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Managing excited catatonia: A suggested approach

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Anshuman A. Srivastava, MD
PGY-3 Psychiatry Resident

Emil M. Achmad, MD, MS
Assistant Professor

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Department of Psychiatry
Mount Sinai Beth Israel
Icahn School of Medicine at Mount Sinai
New York, New York

Our algorithm can facilitate identification and treatment of this challenging syndrome

Catatonia is often difficult to identify and treat. The excited catatonia subtype can be particularly challenging to diagnose because it can present with symptoms similar to those seen in mania or psychosis. In this article, we present 3 cases of excited catatonia that illustrate how to identify it, how to treat the catatonia as well as the underlying pathology, and factors to consider during this process to mitigate the risk of adverse outcomes. We also outline a treatment algorithm we used for the 3 cases. Although we describe using this approach for patients with excited catatonia, it is generalizable to other types of catatonia.

Many causes, varying presentations

Catatonia is a psychomotor syndrome characterized by mutism, negativism, stereotypy, waxy flexibility, and other symptoms.[1] It is defined by the presence of ≥3 of the 12 symptoms listed in the ***Table***[2] (***page 29***). Causes of catatonia include metabolic abnormalities, endocrine disorders, drug intoxication, neurodevelopmental disorders, medication adverse effects, psychosis, and mood disorders.[1,3]

A subtype of this syndrome, excited catatonia, can present with restlessness, agitation, emotional lability, poor sleep, and altered mental status in addition to the more typical symptoms.[1,4] Because excited catatonia can resemble mania or psychosis, it is particularly challenging to identify the underlying disorder causing it and appropriate treatment.

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Fink et al[4] discussed how clinicians have interpreted the different presentations of excited catatonia to gain insight into the underlying diagnosis. If the patient's thought process appears disorganized, psychosis may be suspected.[4] If the patient is delusional and grandiose, they may be manic, and when altered mental status dominates the presentation, delirium may be the culprit.[4]

Regardless of the underlying cause, the first step is to treat the catatonia. Benzodiazepines and electroconvulsive therapy (ECT) are the most well validated treatments for catatonia and have been used to treat excited catatonia.[1] Excited catatonia is often misdiagnosed and subsequently mistreated. In the following 3 cases, excited catatonia was successfully identified and treated using the same approach (***Figure, page 31***).

Table

Symptoms of catatonia

Stupor	Mannerism
Catalepsy	Stereotypy
Waxy flexibility	Agitation
Mutism	Grimacing
Negativism	Echolalia
Posturing	Echopraxia

Source: Reference 2

CASE 1

Mr. A, age 27, has a history of bipolar I disorder. He was brought to the hospital by ambulance after being found to be yelling and acting belligerently, and he was admitted to the inpatient psychiatry unit for manic decompensation due to medication nonadherence. He was started on divalproex sodium 500 mg twice a day for mood stabilization, risperidone 1 mg twice a day for adjunct mood stabilization and psychosis, and lorazepam 1 mg 3 times a day for agitation. Mr. A exhibited odd behavior; he would take off his clothes in the hallway, run around the unit, and randomly yell at staff or to himself. At other times, he would stay silent, repeat the same statements, or oddly posture in the hallway for minutes at a time. These behaviors were seen primarily in the hour or 2 preceding lorazepam administration and improved after he received lorazepam.

Mr. A's treating team completed the Bush-Francis Catatonia Rating Scale (BFCRS), which yielded a positive catatonia screen of 7/14. As a result, divalproex sodium and risperidone were held, and lorazepam was increased to 2 mg twice a day.

After several days, Mr. A was no longer acting oddly and was able to speak more spontaneously; however, he began to exhibit overt signs of mania. He would speak rapidly and make grandiose claims about managing millions of dollars as the CEO of a famous company. Divalproex sodium was restarted at 500 mg twice a day and increased to 500 mg 3 times a day for mood stabilization. Mr. A continued to receive lorazepam 2 mg 3 times a day for catatonia, and risperidone was restarted at 1 mg twice a day to more effectively target his manic symptoms. Risperidone was increased to 2 mg twice a day. After this change, Mr. A's grandiosity dissipated, his speech normalized, and his thought process became organized. He was discharged on lorazepam 2 mg 3 times a day, divalproex sodium 500 mg 3 times a day, and risperidone 2 mg twice a day. Mr. A's length of stay (LOS) for this admission was 11 days.

CASE 2

Mr. B, age 49, presented with irritability and odd posturing. He has a history of schizoaffective disorder, bipolar type for which he was receiving a maintenance regimen of lithium 600 mg/d at bedtime and risperidone 2 mg/d at bedtime. He had multiple previous psychiatric admissions for catatonia. On this admission, Mr. B was irritable and difficult to redirect. He yelled at staff members and had a stiff gait. The BFCRS yielded a positive screening score of 3/14 and a severity score of 8/23. As a result, the treatment team conducted a lorazepam challenge.

After Mr. B received lorazepam 1 mg IM, his thought organization and irritability improved, which allowed him to have a coherent conversation with the interviewer. His gait stiffness also improved. His risperidone and lithium were held, and oral lorazepam 1 mg 3 times a day was started for catatonia. Lorazepam was gradually increased to 4 mg 3 times a day. Mr. B became euthymic

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Clinical Point

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Excited catatonia

Clinical Point

It was initially difficult to identify catatonia because all 3 patients' symptoms were nearly identical to those of mania

and redirectable, and had an improved gait. However, he was also tangential and hyperverbal; these symptoms were indicative of the underlying mania that precipitated his catatonia.

Divalproex sodium extended release (ER) was started and increased to 1,500 mg/d at bedtime for mood stabilization. Lithium was restarted and increased to 300 mg twice a day for adjunct mood stabilization. Risperidone was not restarted. Toward the end of his admission, Mr. B was noted to be overly sedated, so the lorazepam dosage was decreased. He was discharged on lorazepam 2 mg 3 times a day, divalproex sodium ER 1,500 mg/d at bedtime, and lithium 300 mg twice a day. At discharge, Mr. B was calm and euthymic, with a linear thought process. His LOS was 25 days.

CASE 3

Mr. C, age 62, presented to the emergency department (ED) because he had exhibited erratic behavior and had not slept for the past week. He has a history of bipolar I disorder, hypothyroidism, diabetes, and hypertension. For many years, he had been stable on divalproex sodium ER 2,500 mg/d at bedtime for mood stabilization and clozapine 100 mg/d at bedtime for adjunct mood stabilization and psychosis. In the ED, Mr. C was irritable, distractible, and tangential. On admission, he was speaking slowly with increased speech latency in response to questions, exhibiting stereotypy, repeating statements over and over, and walking very slowly.

The BFCRS yielded a positive screening score of 5/14 and a severity score of 10/23. Lorazepam 1 mg IM was administered. After 15 minutes, Mr. C's speech, gait, and distractibility improved. As a result, clozapine and divalproex sodium were held, and he was started on oral lorazepam 1 mg 3 times a day. After several days, Mr. C was speaking fluently and no longer exhibiting stereotypy or having outbursts where he would make repetitive statements. However, he was tangential and irritable at times, which were signs of his underlying mania. Divalproex sodium ER was restarted at 250 mg/d at bedtime for mood stabilization and gradually increased to 2,500 mg/d at bedtime. Clozapine was also restarted at 25 mg/d at bedtime and gradually increased to 200 mg/d at bedtime. The lorazepam was gradually tapered and discontinued over the course of 3 weeks due to oversedation.

At discharge, Mr. C was euthymic, calm, linear, and goal-directed. He was discharged on divalproex sodium ER 2,500 mg/d at bedtime and clozapine 200 mg/d at bedtime. His LOS for this admission was 22 days.

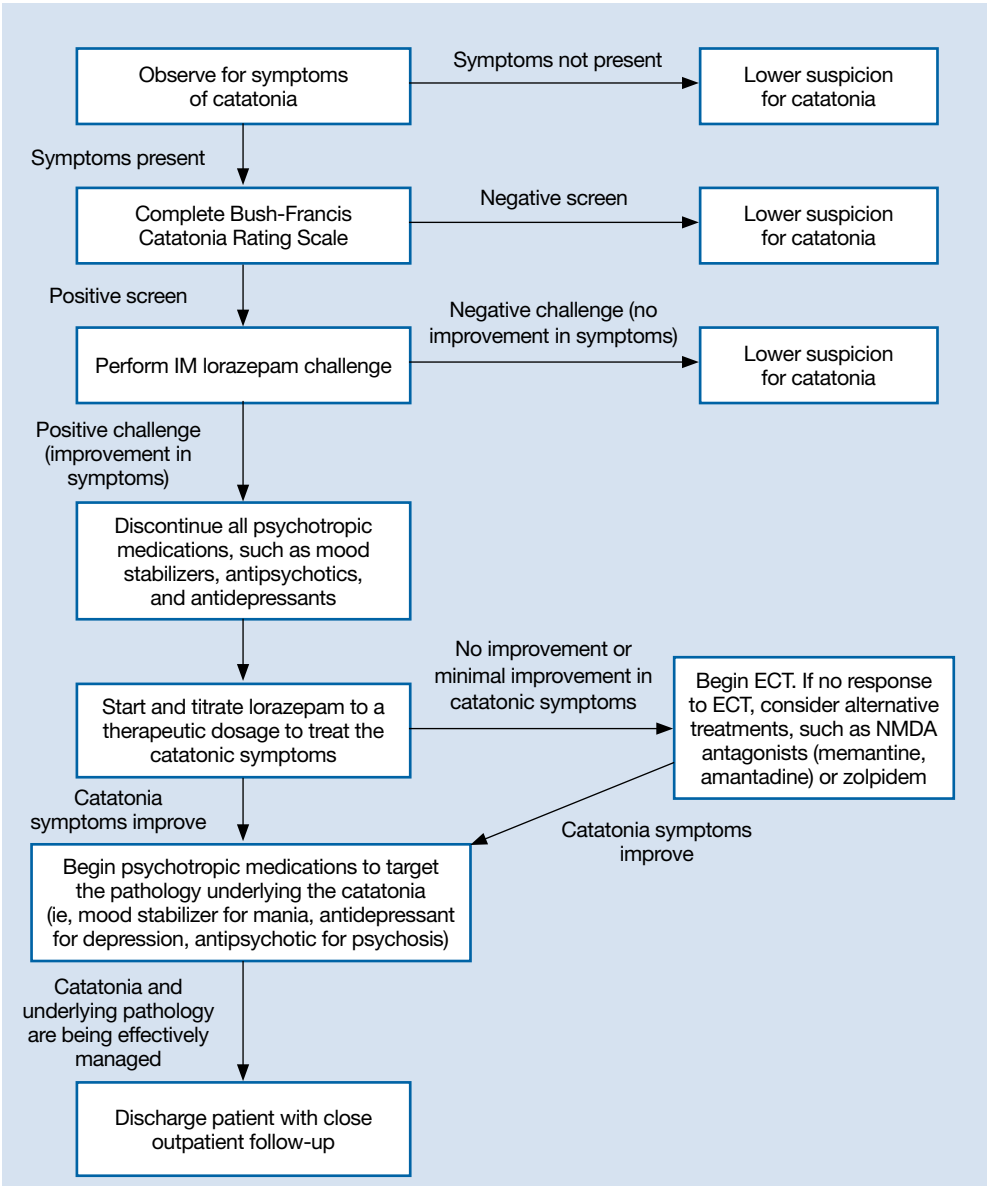
A stepwise approach can improve outcomes

The ***Figure*** (***page 31***) outlines the method we used to manage excited catatonia in these 3 cases. Each of these patients exhibited signs of excited catatonia, but because those symptoms were nearly identical to those of mania, it was initially difficult to identify catatonia. Excited catatonia was suspected after more typical catatonic symptoms—such as a stiff gait, slowed speech, and stereotypy—were observed. The BFCRS was completed to get an objective measure of the likelihood that the patient was catatonic. In all 3 cases, the BFCRS resulted in a positive screen for catatonia. Following this, the patients described in Case 2 and Case 3 received a lorazepam challenge, which confirmed their catatonia. No lorazepam challenge was performed in Case 1 because the patient was already receiving lorazepam when the BFCRS was completed. Although most catatonic patients will respond to a lorazepam challenge, not all will. Therefore, clinicians should maintain some degree of suspicion for catatonia if a patient has a positive screen on the BFCRS but a negative lorazepam challenge.

In all 3 cases, after catatonia was confirmed, the patient's psychotropic medications were discontinued. In all 3 cases, the antipsychotic was held to prevent progression to neuroleptic malignant syndrome (NMS) or malignant catatonia. Rasmussen et al[3] found that 3.6% of the catatonic patients in their sample who were treated with antipsychotics developed NMS. A review of prospective studies looking at patients treated with antipsychotics found the incidence of NMS was .07% to 1.8%.[5] Because NMS is often clinically indistinguishable

Figure

Managing excited catatonia: An algorithm



ECT: electroconvulsive therapy; NMDA: *N*-methyl-D-aspartate

Clinical Point

As the catatonia resolved with lorazepam, the manic symptoms became more easily identifiable

from malignant catatonia,[4,6] this incidence of NMS may have represented an increased incidence in malignant catatonia.

In all 3 cases, the mood stabilizer was held to prevent it from complicating the clinical picture. Discontinuing the mood stabilizer and focusing on treating the catatonia before targeting the underlying mania increased the likelihood of differentiating the patient's catatonic symptoms from manic symptoms. This resulted in more precise medication selection and titration by allowing us to identify the specific symptoms that were being targeted by each medication.

Oral lorazepam was prescribed to target catatonia in all 3 cases, and the dosage was gradually increased until symptoms began to resolve. As the catatonia resolved, the manic symptoms became more easily identifiable, and at this point a mood stabilizer was started and titrated to a therapeutic dose to target the mania. In Case 1 and Case 3, the antipsychotic was restarted to

Excited catatonia

Related Resources

- Dubovsky SL, Dubovsky AN. Catatonia: how to identify and treat it. Current Psychiatry. 2018;17(8):16-26.
- Crouse EL, Joel B. Moran JB. Catatonia: recognition, management, and prevention of complications. Current Psychiatry. 2018;17(12):45-49.

Drug Brand Names

Clozapine • Clozaril
Lithium • Eskalith, Lithobid
Lorazepam • Ativan
Risperidone • Risperdal
Divalproex sodium • Depakote

Clinical Point

For 2 of these patients, our approach resulted in a shorter length of stay compared to their previous hospitalizations

treat the mania more effectively. It was not restarted in Case 2 because the patient's mania was effectively being managed by 2 mood stabilizers. The risks and benefits of starting an antipsychotic in a catatonic or recently catatonic patient should be carefully considered. In the 2 cases where the antipsychotic was restarted, the patients were closely monitored, and there were no signs of NMS or malignant catatonia.

As discharge approached, the dosages of oral lorazepam were reevaluated. Catatonic patients can typically tolerate high doses of benzodiazepines without becoming overly sedated, but each patient has a different threshold at which the dosage causes oversedation. In all 3 patients, lorazepam was initially titrated to a dose that treated their catatonic symptoms without causing intolerable sedation. In Case 2 and Case 3, as the catatonia began to resolve, the patients became increasingly sedated on their existing lorazepam dosage, so it was decreased. Because the patient in Case 1 did not become overly sedated, his lorazepam dosage did not need to be reduced.

For 2 of these patients, our approach resulted in a shorter LOS compared to their previous hospitalizations. The LOS in Case 2 was 25 days; 5 years earlier, he had a 49-day LOS for mania and catatonia. During the past admission, the identification and treatment of the catatonia was delayed, which resulted in the patient requiring multiple transfers to the medical unit for unstable vital signs. The LOS in Case 3 was 22 days; 6 months prior to this admission, the patient had 2 psychiatric admissions that totaled 37 days. Although the patient's presentation in the 2 previous admissions was similar to his presentation as described in Case 3, catatonia had not been identified or treated in either admission. Since his catatonia and mania were treated in Case 3, he has not required a readmission. The patient in Case 1 was previously hospitalized, but information about the LOS of these admissions was not available. These results suggest that early identification and treatment of catatonia via the approach we used can improve patient outcomes.

References

1. Fink M, Taylor MA. The many varieties of catatonia. Eur Arch Psychiatry Clin Neurosci. 2001;251(Suppl 1): 8-13.
2. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. American Psychiatric Association; 2013:119-121.
3. Rasmussen SA, Mazurek MF, Rosebush PI. Catatonia: our current understanding of its diagnosis, treatment and pathophysiology. World J Psychiatry. 2016;6(4): 391-398.
4. Fink M, Taylor MA. *Catatonia: A Clinician's Guide to Diagnosis and Treatment*. Cambridge University Press; 2003.
5. Adityanjee, Aderibigbe YA, Matthews T. Epidemiology of neuroleptic malignant syndrome. Clin Neuropharmacol. 1999;22(3):151-158.
6. Strawn JR, Keck PE Jr, Caroff SN. Neuroleptic malignant syndrome. Am J Psychiatry. 2007;164(6):870-876.

Bottom Line

Excited catatonia can be challenging to diagnose and treat because it can present with symptoms similar to those seen in mania or psychosis. We describe 3 cases in which we used a stepwise approach to optimize treatment and improve outcomes for patients with excited catatonia. This approach may work equally well for other catatonia subtypes.