Catatonia Update

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ABSTRACT

Catatonia is a neuropsychiatric condition characterized by physical presentations ranging from profound immobility to excessive motor activity. Emotional aspects of catatonia vary clinically between psychomotor retardation and extreme excitability. In the past, catatonia was considered to be a variant of schizophrenia. However, the disorder actually occurs as a clinical expression of many different psychiatric, neurologic, or medical diagnoses. A prompt diagnostic evaluation should identify any underlying diseases with consideration of somatic pathologies, especially those affecting central nervous system function. The workup focuses on a range of metabolic, traumatic, infectious, degenerative, autoimmune, drug-related, or other possible conditions, including psychiatric etiologies. Appropriate interventions should be instituted as quickly as possible to avoid complications like dehydration or deep vein thromboses. Symptomatic treatment commonly includes various pharmaceuticals or electroconvulsive therapy. Benzodiazepine drugs are, and have long been, the most preferred pharmacotherapy. These medications are usually fast acting and effective, are safe, and remain the catatonia treatment of choice.

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Catatonia is a neuropsychiatric condition clinically characterized by varying degrees of immobility or excessive motor activity. Catatonia is diagnosed by documenting 3 or more of the following: mutism, posturing, catalepsy, mannerisms, grimacing, negativism, waxy flexibility, stereotypy, stupor, agitation, echolalia, or echopraxia.[1] A lack of detail in the patient's history can complicate diagnosis. Subtypes of catatonia may include psychomotor retardation and excitation.[2] Either of these subtypes can occur among several different psychiatric, neurologic, and medical conditions. Previously, catatonia was classified as a variant of schizophrenia; however, it is now recognized as occurring in affective disorders and many other ailments as well.[1] Benzodiazepines are usually the preferred pharmacotherapy for treatment of catatonia.[2] Described here is the case of a woman who presented with catatonia. The history of the disorder, diagnostic considerations, and treatment recommendations are also discussed.

CLINICAL VIGNETTE

A 24-year-old woman presented to the hospital with an insidious onset of stupor, catalepsy, and mutism that began with depression, anhedonia, and suicidal thoughts 4 months prior. Recently, she had reportedly become mute, stopped eating, and lost weight. This dysfunction prompted a hospital admission.

The initial history from her parents revealed no psychiatric, medical, or substance use problems and no medicinal allergies. Later, her mother reported that after failing a high school final examination, her daughter evidenced a period of suspiciousness. Her father provided further details about an episode at age 12 years of elated mood and hypersexuality. Despite no intervention, she returned to normal function. In later years, she became a teacher, doing well with no psychiatric concerns. The physical examination was unremarkable except for generalized weakness. The patient exhibited psychomotor retardation, mutism, posturing, and waxy flexibility. The workup included a complete blood count, comprehensive metabolic panel, thyroid function tests, serum muscle enzyme studies, syphilis serology, a pregnancy test, toxicology screens, and a CT head scan without contrast. All assessment results were within normal limits. The diagnosis was bipolar affective disorder, with catatonia (*DSM-5* criteria).

Nasogastric tube feedings were initiated, facilitating lorazepam and olanzapine administration. The following day, the patient evidenced improvement, becoming responsive to verbal commands and less rigid. She exhibited sustained progress, and the nasogastric tube was removed. Her improvement continued, and she was discharged home. Clinic follow-up included a taper off of lorazepam. Since this was her second affective episode, mood stabilizer pharmacotherapy with divalproex was prescribed. The patient returned again to her usual baseline level of function. Olanzapine was continued as a maintenance medication, and management stressed medication compliance, adequate sleep, and maintaining outpatient care.

DISCUSSION

In 1874, Karl Kahlbaum first described catatonia in his book *Die Katatonie*.[3] Later, Emil Kraepelin included catatonia in dementia praecox, together with

hebephrenia and paranoia.[2] Eugen Bleuler, in 1908, renamed dementia praecox as schizophrenia, with catatonia as one of its subtypes.[2]

For over a century, catatonia was considered a manifestation only of schizophrenia. However, studies[2] of patients diagnosed with catatonia revealed that few of them met strict criteria for schizophrenia. Pharmacotherapy with antipsychotic medications often was documented as ineffective.[2] In an investigation[4] of 12 subjects with catatonia, 8 were initially diagnosed with schizophrenia and later reassigned to a bipolar disorder designation. Research results led to concern regarding catatonia and whether to consider it as a subtype of schizophrenia or some other condition.[2,5] Subsequently, revisions in the *DSM-IV*[6] defined a new category: catatonia, secondary to medical conditions. The *DSM-5*[1] reclassifies catatonia into 3 diagnostic types: (1) catatonia associated with several different psychiatric diagnoses, (2) catatonia associated with different medical conditions, and (3) an unspecified type.

The pathogenesis of catatonia is controversial, hypothesized to be caused by decreased γ-aminobutyric acid ($GABA_A$) and dopamine (D_2) receptor activity and increased activity at *N*-methyl-D-aspartate (NMDA) receptors.[7] Functional magnetic resonance imaging in catatonic subjects with a schizophrenia diagnosis revealed decreased activation in the motor cortex and disturbed hemispheric localization.[8] Other research[7] indicated a decreased density of $GABA_A$ receptors in the left sensorimotor cortex and right parietal cortex in people with akinetic catatonia. An investigation[8] utilizing single-photon emission tomographic imaging depicted a decrease in regional cerebral blood flow of the right frontoparietal cortex.

Catatonia is now recognized as a part of many different conditions. These conditions include psychiatric, medical, neurologic, autoimmune, intoxication, and drug withdrawal presentations.[2,9–15] Reportedly, between 10% and 15% of patients presenting with psychiatric complaints may actually exhibit catatonia.[16]

Affective disorders constitute the largest subgroup of patients who are diagnosed as meeting criteria for catatonia.[2] Approximately 46% of persons exhibiting catatonia were diagnosed with a comorbid mood disorder.[2] Patients with other psychiatric and psychotic disorders like schizophrenia and its variants, anxiety presentations, Tourette's disorder, and autism spectrum disorders also may present with catatonia.[17]

Catatonia is observed among a variety of neurologic disorders affecting the basal ganglia, limbic system, or the frontal, parietal, and temporal lobes. The pathology might include contusion, atrophy, neoplasm, or space-occupying lesions.[17] Catatonia can also occur in brain stem disorders with lesions at the third ventricle, thalamus, globus pallidus, caudate, and anterior putamen.[17] When individuals experience fear, the amygdala may produce catatonic-like states by acting on the stratum and supplementary motor areas. Neuroleptic malignant syndrome has been hypothesized to be a catatonic subtype.[2]

A type of autoimmune encephalitis, NMDA antibody encephalitis, is precipitated by NMDA receptor-1 antibodies and creates psychiatric symptomatology like catatonia.[18,19] There is an association with many underlying medical conditions (eg, ovarian teratomas), but the exact etiopathogenesis of NMDA antibody encephalitis remains unclear.[20] This illness can occur at any age but most often presents in young adults and in females (up to 80% of all cases).[18]

Infectious causes of catatonia might include various encephalitides of viral, bacterial, spirochetal, hydatid, and related autoimmune etiologies.[17] Several metabolic derangements may cause such symptomatology: hyponatremia, homocystinuria, hypercalcemia, porphyrias, and hepatic or renal failure. Among the endocrine disorders, catatonia has been described in persons with hypothyroidism, hyperthyroidism, hyperparathyroidism, and adrenal carcinoma. Intoxications and drug withdrawal can induce catatonia in cases precipitated by alcohol, sedatives, opiates, stimulants, hallucinogens, and possibly disulfiram.[17]

Clinically, it is important to understand these differentials to help determine diagnostic, follow-up, and treatment plans. The *DSM-5* criteria for diagnosis requires 3 or more of 12 clinical symptoms; however, consistent reference definitions are lacking. Multiple catatonia rating scales have been published with good interrater reliability and might assist in diagnosis and even more so to track treatment response.[21] Of these, the Bush-Francis Catatonia Rating Scale[22] is the most widely used in clinical practice and research.[21] A revised version[23] is used to evaluate persons with chronic schizophrenia. Electroencephalographic tracings might be another useful evaluation tool for catatonia.[24]

Timely clinical identification and intervention are imperative; delay may be associated with malnutrition or dehydration, infections, decubitus ulcers, contractures, and deep vein thromboses.[25] Pulmonary embolism is a potential cause of death in patients exhibiting catatonias, possibly facilitated by immobilization.[17]

In the 1930s, amobarbital was prescribed to treat patients with catatonia.[2] By the 1940s, ECT emerged and has remained a powerful alternative intervention, especially in treatment-resistant cases, although only as a short-term remediation.[2,26] In the 1980s, benzodiazepines became the favored pharmacotherapy, since these agents have a good safety profile, induce a rapid response, and are easy to administer.[2] Efficacy is dose dependent with the initial prescription of lorazepam 1–2 mg every 4–12 hours and titrated upward as tolerated to a usual range of 8–24 mg/d.[27–29] Response can be immediate in some patients but can take a few days to 1 week and even longer in some cases.[27,30] Patients with catatonia and a diagnosis other than schizophrenia evidenced over 80% recovery after benzodiazepine pharmacotherapy, while only 20%–30% recovered among those with a diagnosis of schizophrenia who were prescribed similar benzodiazepine medications.[31] These drugs are usually discontinued as symptoms resolve;

however, some patients may relapse quickly, warranting prolonged prescribing.[32,33] All underlying pathology should be treated following conventional guidelines. Supplementary Table 1 provides references on the use of lorazepam in patients with catatonia.

ECT is a definitive treatment for catatonia. Positive response rates at over 80% are documented even among patients in whom trials of lorazepam failed to help. Early initiation of ECT is advised to prevent medical complications. Predictors of good response may include acute onset with an affective disorder, while poor response is associated with an underlying neurologic disease. Right unilateral ECT may be effective and better tolerated than bilateral applications, but that contention is not uniformly accepted.[34,35] For malignant catatonia, ECT is generally considered as first-line therapy.[36] With the use of flumazenil, ECT can remain effective even in cases following lorazepam administration. Supplementary Table 2 provides references on the use of ECT in patients with catatonia.

ECT may not always be readily available, so in such cases, antipsychotic drugs can be coprescribed with benzodiazepines.[37] Such pharmaceuticals also help mitigate the possibility of mania or psychosis emergence and aid in treatment-resistant cases. Neuroleptic malignant syndrome is a risk when providing this pharmacotherapy, especially with first-generation versions; thus, second-generation antipsychotic drugs often are favored initially.[37] Antipsychotic medications are contraindicated in malignant catatonia, as they can worsen that symptomatology.[2,14] ECT is recommended in these cases. Amantadine, an NMDA antagonist, is also documented to have efficacy in some subjects.[7]

Our vignette highlights the importance of prompt recognition and intervention in bipolar disorders, especially for younger people. Due to the lack of characteristic discrete episodes, diagnosis might be difficult compared to adult-onset bipolar disorder cases.[38] As a result, some children with bipolar illness are often not identified until after experiencing years of psychopathology.[38] In the case presented here, the first 2 episodes of bipolar disorder went undiagnosed, and the patient subsequently presented as an adult with catatonia. Approximately 50% of adults with bipolar disorder experienced an onset of illness in childhood or adolescence, which sometimes results in a treatment delay.[39] The time-lag between the onset of first clinical symptom and initiation of medical intervention is correlated inversely with the age at disease onset.[40,41] In psychiatric illness–induced catatonias, early age at onset is associated with a more complicated course and an increased risk of substance abuse, suicide, and other comorbidities.[42]

CONCLUSION

It is important not to associate catatonia only in people with schizophrenia or other psychiatric diagnoses. Catatonias exist in many other medical and neurologic conditions, all of which require prompt recognition and intervention. As illustrated in the vignette, bipolar disorders should be within the differential diagnoses. The vignette also documented the effectiveness of lorazepam for diminishing symptoms of catatonia. The dual pharmacotherapy utilizing lorazepam and olanzapine was effective in managing our patient's clinical condition and potentially had efficacy in preventing the emergence of mania or psychosis.

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Supplementary material follows this article.

THE PRIMARY CARE COMPANION
FOR CNS DISORDERS

Supplementary Material

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List of Supplementary Material for the article

1. Table 1
2. Table 2

Disclaimer

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Table 1. References Citing the Prescribing of Lorazepam for Patients With Catatonia

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Table 2. References Citing ECT Treatments for Patients With Catatonia

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