NMS), which was then successfully treated with electroconvulsive therapy (ECT). After the resolution of NMS with treatment with dantrolene, this patient developed malignant catatonia. The patient subsequently returned to his baseline level of functioning after ECT.

Case Report

The patient was a 27-year-old man with past medical history significant for dysembryplastic neuroepithelial tumor, status postresection in 2000 and 2006; seizure disorder; and hypothyroidism. His past psychiatric history was significant for autism spectrum disorder, mental retardation, and unspecified mood disorder. He was a resident of a group home. Most recently, he was admitted to an inpatient psychiatric unit for management of aggressive behaviors. During this admission, haloperidol was added to his medication regimen. His other medications at that time were buspirone, clonidine, valproic acid extended release, fluvoxamine, oxcarbazepine, quetiapine, and trazodone. Within a few days, the patient became drowsy and confused and was found to have a high fever. His workup revealed leukocytosis and elevated creatine phosphokinase (CPK). His brain magnetic resonance image (MRI) and electroencephalogram (EEG) were negative. A diagnosis of aspiration pneumonia was made. He was started on intravenous antibiotics. His CPK level kept increasing from 437 to 1,685 µg/L within the next few days. Due to his recent exposure to 2 different antipsychotics, with fever, rigidity, and CPK elevation, NMS was considered and dantrolene was started. Within the next 2 days, his CPK level started to decrease, and his fever and leukocytosis resolved. His antibiotics were stopped. Soon thereafter, he started to develop mutism, immobility, staring, and negativism. His symptoms were consistent with catatonia.

Further workup by the neurology department to rule out seizures, tumor recurrence, and cerebrospinal fluid infection with EEG, MRI, and lumbar puncture were inconclusive, and he was diagnosed with catatonia. Initial treatment with clonazepam did not improve his symptoms.

He was started on ECT. After the second treatment, his symptoms of catatonia started to improve; he became more verbal, with decreased negativism and less staring. After the fifth ECT session, he returned to his baseline level and was able to walk to the bathroom without assistance and perform other activities of daily living. Quetiapine was restarted at low doses with close monitoring and was eventually titrated up to control his behaviors. He continued to be carefully monitored for NMS or catatonic symptoms.

Discussion

Catatonia is a syndrome comprised of symptoms such as motor immobility, excessive motor activity, extreme negativism, and stereotyped movements. It mainly occurs in primary mood disorders or psychotic illnesses and medical and surgical conditions such as neoplasms, encephalitis, head traumas, diabetes, and metabolic disorders.[1] However, neuroleptics may induce catatonia-like symptoms known as NMS.[1] NMS is a rare syndrome observed in only 0.2% of psychiatric patients.[2] Patients typically show symptoms such as altered mental state, muscle rigidity, tremor, tachycardia, hyperpyrexia, leukocytosis, and elevated serum CPK level.[2] Any antipsychotic drug, including the atypicals, can cause NMS.[3] NMS is an idiosyncratic response to dopamine receptor antagonist medications.[3]

Both NMS and catatonia share similar symptoms. On a symptomatic level, both conditions share specific symptoms such as akinesia, muscle rigidity, stupor, and mutism. However, there are clear differences in behavioral symptoms between catatonia and NMS. Quetiapine has been reported to cause NMS.[2] A possible mechanism could be the cytochrome P450 A4 induction, resulting in decreased blood concentration of quetiapine.[2] Studies[2] also indicate reduced γ-aminobutyric acid receptors in the left sensorimotor cortex in catatonic patients. Other mechanisms include sudden and massive dopamine blockade resulting in NMS malignant catatonia.[2] NMS is usually a self-limited disorder, with most episodes resolving within 2 weeks. Many patients have been reported to recover within 7 to 11 days.[2]

In our case, 2 series of ECT had an effect on the patient's residual catatonic state when the pharmacologic

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Prim Care Companion CNS Disord 2019;21(4):18l02376

To cite: Che J, Muralidhara S, Egbert M, et al. Catatonia after neuroleptic malignant syndrome successfully treated with electroconvulsive therapy. *Prim Care Companion CNS Disord*. 2019;21(4):18l02376.

To share: https://doi.org/10.4088/PCC.18l02376

treatment with a benzodiazepine failed to treat the catatonic symptoms. Literature supports the notion that if a catatonic patient is treated with 6–16 mg of lorazepam per day with no improvement within 5 days, ECT is recommended as the next best option.[2] ECT has been very effective for catatonia, either as a first-line treatment or failing after pharmacologic treatment.

Conclusion

In this case report, we presented the course and onset of NMS and catatonia in our patient, which was most likely due to the use of 2 antipsychotic agents. The diagnosis was complicated by the patient's previous history of dysembryoplastic neuroepithelial tumors, seizures, autistic behavior, and development of pneumonia. After ruling out and treating other courses of possible NMS-like symptoms, the patient developed catatonia. Our patient had a complete recovery after ECT treatment.

Published online: August 15, 2019.

Potential conflicts of interest: None.

Funding/support: None.

Patient consent: Signed consent was received from the patient to publish this report, and information was de-identified to protect anonymity.

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