Are we failing to diagnose and treat the many faces of catatonia?

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I had seen many new and exciting presentations of psychopathology during my intern year, yet one patient was uniquely memorable. When stable, he worked as a counselor, though for any number of reasons (eg, missing a dose of medication, smoking marijuana) his manic symptoms would emerge quickly, the disease rearing its ugly head within hours. He would become extremely hyperactive, elated, disinhibited (running naked in the streets), and grandiose (believing he was working for the president). He would be escorted to our psychiatric emergency department (ED) by police, who would have to resort to handcuffing him. His symptoms were described by ED and inpatient nursing staff and residents as "disorganized," "psychotic," "agitated,"' or "combative." He would receive large doses of intramuscular (IM) haloperidol, chlorpromazine, and diphenhydramine in desperate attempts to rein in his mania. Frustratingly—and paradoxically—this would make him more confused, disoriented, restless, and hyperactive, and often led to the need for restraints.

This behavior persisted for days until an attending I was working with assessed him. The attending observed that the patient did not know his current location, day of the week or month, or how he ended up in the hospital. He observed this patient intermittently staring, making abnormal repetitive movements with his arms and hands, occasionally freezing, making impulsive movements, and becoming combative without provocation. His heart rate and temperature were elevated; he was diaphoretic, especially after receiving parenteral antipsychotics. The attending, a pupil of Max Fink, made the diagnosis: delirious mania, a form of catatonia.[1,2] Resolution was quick and complete after 6 bilateral electroconvulsive therapy (ECT) sessions.

Catatonia, a neuropsychiatric phenomenon characterized by abnormal speech, movement, and affect, has undergone numerous paradigm shifts since it was recognized by Karl Ludwig Kahlbaum in 1874.[3] Shortly after Kahlbaum, Emil Kraepelin held the belief that catatonia was a subtype of dementia praecox, or what is now known as schizophrenia.[4] Due to this, patients were likely receiving less-than-optimal treatments, because their catatonia was being diagnosed as acute psychosis. Finally, in DSM-5, catatonia was unshackled from the constraints of schizophrenia and is now an entity of its own.[5] However, catatonia is often met with incertitude (despite being present in up to 15% of inpatients),[1] with its treatment typically delayed or not even pursued. This is amplified because many forms of catatonia are often misdiagnosed as disorders that are more common or better understood.

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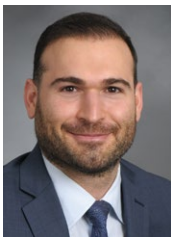

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Clinical Point

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Potential catatonia presentations

Delirious mania. Patients with delirious mania typically present with acute delirium, severe paranoia, hyperactivity, and visual/auditory hallucinations.[2,6,7] They usually have excited catatonic signs, such as excessive movement, combativeness, impulsivity, stereotypy, and echophenomena. Unfortunately, the catatonia is overshadowed by extreme psychotic and manic symptoms, or delirium (for which an underlying medical cause is usually not found). As was the case for the patient I described earlier, large doses of IM antipsychotics usually are administered, which can cause neuroleptic malignant syndrome (NMS) or precipitate seizures.[8]

Neuroleptic malignant syndrome. NMS is marked by fever, elevated blood pressure and heart rate, lead-pipe rigidity, parkinsonian features, altered mental status, and lab abnormalities (elevated liver enzymes or creatinine phosphokinase). This syndrome is preceded by the administration of an antipsychotic. It has features of catatonia that include mutism, negativism, and posturing.[9] NMS is commonly interpreted as a subtype of malignant catatonia. Some argue that the diagnosis of malignant catatonia yields a more favorable outcome because it leads to more effective treatments (ie, benzodiazepines and ECT as opposed to dopamine agonists and dantrolene).[10] Because NMS has much overlap with serotonin syndrome and drug-induced parkinsonism, initiation of benzodiazepines and ECT often is delayed.[11]

Retarded catatonia. This version of catatonia usually is well recognized. The typical presentation is a patient who does not speak (mutism) or move (stupor), stares, becomes withdrawn (does not eat or drink), or maintains abnormal posturing. Retarded catatonia can be confused with a major depressive episode or hypoactive delirium.

Catatonia in autism spectrum disorder. Historically, co-occurring catatonia and autism spectrum disorder (ASD) was believed to be extremely rare. However, recent retrospective studies have found that up to 17% of patients with ASD older than age 15 have catatonia.[12] Many pediatric psychiatrists fail to recognize catatonia; in 1 study, only 2 patients (of 18) were correctly identified as having catatonia.[13] The catatonic signs may vary, but the core features include withdrawal (children may need a feeding tube), decreased communication and/or worsening psychomotor slowing, agitation, or stereotypical movements, which can manifest as worsening self-injurious behavior.[14,15]

An approach to treatment

Regardless of the etiology or presentation, first-line treatment for catatonia is benzodiazepines and/or ECT. A lorazepam challenge is used for diagnostic clarification; if effective, lorazepam can be titrated until symptoms fully resolve.[16,17] Doses >20 mg have been reported as effective and well-tolerated, without the feared sedation and respiratory depression.[6] An unsuccessful lorazepam challenge does not rule out catatonia. If benzodiazepine therapy fails or the patient requires immediate symptom relief, ECT is the most effective treatment. Many clinicians use a bilateral electrode placement with high-energy dosing and frequent sessions until the catatonia resolves.[1,18]

In my experience, catatonia in all its forms remains poorly recognized, with its treatment questioned. Residents—especially those in psychiatry—must understand that catatonia can result in systemic illness or death.

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Clinical Point

Residents must understand that catatonia can result in systemic illness or death