

## Case Series

## CATATONIA IN ADOLESCENTS: A CASE SERIES

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Corresponding Author:  
**Dr. Aarti Karahda,**  
 Email: karahdaarti@gmail.com

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**Mohit Gupta<sup>1</sup>, Madhusudhan Gupta<sup>2</sup>, Ashish Aggarwal<sup>3</sup>, Aarti Karahda<sup>4</sup>, Ritika Jindal<sup>5</sup>**

<sup>1</sup>Assistant Professor, Department of Pediatrics, NC Medical College, Israna, Panipat, Haryana, India.

<sup>2</sup>Associate Professor, Department of Pediatrics, NC Medical College, Israna, Panipat, Haryana, India.

<sup>3</sup>Assistant Professor, Department of Psychiatry, Kalpana Chawla Government Medical College, Karnal, Haryana, India.

<sup>4</sup>Senior Resident, Department of Psychiatry, Kalpana Chawla Government Medical College, Karnal, Haryana, India.

<sup>5</sup>Associate professor, Department of Anaesthesia, Kalpana Chawla Government Medical College, Karnal, Haryana, India.

**Abstract**

Catatonia is a neuropsychiatric syndrome with well researched clinical features. Though the criteria for diagnosis of catatonia are well established, there is paucity of understanding regarding the various etiology of this syndrome. Also, most of the data is available from adult patients. There is a paucity of literature on catatonia in children and adolescents. We hereby present a case series of 3 cases all of the patients being adolescents, who presented with features suggestive of catatonia as presenting features and which were all diagnosed to be because of different underlying conditions. The importance of diagnosis of underlying disorder and prompt management of catatonia in adolescents is emphasized.

**INTRODUCTION**

Catatonia is a neuropsychiatric syndrome characterized by abnormal movements, behaviors, and withdrawal. Catatonic symptoms can be subdivided into the following four categories: motor phenomena (stupor, catalepsy, rigidity, flexibilitas cerea), withdrawal (mutism, staring gaze, negativism, refusal to eat and drink), excitement (hyperactivity, combativeness, autonomous instability, undirected potentially dangerous aggression) and odd repetitive behavior (grimacing echolalia, echopraxia, stereotypies, mannerisms, perseveration, command-automatism, Mitmachen, Gegenhalten, and ambitendency).<sup>[1]</sup>

Kahlbaum established that psychomotor syndrome not only was associated with affective illnesses but could be seen in various organic disorders, including epilepsy. Convulsions, fits, and tetanic muscular clenching were common elements in his case histories, and these could have a complex and variable relationship with the course of the disease. He identified catatonia as a "symptom complex", characterized by psychotic negativism, catalepsy, mutism, stereotypies, verbigeration (speech consisting of repeated, meaningless words and sentences), and muscular symptoms, he anticipated that etiology of catatonia would ultimately be found to lie in pathological changes of brain structure.<sup>2</sup> Catatonia has various subtypes classified as

chronic or acute, isolated acute episodes versus chronic recurrent or periodic catatonia retarded or excited forms.<sup>3</sup> Kahlbaum proposed catatonia as more than a non-specific, cross-sectional syndrome and has a cyclic, alternating course.<sup>[2]</sup>

Catatonia has been observed in a plethora of psychiatric disorders, primarily mood and psychotic disorders, but sometimes in cases of, for example, autism or obsessive compulsive disorder (OCD) as well. While catatonia could be an independent diagnosis, it was also associated with a wide range of etiologies, such as schizophrenia, mood disorders, general medical conditions (GMC), substance withdrawal. Catatonia occurs in all kind of illnesses ranging from psychotic spectrum to mood disorders, mental retardation, and organic brain syndrome suggestive of catatonia being an illness of multifactorial etiology.<sup>[3,4]</sup>

Traditionally catatonia has only been associated with schizophrenia as was diagnosed and mentioned in ICD 10 and DSM IV. However, with advanced understanding regarding the etiopathogenesis, it has been now mentioned that it can occur in number of psychiatric, neurological as well as medical conditions, as a result, in DSM-5 it has become possible to classify catatonia as 'catatonia as specifier' or 'catatonic disorder not otherwise specified' or, 'unspecified catatonia'.<sup>[5]</sup> Also, ICD 11 mentions catatonia as an independent identity.

In adolescents, catatonia is associated with high morbidity and mortality. Catatonia is still considered clinically rare in children and adolescents probably due to varied presentations, etiology and diagnostic issues, often leading to underdiagnosis and under treatment.<sup>[6,7]</sup> Although pediatric catatonia can occur in the setting of psychiatric, neurologic, autoimmune, metabolic and toxic etiologies, it is often either confused with other conditions or not considered at all.<sup>[8]</sup>

Considering the variety of possible etiological diagnosis in adolescents presenting with catatonia and the importance of prompt recognition of correct diagnosis, we present three cases to illustrate the presentation, diagnostic evaluation, varied etiology and management of catatonia in this population. In each case described below, the patients' confidentiality has been maintained.

#### **Case 1**

A 16 years old boy presented with 7 days history of decreased food intake, not speaking, decreased interaction and sitting abnormally still in odd postures for hours. The history revealed an episode of jerky movements of hands, feet and whole body, clenching of teeth, frothing from mouth with passing of urine and followed by unconsciousness 7 days back. The patient was admitted in psychiatry department for diagnostic work up and treatment. Detailed history revealed a past history of similar symptoms 2 years back when he was admitted to hospital following an episode of loss of consciousness and mutism for 2 weeks and records showed treatment with valproate 1000mg and lorazepam 4mg. He discontinued all the medications after a period of 2 months of being asymptomatic. Examination revealed young aged boy with symptoms of waxy flexibility, echolalia, echopraxia, grimacing, mutism, negativism, staring and posturing. His routine investigations along with CT Head and fundus examination was normal his EEG was also done and as reportedly normal. Patient was started on sodium valproate 1000mg and lorazepam 4mg gradually increased to 14 mg per day over a period of 3 days. Subsequently the patient showed significant improvement. He was subsequently discharged on Sodium valproate 1000mg per day and lorazepam 2 mg per day.

#### **Case 2**

A 14 years old girl was referred from pediatrics OPD for psychiatric consultation. The girl presented with a history of 2 days of not speaking, not eating food, repeating others actions, staring for long period of time and standing at one place for long period of time. There was no other significant history available. The symptoms had an acute onset without any apparent precipitating factor. On examination, the patient had squint (present since birth). Detailed examination revealed catatonic signs and symptoms in the form of mutism, staring, posturing, echopraxia, posturing, psychological pillow, forced grasping, mitgahen, automatic obedience, withdrawal, grimacing and stereotypies.

The patient was admitted and lorazepam challenge test was done. After 4 mg of IV lorazepam, there was marked improvement in catatonic symptoms. However, the symptoms reappeared after about 2 hours. Her routine investigations including work up for organic causes was done and was negative. The patient was started on lorazepam 8 mg per day along with IV fluids. Lorazepam was increased gradually up to 24 mg per day gradually over a period of 6 days. To our surprise this was the maximum dose found in literature and hence given to the patient. The family members were counselled and to our surprise, no sedation was reported from this dose. After about 2 days of 24 mg lorazepam per day, the patient started showing improvement and her symptoms resolved completely after 2 days. The dose of lorazepam was gradually decreased and patient was discharged after a period of 1 week of being symptom free on 8 mg of lorazepam. No additional symptoms were reported by the patient and no overt psychopathology was elicited after resolution of her catatonic symptoms.

#### **Case 3**

A 13 years old boy was brought to psychiatry OPD with history of 3 days of not eating food and not talking at all despite frequent coaxing. Detailed history from parents reported history of some abnormal behavior for the past 2 months but even on further coaxing, they were not willing to reveal much details. However, they reported patient to be complaining of easy tiredness, decreased appetite, mild abdominal pain and fever for the last 5 days prior to onset of these symptoms. On examination, the patient was febrile, he had catatonic symptoms in the form of mutism, negativism, grimacing, waxy flexibility, posturing, staring, echolalia, withdrawal. The patient was admitted and investigated thoroughly. Abnormal findings in his investigations revealed Total Leucocyte count to be 3300, Serum Widal titers being positive and blood culture positive for salmonella Typhi. No other abnormal findings were recorded. His MRI Brain and EEG were normal. He was started on inj lorazepam 8 mg per day along with Inj ceftriaxone 1g twice daily along with Inj PCM on as as and when required basis. The patient did not respond much in his catatonic symptoms and hence his lorazepam dose was increased to 16 mg per day by 4th day. By fifth day, he was afebrile and his repeat counts were 6500. His injection ceftriaxone was stopped on 7th day. Subsequently, his catatonic symptoms also responded and by 8th day, he did not have any catatonic signs and symptoms. His lorazepam was tapered over next 2 days and he was started on olanzapine 10 mg per day in view of history of psychotic symptoms. He has been followed up in psychiatry OPD with improvement in psychosis and no emergence of catatonic symptoms.

## DISCUSSION

Here we highlight the diagnosis made in each case and the possible reasons supporting our diagnosis.

### Case 1

We diagnosed this case as a case of post ictal recurrent catatonia. Catatonia may have a complex relationship with seizures and epilepsy; it can precede, occur during, or continue after ictus. Carefully noting the history from the patient and collateral sources is absolutely crucial to establish the chronology of catatonic symptoms in relation to first onset of seizures, seizure frequency, depth of seizure control, change in ictal semiology and treatment history. Looking at the symptoms and history with support from EEG diagnosis of seizure was certain. Other differentials leading to this were considered postictal akinesia, postictal delirium, hypoactive delirium, nonconvulsive status epilepticus, postictal psychosis and non-psychiatric stupor (characterized by immobility, mutism, absence of response to stimuli; this kind of stupor has other causes than catatonia, such as head trauma, anoxia, epilepsy and encephalopathy of unknown origin).<sup>[9]</sup>

Another important distinction that must be made in this case is between postictal delirium (occurring in 35% of seizure cases and reported to last sometimes up to 10 days) and postictal catatonia as it may substantially influence treatment decision-making. Whereas benzodiazepines are used first in cases of catatonia, in delirium such compounds may aggravate symptoms. In our case a diagnosis of catatonia was considered most plausible based on symptomatology (e.g. lack of day-night differences in symptomatology), absence of adverse effects after the lorazepam 'challenge test' and repetitive EEGs showing no signs of epileptic activity.

### Case 2:

This patient was diagnosed as a case of catatonia with no apparent etiology possibly fitting into idiopathic catatonia.<sup>[10]</sup>

There exists a specific variety of catatonic syndrome that has been described previously by various authors. One should be aware that this is a separate subcategory of catatonia. It is also possible that such a variety of catatonia remains underdiagnosed. Though majority of patients with catatonia have an identifiable underlying cause (e.g., an underlying medical or psychiatric disorder), there are cases, like our case 2, where no such cause can yet be identified. There is a need to understand the possible mechanisms responsible for the catatonic symptoms in such patients. In the pediatric population, it is important for the family members to know that other symptoms might emerge after some time or may not come at all, and families must be psychoeducated about the possibility for a psychiatric disorder to emerge in the future. An interesting point in this patient that we wish to highlight is that she responded to very high doses 24

mg of lorazepam, which is probably the maximum dose recommended for the management of catatonia.<sup>[11]</sup>

### Case 3:

The patient was diagnosed to be having catatonia due to organic infective condition (typhoid) along with psychosis NOS. though the patient had history of psychosis prior to onset of catatonia, but the catatonic symptoms developed after the development of fever and investigations revealed typhoid fever. This is of utmost importance has patients with psychiatric history or diagnosis are considered as being functional and organic conditions are often not looked into and addressed.<sup>[12]</sup> Although neuro-psychiatric manifestations are reported in nearly 50–75% of patients at any phase of enteric fever, the chance of their misdiagnosis and deferred diagnosis of the prime illness is quite common. Atypical symptoms are commonly attributed to be a part of 'typhoid toxemia', the acute febrile phase of the illness. Multi-factorial causation is highly likely including metabolic derangements, toxins and auto-immune phenomenon, though susceptible personality traits have been a proven risk factor.<sup>[13]</sup> The presence of abnormal behavior prior to the onset of typhoid fever and catatonia could be a very important factor to predispose the patient to develop catatonia associated with typhoid fever in our case. Although the prognosis is usually favorable, prompt identification of any neuro-psychiatric symptoms in enteric fever needs to be taken seriously and managed appropriately. Numerous isolated case reports and series have reported from mild confusion to typhoid encephalopathy, although isolated catatonia syndrome is a rare association.<sup>[14-16]</sup>

## CONCLUSION

The timely relief of catatonia would allow clinicians to communicate with the patients and begin treating the associated etiologies, if present. One needs to look into catatonia as an independent condition with multi factorial causation and proper identification of the underlying cause is important for the correct immediate and especially long-term management of these patients. Given its prevalence and treatability, pediatric catatonia ought to be regularly considered by all clinicians whose clinical duties may include the care of children and adolescents. Psychiatrists working in a consultation-liaison setting may also be asked by their colleagues to guide the diagnosis and treatment of pediatric catatonia, and should thus be comfortable with the unique considerations of this disease as well as its overlap with catatonia in adults.

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