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Catatonia and associated psychopathologies in adolescence: case series and a brief review

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ABSTRACT

Catatonia is a severe motor dysregulation syndrome observed in both psychiatric and medical conditions, with potentially fatal outcomes if untreated. This report presents three adolescent cases diagnosed with catatonia; each associated with distinct psychiatric pathologies. Despite varying symptoms, including negativism, mutism, echolalia, and rigidity, all cases responded to benzodiazepine treatment, with one requiring electroconvulsive therapy due to malignant catatonia. This brief review discusses the diagnostic challenges, treatment approaches, and importance of early intervention in catatonia. The comparison of symptoms across cases highlights the role of differential diagnosis in optimizing treatment outcomes for pediatric catatonia.

Keywords: Catatonia, adolescents, benzodiazepines, electroconvulsive therapy, differential diagnosis

INTRODUCTION

Catatonia is a motor dysregulation syndrome rather than a diagnosis and may have psychiatric, neurological, autoimmune or toxic etiologies.[1] The incidence in psychiatry clinics among adults varied from 6 to 38%,[2] while it has been reported between 0.6-17.7% in child and adolescent psychiatry clinics.[3,4] Although rare, morbidity and mortality are high in children and adolescents.[5,6]

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5),[7] the diagnostic criteria for catatonia require at least three of the following twelve symptoms: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerism, stereotypy, agitation, grimacing, echolalia, and echopraxia. However, these criteria have been critiqued for redundancy, and the absence of time criteria poses challenges, particularly in pediatric populations. Common signs of catatonia in all cases are posturing, waxy flexibility, staring, negativism, withdrawal and social withdrawal. Catalepsy is considered an advanced degree of posturing,[8] while some authors consider it under the heading of waxy flexibility. Posturing and waxy flexibility were observed in all cases, so catalepsy could be considered an indirect positive symptom. This is why the DSM-5 criteria are criticized for repetition. More than 40 symptoms associated with catatonia have been described in literature. In children and adolescents, withdrawal, refusal to eat or drink, and social withdrawal are often prominent symptoms that demand special attention. The absence of time criteria in DSM-5 is also considered as a limitation.

Excitation, agitation, talkativeness, and disorganization, when observed alongside catatonic symptoms, are collectively referred to as excited catatonia (also known as delirious mania or Bell's mania).[9] Although excited catatonia and delirious mania are often considered part of the same spectrum, the term excited catatonia is more appropriate when catatonic symptoms predominate, as delirious symptoms tend to be more severe in cases classified as delirious mania. One of the most severe complications of catatonia is malignant catatonia, which has been frequently reported in undiagnosed or untreated cases.[6] Malignant catatonia is characterized by autonomic and thermoregulatory dysfunctions, with clinical manifestations including fluctuations in blood pressure and heart rate, diaphoresis, high fever, laboratory abnormalities, and a general deterioration of the patient's condition. If left untreated, the mortality rate for malignant catatonia ranges from 10% to 20%, with respiratory failure being the most common cause of death.[10,11] Another less common subtype, periodic catatonia, is defined by the rapid onset of repetitive hypokinetic and hyperkinetic episodes lasting 4 to 10 days.[12] These episodes can recur over weeks or even years. Some cases of periodic catatonia exhibit a familial pattern with autosomal dominant inheritance, linked to abnormalities on chromosome 15q15, which are associated with syndromes such as Prader-Willi and Angelman.[13]

Bush Francis Catatonia Rating Scale,[8] Modified Rogers Scale, Northoff Catatonia Rating Scale, Brauning Catatonia Evaluation Scale and Kanner Scale are used frequently.[14]

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The Bush Francis Catatonia Rating Scale (BFCRS), which is the gold standard in studies and is used widely, consists of 23 items (30 findings) and was developed based on the definitions in DSM and international statistical classification of diseases (ICD) classifications, as well as Kahlbaum, Krapelin. Pediatric Catatonic Rating Scale (PCRS) was developed as a modified version of the BFCRS for children and adolescents.[15] In addition to the 17-item of the BFCRS, six symptoms were added based on the analysis of 463 catatonic cases and the review of descriptions of pediatric catatonia: (1) Withdrawal was divided into refusal to eat/drink and social withdrawal to facilitate differentiation with other pediatric psychiatric disorders such as eating disorders or autism spectrum disorders (ASD) (2) Incontinence, a symptom of general psychomotor regression, highly reported in children with catatonia[16-18] (3) Automatic compulsive movements were proposed instead of automatic obedience and were considered a presentation of psychomotor automatism (4) Schizophasia and acrocyanosis were added as an indication of malignant catatonia in youths in addition to autonomic abnormality, and (5) grimacing was combined with mannerism (**Table 1**).

Dysfunction in multiple neurotransmitter systems has been implicated in the pathophysiology of catatonia. The glutamatergic system, mediated by N-methyl-D-aspartate (NMDA) receptors, plays a critical role, as evidenced by the induction of catatonia-like symptoms in healthy individuals following the administration of ketamine, an NMDA receptor antagonist.[19] Similarly, the presence of catatonic symptoms in anti-NMDA receptor encephalitis and the therapeutic efficacy of amantadine, an NMDA receptor agonist, further underscore the involvement of NMDA receptor dysregulation in catatonia.[20] The GABAergic system is equally significant, with high doses of benzodiazepines, which act on the benzodiazepine-sensitive GABA-A receptor complex, demonstrating robust efficacy in alleviating catatonic

Table 1. Comparison of catatonia symptoms in evaluation

	DSM-5	BFCRS	PCRS
Motor symptoms			
Catalepsy (passive induction of a posture held against gravity)	+	+	+
Posturing (maintaining constant posture)	+	+	+
Stereotypy (repetitive, abnormally frequent, non-goal directed movements)	+	+	+
Stupor/immobility (no psychomotor activity or decreased)	+	+	+
Agitation (not influenced by external stimuli)	+		
Mannerism (odd, purposeful movements)	+	+	+
Waxy flexibility (allows repositioning)	+	+	+
Grimacing (maintenance of odd facial expressions)	+	+	
Echopraxia (mimicking movements)	+	+	+
Negativism (resistant to instructions)	+	+	+
Staring (fixed gaze, decreased blinking)		+	+
Grasp reflex (automatic closure of palm when touched)		+	
Rigidity (rigid position despite efforts to be moved)		+	+
Impulsivity (suddenly engages in inappropriate behavior without provocation)		+	
Automatic obedience (exaggerated cooperation)		+	
Mitgehen (arm rising with light pressure, “anglepoise lamp”)		+	
Gegenhalten (resistance to passive movements)		+	
Ambitendency (indecisive hesitant movement)		+	
Perseveration (repeating same topic or movement)		+	
Automatic compulsive movements (involuntary muscle activity exhibited in posture, attitudes, mimic or gesture due to inhibition or forced motoraction			+
Excitement (extreme hyperactivity)		+	+
Other symptoms			
Mutism (verbally unresponsive or very little response)	+	+	+
Echolalia (mimicking speech)	+	+	+
Verbigeration (repetition of nonsensical phrases)		+	+
Withdrawal (refusal to eat/drink)			+
Social withdrawal (refusal to make eye contact)			+
Schizophasia (scrambled speech)			+
Acrocyanosis (cyanosis of the extremities)			+
Autonomic abnormality (change in temperature, BP, pulse, laboratory findings and diaphoresis)			+
Incontinence (nocturnal enuresis, daytime urinary incontinence, fecal incontinence)			+
Combativeness (belligerence, aggression)		+	

DSM-5: Diagnostic and Statistical Manual of Mental Disorders-5, BRCRS: Bush Francis Catatonia Rating Scale, PCRS: Pediatric Catatonic Rating Scale, BP: Blood pressure

symptoms. This response highlights potential changes in GABA-A receptor function as a key mechanism. Although alterations in GABAergic and glutamatergic activity likely contribute to various catatonic phenotypes, the relative involvement of these systems may vary across different types of catatonias. The role of the dopaminergic system, while less well-defined, warrants further investigation to fully elucidate its contribution to catatonia.[21]

Here three adolescents with catatonia will be presented and clinical presentation, differential diagnosis and management of catatonia will be reviewed. The catatonia symptoms of cases were compared in **Table 2**. The informed consents were received from the cases and their families for this report.

Table 2. Comparison of catatoni symptoms between cases

	Case 1	Case 2	Case 3
Catatonic symptoms			
Catalepsy			
Posturing	+	+	+
Stereotypy			
Stupor/immobility	+	+	
Agitation			+
Mannerism			
Waxy flexibility	+	+	+
Grimacing	+		
Echopraxia			+
Negativism	+	+	+
Staring	+	+	+
Grasp reflex	+		
Rigidity	+		
Impulsivity			+
Automatic obedience	+		
Mitgehen			
Gegenhalten			
Ambitendency	+		+
Perseveration			+
Automatic compulsive movements			
Excitement			+
Mutism	+	+	
Echolalia	+		+
Verbigeration			+
Withdrawal	+	+	+
Social withdrawal	+	+	+
Schizophasia			+
Acrocyanosis			
Autonomic abnormality	+		
Incontinence	+		
Combativeness			+
Other symptoms			
Psychotic symptoms	+	+	+
Disorganization in speech and behavior	+		+
Depressed mood	+	+	
Emotional lability			+

CASE PRESENTATIONS

Case 1

A 17-year-old male with a history of bipolar affective disorder (BAD) for 11 months, Specific Learning Disorder, and attention deficit hyperactivity disorder (ADHD) since the age of eight, was brought to the emergency department by his family due to a progressive decline in sleep, movement, speech, and daily functioning over the past week. The family described an abrupt onset of symptoms, beginning with episodes of lying motionless on the sofa for extended periods, followed by standing motionlessly in front of the refrigerator for hours and refusing to eat or drink for three consecutive days.

The patient had been receiving olanzapine for BAD; however, it had been switched to aripiprazole (5 mg/day) three weeks prior due to metabolic side effects. He had no family history of psychiatric disorders. On examination, the patient was uncooperative, mute, slow in movements, and exhibited rigidity. He refused to sit, stood with a dull expression, stared at a fixed point, avoided eye contact, and did not follow commands. He displayed negativism, resisting passive movement of his extremities.

Clinical findings included subfebrile fever, tachycardia, hypertension, and excessive sweating. Laboratory results revealed impaired liver function tests, leukocytosis, and elevated creatine kinase (CK) levels, although imaging (brain MRI), EEG, lumbar puncture, blood cultures, and toxicology were unremarkable. Due to the severity of his condition, diazepam (10 mg, intramuscular) was administered, but minimal improvement was observed after two days of treatment. Oral lorazepam (2 mg/day) was initiated, leading to slight improvement, including eating, drinking, and answering simple questions.

The patient was diagnosed with Malignant Catatonia related to mood disorder and admitted to an inpatient psychiatry clinic. Despite increasing lorazepam to 10 mg/day, symptoms persisted, including echolalia, ambivalence, and agitation. Bilateral electroconvulsive therapy (ECT) was initiated, with treatments administered twice weekly. After eight weeks, his symptoms significantly improved, and his BFCRS (Bush Francis Catatonia Rating Scale) score reduced from 29/69 to 16/69 within one week of ECT initiation and further to 3/69 after three weeks.

During treatment, his mood and motor symptoms improved; however, persistent grandiose and persecutory delusions with disorganized speech and behavior became evident. Treatment with valproic acid (1000 mg/day) and clozapine (300 mg/day) was initiated alongside ongoing ECT. While mood symptoms resolved, psychotic symptoms persisted, leading to a diagnosis of schizoaffective disorder. The patient was discharged on mood stabilizers and antipsychotics for outpatient follow-up. Despite poor insight and persistent psychotic symptoms during six months of follow-up, he managed daily activities effectively.

Case 2

A 16-year-old female with no prior medical or psychiatric history presented to the emergency department with confusion, weakness, and fainting. The family reported a 13 kg weight loss over two months, insomnia, a lack of self-care,

and excessive fear for her family's safety. The patient refused to allow her father to go to work or her sister to bathe. Upon examination, she was agitated, mute, and immobile, with no evaluable orientation or thought content. Findings included negativism, mutism, irritability, and avoidance of eye contact.

Initial assessments revealed electrolyte imbalances, hypoglycemia, and ketoacidosis. Fluid therapy was initiated, and imaging (brain MRI), EEG, and urine toxicology tests were normal. A diagnosis of catatonia related to psychiatric pathology was established, and lorazepam (2 mg/day) was administered. Although this improved her ability to provide brief responses, she continued refusing food and required hospitalization with nasogastric feeding.

Further history revealed an episode one year prior involving disinhibition, agitation, and hallucinations, treated briefly with olanzapine before being discontinued without follow-up. Subsequently, she exhibited withdrawal, repetitive questioning, restlessness, and emotional outbursts. Her family noted increasing social isolation over the two months preceding hospitalization.

During inpatient care, the patient gained 3 kg over two weeks and showed decreased motor hypoactivity but continued mutism. She was discharged on olanzapine (15 mg/day) and lorazepam (2 mg/day), with lorazepam gradually discontinued during follow-up. Olanzapine was increased to 20 mg/day, leading to improvements in sleep, eating, and self-care, though short answers, affective bluntness, and persecutory delusions persisted.

Her clinical history suggested catatonia secondary to schizoaffective disorder. While she could not return to school, psychotic symptoms were manageable with ongoing follow-up.

Case 3

A 15-year-old female, previously healthy, presented to the emergency department with bizarre behavior, pressured speech, and insomnia. The insomnia began 20 days earlier, with her sleeping only 2.5 hours per night. After three days, she developed crying episodes and fears that others doubted her abilities. She expressed irrational fears, such as aversion to certain letters ("A" and "E") and concern about bread being infected with a virus.

The patient was agitated, talkative, and fearful during examination, with disorganized speech and persecutory delusions. Neurological findings included echolalia, ambivalence, excitation, and waxy flexibility. Despite disorganized thought processes, her orientation was intact. A family history of schizophrenia was noted in a distant relative.

Vital signs and laboratory evaluations, including imaging (brain MRI), EEG, lumbar puncture (negative for encephalitis autoantibodies), and urine toxicology, were unremarkable. After oral lorazepam (1 mg), the patient began eating, drinking, and calming down. Lorazepam was increased to 4 mg/day, leading to resolution of catatonia symptoms within two weeks.

She was diagnosed with catatonia related to mood disorder and discharged on olanzapine (15 mg/day) and lithium (900 mg/day). During outpatient follow-up, no mood or catatonia symptoms were observed after four weeks.

DISCUSSION

The diagnosis of catatonia in each case was based on DSM-5 criteria and the BFCRS. Approximately 20% of catatonia cases in children and adolescents are associated with underlying medical conditions.[22] During the evaluation, medical pathologies such as infectious, neurological, and metabolic diseases must be thoroughly investigated. Common associated conditions include genetic disorders (e.g., Prader-Willi syndrome, Down syndrome, Huntington disease), neurological conditions (e.g., seizures, trauma), infectious diseases (e.g., viral encephalitis), toxic states (e.g., Ecstasy-induced), and autoimmune disorders.[22,23] Effective prognosis and significant improvement in catatonia can be achieved when specific etiological treatment is combined with catatonia treatment.[22,24,25] The Causality Assessment Score (CAUS) can assist in distinguishing autoimmune from non-autoimmune cases.[25]

Autoimmune disorders such as anti-NMDA receptor encephalitis,[26] systemic lupus erythematosus (SLE),[27] Hashimoto encephalopathy (HE),[28] and pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS) or pediatric acute-onset neuropsychiatric syndrome (PANS)[29,30] often present with acute-onset severe psychiatric symptoms, including catatonia. Diagnosis in these cases relies on identifying antibody levels in cerebrospinal fluid and evaluating neurological findings such as atypical EEG patterns and cranial MRI results.

When catatonia occurs without any underlying organic disease, a detailed psychiatric examination and long-term follow-up are essential. In adults, mood disorders, schizophrenia, posttraumatic stress disorder, and eating disorders are the most commonly associated psychiatric conditions, whereas in children and adolescents, schizophrenia spectrum disorders, ASD, Down syndrome, mood disorders, and obsessive-compulsive disorders are more prevalent.[2] Catatonia is more common in males within pediatric populations, likely due to the higher prevalence of schizophrenia spectrum disorders in this demographic, as opposed to the higher prevalence of mood disorders in adult females.[31-33] Differential diagnosis in children with ASD and developmental disorders can be particularly challenging since overlapping symptoms such as stereotypies and echolalia may mask catatonic features.[34] A prevalence of catatonia in ASD has been reported at 17%, with four core symptoms aiding in differential diagnosis:[35] 1. Increasing psychomotor and verbal slowness, 2. Difficulties in initiating and completing actions, 3. Increased reliance on physical or verbal prompts, 4. Increased passivity and apparent lack of motivation.

In the present cases, underlying medical and psychiatric conditions were carefully evaluated. Case 1 involved BAD and a family history of mood disorders in Case 2 suggested a psychiatric etiology for catatonia. In Case 3, the absence of prior psychiatric symptoms supported a diagnosis of acute-onset catatonia with manic/mixed mood symptoms. Regardless of the underlying cause, benzodiazepines and ECT remain the cornerstone of catatonia treatment.[2]

In Case 1, significant weight gain (20 kg over five months) during olanzapine treatment for BAD required a switch to aripiprazole. The slower titration and reduced mood-stabilizing effects of aripiprazole compared to olanzapine may have contributed to psychotic depressive symptoms and catatonia.

The absence of improvement with benzodiazepines and the clinical presentation of malignant catatonia necessitated ECT, which resulted in marked improvement.[36] The decrease in BFCRS scores and clinical symptoms highlights the utility of BFCRS for tracking symptom recovery in children.

In Case 2, severe refusal to eat and drink resulted in electrolyte imbalances, dehydration, and ketoacidosis, posing life-threatening risks. Benzodiazepines such as lorazepam produced a rapid response,[37] but nasogastric feeding was essential to prevent refeeding syndrome. This highlights the importance of timely fluid and nutritional support in catatonia management.[38]

In Case 3, primary symptoms such as disorganized speech and behavior, affective lability, and excitation were associated with manic/mixed episodes. Treating only mood and psychotic symptoms without addressing catatonia could have worsened the condition. This case underscores the importance of detailed psychiatric evaluation and targeted treatment for excited catatonia, as excessive motor activity in excited catatonia may increase the risk of progression to malignant catatonia. Fortunately, malignant catatonia was not observed in this case.

Antipsychotics during the acute phase of catatonia are not recommended due to the risk of malignant catatonia and deterioration of catatonic symptoms. Some reports suggest limited benefits of second-generation antipsychotics like aripiprazole[39,40] and quetiapine[41] during acute catatonia treatment. However, the lack of repeated assessment and concurrent benzodiazepine administration in these studies limits the ability to determine the true efficacy of these drugs. Antipsychotics should be introduced cautiously after the resolution of catatonic symptoms to manage underlying psychiatric disorders. Given the higher sensitivity of children to neuroleptic-induced motor symptoms,[42] these drugs must be used sparingly in pediatric cases. In Case 1, antipsychotics were introduced after the stabilization of catatonia to address persistent psychotic symptoms. Despite the addition of mood stabilizers and antipsychotics, psychotic symptoms such as disorganized behavior, derailments in thought processes, and grandiose and persecutory delusions persisted. This highlights the importance of long-term follow-up in cases where initial diagnoses may shift, as demonstrated by the reclassification of this case to a schizophrenia spectrum disorder.[43]

The findings in these cases emphasize the need for individualized treatment plans, combining benzodiazepines, supportive care, and cautious use of antipsychotics to manage catatonia and its underlying causes effectively.

CONCLUSION

This study is subject to several limitations. The small sample size of only three cases restricts the generalizability of the findings, and larger case series or cohort studies would provide more robust evidence. Additionally, the absence of a control group or randomized comparisons limits the ability to draw definitive conclusions about the effectiveness of treatment methods. Limited information on long-term functionality and psychiatric recovery, particularly regarding the persistence of psychotic symptoms in Case 1, further highlights the need for extended follow-up studies. Catatonia, a complex syndrome associated with various medical and psychiatric conditions, requires a thorough differential diagnosis in children and adolescents. Timely recognition and treatment of medical etiologies are crucial for improving prognosis. While benzodiazepines are the primary treatment option, ECT remains highly effective for refractory cases. However, untreated catatonia carries a significant risk of progression to malignant catatonia, which is associated with a high mortality rate. A multidisciplinary approach, including medical management, supportive care, and psychiatric follow-up, is essential for successful treatment. The lack of randomized controlled studies in pediatric populations remains a critical limitation that warrants further research.

ETHICAL DECLARATIONS

Informed Consent

All parents signed an explicit and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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