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## Case based Review

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# Diagnostic overshadowing clouding the efficient recognition of pediatric catatonia: a case series

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**Abstract**

Catatonia is a neuropsychiatric condition that causes disruption of movement, emotion, and behaviors. Children and adults with underlying psychiatric conditions are particularly susceptible to developing catatonia, which may result in medical and psychiatric complications. Although catatonia research has been growing at a rapid rate in the last 20 years, it continues to be met with inefficiencies in its diagnosis and incertitude in its treatment. In the pediatric population, catatonia is plagued by diagnostic overshadowing, where the catatonia is erroneously attributed to existing pathologies that lead to a prolonged disease state. This paper describes three pediatric patients with catatonia that fell victim to diagnostic overshadowing. More rigorous training and education are imperative to improve the efficient recognition and treatment of children with catatonia.

## Introduction

Catatonia, a neuropsychiatric phenomenon, characterized by a constellation of abnormal movements, behaviors, and affect has gone through many paradigms shifts since its recognition by Karl Kahlbaum in 1874.[1] For over a century since its inception, it was primarily thought to be a subtype of schizophrenia leading to the belief that catatonia occurred less frequent than in actuality.[2] Catatonia finally became an independent entity in the latest edition of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).[3] However, if one strictly uses the current DSM for diagnosis, it can limit the recognition as it does not account for the phenotypic heterogeneity of the disorder, especially in the pediatric population.[4] Scales like the Bush-Francis Catatonia Rating Scale (BFCRS), Northoff, or Braunig Catatonia Rating scale can be useful in assessing the severity and monitoring of symptoms.[5–7]

Over the last two decades, research in catatonia and special populations (including the neurodevelopmental population) has expanded exponentially.[8] Several studies have estimated that between 12% and 20% of patients with autism spectrum disorder (ASD) and 18%–32% of children with an underlying psychotic disorder have comorbid catatonia.[9–15] Catatonia can also be present in various genetic abnormalities (i.e., Down syndrome, 22q13.3 deletion syndrome, Pradar-Willi, and Huntington's disease), tic disorders, intellectual disability, metabolic, endocrine, neurologic, and infectious diseases.[16–25]

Despite the growing number of studies, the recognition of catatonia seldom occurs in an efficient manner.[26] In many of the documented reports, the diagnosis can be delayed from months to years, which can lead to worsening functioning, poor self-care, physical injury/mutilation due to self-injury, and premature death.[13,27–31] This can be attributed to a variety of factors including lack of confidence in assessing and eliciting different catatonic features, and lack of knowledge of electroconvulsive therapy (ECT) in children, an extremely effective treatment for catatonia.[26,30,32]

Diagnostic overshadowing is a recurrent determinant in the delay of care of children with catatonia. Diagnostic overshadowing frequently gets applied in patients with an existing mental illness (commonly found in patients with intellectual disability and substance use disorders).[33–35] It is defined as attributing any new or worsening symptom/s to the underlying mental disorder, which frequently occurs in catatonia.[23,36] This oftentimes gets applied to children with ASD as many catatonic features overlap with the baseline disorder (i.e., stereotypy, mutism, echophenomenon, etc.) making it difficult to assess using the classic catatonia scales. Catatonia in ASD typically manifests as worsening slowness, difficulty in initiating and completing tasks, requiring physical and verbal assistance to perform basic actions, and increased passivity and poor motivation. There is also a general decrease in baseline functioning and worsening of the existing overlapping autistic and catatonic features including speech abnormalities (decreased speech output, alterations of tone, echolalia, and preservation). There have been scales developed specifically designed for ASD and catatonia such as the attenuated behavioral questionnaire (ABQ), which is a set of 34 items consisting of motor, affective, and behavioral symptoms.[11] The Kanner scale is also useful in developmental disorders.[37] The Pediatric Catatonia Rating Scale is a

validated tool for delineating catatonia among a wide array of psychiatric disorders in children and adolescents.[38] Nevertheless, diagnostic overshadowing persists in these patients. This typically results in the administration of multiple classes of psychotropic medications including selective serotonin reuptake inhibitors, first- and second-generation antipsychotics, atypical antidepressants, and alpha-blockers. All providing minimal benefit and potentially causing detriment to the child, such as antipsychotics exacerbating catatonia and/or causing neuroleptic malignant syndrome (NMS), a potentially lethal complication.[39]

Here, we describe cases of catatonia in children who suffered for a prolonged period largely due to diagnostic overshadowing.

## Case 1

A 12-year-old Hispanic girl had a steep decline in her baseline functioning for 4 months preceding her first psychiatric hospitalization. Before this, she was an honor roll student and had a strong family and friend network. She had no prior psychiatric or medical diagnoses. Her mental status decompensation was originally thought to be a major depressive episode as she was more isolated, withdrawn, and minimally engaged with others. Her eating habits decreased substantially. In the hospital, she made poor eye contact, barely spoke to the multiple examiners, and exhibited psychomotor slowing. She nodded when asked about suicidal ideation, however, would not verbalize any specific plan. She was started on escitalopram to target her depressed mood. Several days after this, she became agitated and started to repeatedly hit herself multiple times in the face with her fist and banged her head against the wall. These behaviors emerged suddenly and without provocation and continued intermittently for several days. Physical and 4-point restraints were needed to contain the patient. Intramuscular (IM) chlorpromazine and diphenhydramine were needed. The escitalopram was discontinued as it was thought to be the activating culprit. Several nurses observed the patient to have a bizarre expression and possibly internally preoccupied. The diagnosis at this time remained unclear, but theories of a potential psychotic illness given the steep decline in her functioning and a bipolar spectrum illness were also considered. At this time, more information was becoming known, and the patient did have significant psychological bullying at school and sexual abuse from an unknown perpetrator. Due to this posttraumatic stress disorder (PTSD) was considered. The patient was trialed on 400 mg of quetiapine, prazosin 4 mg, and aripiprazole 20 mg over the span of 3 months. The patient's condition declined during this period, as she routinely required restraints for dangerous self-injurious behaviors including repetitive self-punching, head banging, and scratching. She stopped eating and drinking, which caused her renal function to decline and required intravenous (IV) fluids. A full neurological workup including electroencephalogram, computed tomography head and neck, magnetic resonance imaging brain with and without contrast, were all negative. An autoimmune panel (including anti-N-methyl-D-aspartate antibodies) was negative.

Catatonia was finally considered after it was observed that after receiving a dose of lorazepam, her behaviors improved without apparent sedation. Also, a member of the treatment team observed and elicited mutism, staring, grimacing, negativism, waxy flexibility, stereotypy, impulsivity, and autonomic instability (elevated heart rate). She was started on a total of lorazepam 8 mg. No signs of sedation were observed. She had considerable improvement, but still engaged in intermittent self-injurious behavior and aggression. The lorazepam was not increased despite a positive response with no adverse effects. The patient was transferred to a state psychiatric unit for higher level treatment.

Catatonia may have many different underlying etiologies including psychological or physical trauma.[40,41] Historically, fear was thought to be the primary psychological factor of catatonia. In the earliest descriptions of catatonia, the patients were thought to be in extreme fright. Patients who were willing to be interviewed after they improved recalled feeling threatened and fear of dying. The catatonic reaction can be explained by an evolutionary response where behaviors like "tonic immobility" is developed after severe traumatic events or perceived danger. Some propose this process to be mediated by the vagal nerve.[42–44]

Dhossche et al.[40] highlight research on reactive catatonia as a trauma reaction. While classical symptoms of flashbacks, nightmares, panic attacks, and psychosis are readily recognized as acute stress reactions, catatonia is rarely included. Catatonia would be mistakenly labeled as a brief reactive psychosis, transient psychotic disorder, or pervasive refusal syndrome resulting in an unnecessarily extended recovery time.

The above case illustrates that the patient may have been experiencing significant reliving episodes from her trauma during these agitated moments. However, various catatonic signs were missed and misinterpreted as either behavioral, PTSD, or psychotic. Many members of the treatment team, including the child psychiatrist, child fellows, and residents, fell victim to diagnostic overshadowing. There was a failure to recognize that the self-injurious movements were stereotypy, her abnormal affect was grimacing; in addition, the withdrawal, mutism, negativism, and waxy flexibility, either were not assessed or falsely attributed to other processes. This led to the administration of many different classes of psychotropic medication without improvement as well as an extensive medical workup. The patient was also given a large dose of parenteral antipsychotics and anticholinergic medications, which is known to cause worsening confusion, agitation, seizures, and NMS in patients with catatonia.[39,45]

Lorazepam was started 7 months after the onset of symptoms and was underdosed given the partial response. The usual dose range of lorazepam is between 12 and 16 mg with some cases reporting success with over 25 mg.[30,46–48] Underdosing benzodiazepines is an indicator of a bigger issue that is caused by the general unfamiliarity of catatonia in child psychiatric providers and their hesitancy to use anti-catatonic agents. The fears of potential adverse effects from the benzodiazepines (i.e., oversedation and respiratory depression) are more pronounced than the catatonia itself. It should be noted that ECT was never fully explored for the patient. Similar to benzodiazepines, ECT is met with inexperience and misconceptions.[32]

## Case 2

A 15-year-old white male with a psychiatric history of bipolar disorder, psychosis, and cannabis use disorder presented to the psychiatric emergency room for suicidal ideation and visual hallucinations. He was withdrawn, severely thought blocked, and stared blankly into space. His mother reported that for 1 week, he had suddenly become bizarre, would not talk to anyone, and had a blank stare. He was subsequently diagnosed with an acute manic episode with psychotic features and started on risperidone 1 mg by mouth daily. During his stay in the emergency room, he had several physical holds due to aggressive behavior (trying to hit and bite staff members) and would receive multiple doses of IM haloperidol.

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Catatonia was considered after patient displayed mutism, withdrawal, staring, negativism, repetitive handwringing (stereotypy), and ambitendency. His vital signs were relatively stable except for elevated heart rate. Even after a diagnosis of catatonia, the treatment team continued to administer numerous doses of oral haloperidol. The patient became diaphoretic with tachycardia, fever, and rigidity; he was transferred to the medical emergency room for possible NMS. There, he received diphenhydramine for possible dystonia and was admitted to the medical inpatient unit for sustained elevated creatinine phosphokinase and liver enzymes. After the diagnosis of NMS, the antipsychotics were discontinued, and he was started on oral lorazepam 2 mg by mouth every 4 h with gradual improvement.

While catatonia may have various etiologies including neurological, genetic, endocrine, toxic, and metabolic causes; catatonia is most accompanied by an underlying mood disorder (i.e., bipolar disorder). In the above case, the treatment team attributed bizarre behaviors to affective psychosis while missing glaring features of catatonia like poor oral intake (withdrawal), staring into space (staring), and low speech output (mutism). These catatonic symptoms were overshadowed due to his prior psychotic and mood episodes. Despite the eventual recognition of catatonia, proper treatment was still delayed with the continuation of antipsychotic therapy until he developed NMS and was transferred out of the psychiatric emergency room. The administration of potent dopamine blockers likely precipitated a neuroleptic-induced malignant catatonia or NMS.[49] This further highlights the lack of knowledge of catatonia and anticatatonic treatments amongst all psychiatrists and child psychiatric providers.[32,50]

### *Case 3*

An 18-year-old male with a psychiatric history of ASD was brought to the outpatient clinic for psychiatric care. The patient was diagnosed with ASD when he was 18 months old. He had severe deficits in communicative behaviors, social interactions, and chronic stereotypical behaviors. However, throughout his early adolescent and childhood years, he was relatively stable without pharmacotherapy and did well with behavioral, speech, and occupational therapy.

His parents sought a psychiatric consultation for their son due to a change in his baseline behaviors. They attributed some of the changes due to the pandemic and lockdown. Over the preceding months, he became more anxious and restless. He began pacing in a purposeless manner, making more unusual hand motions, rocking in the chair, and screaming for 15 minutes at a time without provocation. Other behaviors included verbal perseveration, impulsivity, and agitation (without self-injury). His parents initially took him to a neurology physician assistant who prescribed the patient guanfacine for the hyperactivity. The dose was titrated to 2 mg. After 2 months of ineffectiveness, the medication was stopped. It was also discovered that 3 years prior, the patient was medically hospitalized for severely decreased oral intake and malnutrition. This resulted in a low hemoglobin and hematocrit that required a transfusion of packed red blood cells. His food intake gradually improved with the aid of a nutritionist.

On physical examination, the patient was constantly moving, maintained poor eye contact, exhibited echopraxia/echolalia, repetitive hand movements and rocking motions, repeated words continuously, and maintained abnormal postures. A BFCRS was done, and he scored a 24 for staring, echophenomena, stereotypy, mannerism, perseveration, verbigeration, waxy flexibility, catalepsy, excitement, impulsivity, ambitendency, and automatic obedience.

Lorazepam was initiated to target the catatonic symptoms. The dose of 2 mg twice daily was started with an almost immediate resolution in staring, excitement, and ambitendency. He would still scream for long periods of time without reason. Lorazepam was increased to 10 mg in divided doses per day with a resolution in these episodes. The BFCRS was decreased to 14 as he scored for some of his pre-catatonic ASD baseline behaviors. There was no sedation, gait instability, or decreased respiratory drive from this high dose. His condition is currently stable on lorazepam 10 mg for the last 6 months.

## Discussion

These cases illustrate the propensity of clinicians to overlook catatonia in children and waver in terms of its proper treatment. In these cases, diagnostic overshadowing was a prominent feature that played a role in the poor recognition of catatonia. Symptoms of catatonia like mutism, withdrawal, aggression, and staring, were labeled as part of depression, psychosis, mania, or a trauma reaction, while other catatonic features went unnoticed like waxy flexibility, rigidity, negativism, and stereotypy, which either need to be carefully examined or elicited. As shown in the third case, catatonia is oftentimes overshadowed in patients with ASD as many of the ASD symptoms overlap with catatonia. The diagnosis of catatonia could have been made at several points in time including the hospitalization secondary to withdrawal and initial neurology appointment. All these behavioral changes (worsening agitation and stereotypy, impulsivity, and perseveration) were attributed to the patient's baseline ASD. In addition, the catatonia onset may have been several years prior when he was hospitalized due to malnutrition secondary to withdrawal. Several pivotal studies by Wing and Shah[9] and Breen and Hare[11] found ASD to be a risk factor for catatonia with a prevalence of 17% and 20% of ASD patients developing catatonia, respectively. However, as demonstrated by Ghaziuddin et al.,[26] it often goes missed as only 11% were given the correct diagnosis of catatonia.

Catatonia has been confined as a type of schizophrenia for over a century with it only being recognized as its own disorder in the DSM-5 in 2013. But the downstream effects of catatonia's history are still being felt today as it is undertaught in psychiatry residencies with residents and attendings possessing a severe lack of baseline knowledge of the phenomenon.[50,51] In addition, inconsistent diagnostic criteria add confusion and can further delay diagnosis and treatment; many use the DSM-5 criteria for catatonia that has 12 features, but can miss automatic obedience, passive obedience, rigidity, perseveration, verbigeration, ambitendency, grasp reflex, and autonomic instability that are not included.[3,6] Anticatatonic treatments, like ECT are also encountered with misconceptions. Like in our cases, ECT is commonly overlooked and typically reserved for severe, benzodiazepine refractory patients. This is caused by the lack of ECT knowledge in many child psychiatric providers. In a study, only 5.2% of child psychiatry providers believed they had expertise in ECT while 53.8% had negligible knowledge; conversely, 53% and 27% of these same respondents thought it to be unsafe in prepubertal and adolescent patients, respectively.[32] Unsubstantiated beliefs of ECT-induced permanent brain damage continue to be dominant despite contrary evidence.[52–56] During a state of agitated catatonia, patients are more likely to sustain significant physical and psychological

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damage that encompasses cerebral and scalp contusions (from repetitive head banging), soft tissue injury, prolonged time in physical restraints, numerous antipsychotic injections, dehydration, muscle contractures, electrolyte imbalance, skin breakdown, poor nutrition, and time spent away from home; these injuries are overshadowed by the perceived harms of treatment. Furthermore, NMS, the malignant version of catatonia seen in the 2nd case, where a patient would develop rigidity, fever, autonomic instability, and confusion, can lead to death in 5%–20% if left mis- or untreated.[57–60]

In conclusion, education and training are greatly needed to improve the proficiency in proper assessment and treatment of patients with catatonia. Timely catatonia assessment is paramount for any child who has an acute change in baseline functioning or behavior. A detailed history, physical examination, laboratory testing (i.e., toxicology, infectious panel, metabolic, endocrine functioning, etc.), and other imaging or neurological testing, if necessary, should be conducted.[23,48] Training in the various catatonia rating scales should be sought out to properly diagnose and treat patients with catatonia.

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