Partially Treated Catatonia and Incipient Neuroleptic Malignant Syndrome: A Challenging Presentation

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Neuroleptic malignant syndrome (NMS) is a life-threatening condition associated with the use of psychotropic medications. *Incipient NMS*, a term used to describe a NMS presentation not meeting all the criteria, can pose diagnostic and treatment challenges.[1] Catatonia and NMS share similar clinical features, pathology, and treatment. Previous reports[2] described the occurrence of NMS in catatonic patients treated with antipsychotics and suggested its role in progression to NMS. NMS can progress slowly over days to weeks, and early removal of the offending agent can result in an incomplete or milder presentation. Moreover, lorazepam, which is used for treatment of both catatonia and NMS, might alter the presentation. We present a case of incipient NMS in a patient who developed catatonia during treatment with lorazepam and paliperidone.

Case Report

A 25-year-old man with a history of schizoaffective disorder and catatonia, previously maintained on lithium 600 mg by mouth twice/d, clozapine 500 mg/d, and propranolol 20 mg twice/d, presented with manic symptoms secondary to noncompliance with treatment. He had no previous medical history. Lithium was resumed and titrated to 1,500 mg at bedtime. Due to history of noncompliance, clozapine was cross-tapered with paliperidone, and the dose was titrated to 12 mg/d over 7 days. On day 7, he displayed catatonic symptoms of mutism, posturing, staring, and refusal of food and drink. His symptoms mildly improved on lorazepam 6 mg/d. On day 8 on paliperidone, he appeared to be confused, and his temperature spiked to 38°C (100.4°F) briefly. However, his creatine phosphokinase (CPK) level was 194 U/L. As he appeared lethargic the next day, his CPK level was rechecked and was found to have increased to 962 U/L and then subsequently rose to 1,735 U/L on the same day. There was no rigidity or persistent fever to suggest NMS, but he showed vital instability with tachycardia and blood pressure fluctuations. He was transferred to the medical floor for further management and received bromocriptine 2.5 mg 3 times/d, lorazepam 2 mg 3 times/d, and symptomatic treatment. All psychotropics were suspended, and his symptoms improved a few days later. He was transferred back to the psychiatric unit, and his catatonia resolved gradually with lorazepam. He was discharged on lithium 1,500 mg and clozapine 200 mg.

Discussion

The *DSM-IV* used diagnostic criteria developed by Caroff and Mann, which relied on specific criteria that required the presence of fever and rigidity among other signs.[3] This resulted in confusion in the management of many cases that did not meet the high bar proposed, and, hence, the term *incipient NMS* was used to describe such cases. The *DSM-5* addressed this problem by eschewing strict parameters for diagnosis and rather describing the array of symptoms and encouraging a reliance on clinical judgment in diagnosis.[4] Catatonia and NMS share similar pathology, symptoms, and treatment modalities. The literature is replete with cases of catatonia preceding NMS, especially when antipsychotics are used.[5] Some authors[6] recommend against the use of antipsychotics altogether during catatonia, while others including the Maudsley guidelines[7] recommend using atypical antipsychotics during catatonia when NMS is ruled out.

Our patient presented with catatonia, which was treated with lorazepam. However, paliperidone was continued for the management of manic symptoms. He became lethargic and developed mild rhabdomyolysis, and his fever spiked briefly. However, he did not exhibit rigidity of persistent high fever as is usually observed in patients with NMS, possibly due to the use of lorazepam partially masking some NMS signs. As guided by the new changes in the *DSM-5*, the constellation of signs, especially newly developed lethargy and vital instability in the presence of antipsychotics, raised suspicion of NMS.

Discontinuation of paliperidone and use of lorazepam and bromocriptine resulted in improvement in our patient. This case highlights the need to treat catatonia aggressively and for clinicians to be vigilant during its management to recognize early or milder signs of NMS and discontinue antipsychotics when NMS is suspected to prevent further progression.

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