

CLINICAL CASE REPORTS

Catatonia With Mood and Psychotic Features in an Adolescent With Intellectual Disability and Epilepsy

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ABSTRACT

We present the case of a 15-year-old girl with moderate intellectual disability and epilepsy secondary to mesial temporal sclerosis who developed catatonia during a severe mood episode with psychotic features.

Her clinical course was marked by sudden behavioural regression, mutism, motor rigidity and insomnia, requiring inpatient psychiatric admission. After partial remission, she was discharged but relapsed two weeks later with worsening catatonia, urinary incontinence, gait disturbance and behavioural disorganisation, prompting further investigation and readmission.

The overlap between neurological, developmental and psychiatric symptoms posed diagnostic challenges, especially in distinguishing catatonia from seizure-related or medication-induced phenomena. Treatment included benzodiazepines, antipsychotic adjustments and mood stabilisers, with close monitoring of neurological comorbidities. Clinical improvement followed the introduction of lorazepam and replacement of risperidone with aripiprazole.

This case illustrates the complexity of catatonic psychosis in adolescents with neurodevelopmental and epileptic comorbidities, highlighting the importance of early recognition, tailored pharmacological intervention and multidisciplinary collaboration.

Keywords: adolescent; catatonia; epilepsy; intellectual disability; mood disorders; psychotic disorders

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INTRODUCTION

Catatonia is a neuropsychiatric syndrome characterised by abnormalities in movement, behaviour, affect, and volition.⁽¹⁾ Though traditionally associated with schizophrenia, catatonia is increasingly recognised across a range of psychiatric and medical conditions, particularly in neurodevelopmental contexts such as autism spectrum disorder (ASD) and intellectual disability (ID), as well as in epilepsy.⁽¹⁻³⁾ In paediatric populations, the clinical presentation is often atypical, leading to under-recognition and delayed treatment.⁽³⁾

Mesial temporal lobe epilepsy (MTLE) carries well-documented psychiatric comorbidities, including mood disorders, psychosis, and cognitive dysfunction.⁽⁴⁾ The coexistence of epilepsy and ID further complicates the clinical picture, as behavioural changes may be misattributed to developmental baseline or medication side effects.⁽⁵⁻⁷⁾ In such cases, differentiating between seizure-related states (e.g., non-convulsive status epilepticus), psychiatric decompensation, and catatonia requires careful multidisciplinary evaluation.^(5,6)

We describe a diagnostically complex case of an adolescent girl with ID and MTLE who developed catatonia in the context of a severe mood episode with psychotic features. This case highlights the intricate overlap of neurological, developmental, and psychiatric symptomatology and the need for individualised, interdisciplinary management.

CASE PRESENTATION

The patient is a 15-year-old girl with moderate intellectual disability and epilepsy secondary to mesial temporal sclerosis (MTS), diagnosed at 14 months following febrile seizures and confirmed by MRI. Seizure control was achieved by age five with sodium valproate (300 mg BID), although previous withdrawal attempts led to relapse. Her developmental history included global developmental delay, limited autonomy in daily living, impaired executive function, and social cognition deficits. She had been followed by Child and Adolescent Psychiatry and Psychology services since age four, and formal developmental and cognitive assessments supported a diagnosis of global developmental delay later revised to moderate intellectual disability and ADHD, without a documented autism spectrum disorder diagnosis. She had longstanding behavioural difficulties marked with low frustration tolerance and excessively rigid behaviours. The family described high levels of daily caregiving burden, with limited structured behavioural strategies at home.

She attended special education with curricular adaptations and minimal extracurricular engagement. Her family history was notable for schizophrenia in her biological father. At baseline, her regular medication included sodium valproate and quetiapine XR 150 mg at night for behavioural regulation.

The acute episode began shortly after menarche (age 14), coinciding with marked emotional lability and a sudden

decline in functioning. She developed progressive mutism, insomnia, food refusal, hypervigilance, and intense fear when outdoors, suggestive of emerging psychotic symptoms with paranoid ideation. At home, she insisted on closing all windows and doors. A psychiatric emergency visit included brain CT, bloodwork and toxicology, all of which were normal. Olanzapine (2.5 mg/day) was initiated with partial improvement in agitation but persistent mutism. After one month, her caregiver discontinued olanzapine without medical advice, leading to a rapid deterioration: the patient became mute, compulsively undressed, displayed soliloquies and unprovoked laughter, and refused all food and medication. Insomnia became nearly total.

She was admitted to Child and Adolescent Psychiatry (CAP) inpatient unit under suspicion of a first psychotic episode. On examination, she was mute, perplexed, occasionally posturing, and exhibited bizarre behaviour, including undressing and responding to internal stimuli. Risperidone was titrated to 2 mg BID, but side effects emerged—daytime somnolence and tremor—prompting dose adjustment to 0.5 mg AM and 3 mg PM. Over four weeks, she partially improved, regaining autonomy in feeding and hygiene. She began to speak in simple phrases and displayed hyperfamiliar behaviour with staff, often seeking physical contact and showing a childlike demeanour. An EEG showed slowed background rhythm and focal slowing in the right occipital region, without epileptiform activity. MRI was attempted but not completed due to poor cooperation.

She was discharged on risperidone and valproate, but within two weeks experienced a relapse. She became mute again, with urinary incontinence, motor rigidity, and an orthotic gait. Neurology admission included repeat CT, lumbar puncture, metabolic and autoimmune screening (all normal), and a new MRI revealing left hippocampal atrophy consistent with MTS. Risperidone was tapered, and eslicarbazepine was initiated due to concerns about valproate-induced extrapyramidal symptoms.

Transferred again to CAP, she presented with full-blown catatonia: negativism, mutism, posturing, echolalia, rigidity, agitation, and persistent insomnia. Lorazepam was titrated to 2.5 mg TID with clear motor and behavioural improvement. Risperidone was replaced by aripiprazole (10 mg AM + 5 mg PM), aiming to minimise extrapyramidal burden. Quetiapine XR (50 mg) was added at night to target residual agitation and sleep disturbance.

As catatonic symptoms resolved, she developed transient maniform features, including elated humour with inappropriate, unprovoked laughter, affective lability, and persistent hyperfamiliarity. A formal assessment of manic or hypomanic criteria was limited by restricted verbal output and baseline intellectual disability; symptoms were therefore monitored clinically and gradually attenuated without the need for further medication changes beyond the regimen already established. Verbal expression improved, although content remained concrete, and she regained full autonomy

in self-care. At discharge, affect was described as euthymic, with stabilised sleep and appetite, and outpatient psychiatric follow-up was arranged for ongoing monitoring and treatment optimisation. Given her developmental vulnerability, she was referred for gynaecological evaluation and contraceptive

Table 1 - Clinical timeline of clinical presentation, impressions and medical interventions

Date	Symptoms / Clinical Presentation	Clinical Impressions	Interventions
April 2024	Sudden behavioural disorganisation, mutism, hypervigilance, food refusal, insomnia	Acute psychiatric decompensation post-menarche	Psychiatric ER visit; normal CT, bloods and toxicology; olanzapine 2.5 mg/day started
May 2024	Partial behavioural improvement; mutism persists	Deterioration post-medication withdrawal	Olanzapine was discontinued without medical supervision (by the caregiver)
June–July 2024	Compulsive undressing, soliloquies, aimless wandering, refusal to eat/medicate, near-total insomnia	Suspected psychosis with catatonic features	Psychiatric ER visit; admitted to inpatient Child and Adolescent Psychiatry (CAP)
July 2024	Mutism, agitation, bizarre behaviour, soliloquies, posturing, hallucinations	Psychotic episode; Catatonia (partial)	Risperidone up to 2 mg BID → adjusted to 0.5 mg AM + 3 mg PM; EEG (slowed background); MRI attempted but not cooperative
Late July 2024	Improved functioning; regained self-care; hyperfamiliar behaviour; mild dystonia	Partial remission; suspected extrapyramidal symptoms	Risperidone maintained; neurology referral
August 2024	Relapse: mutism, rigidity, urinary incontinence, robotic gait	Suspected medication-induced Parkinsonism vs Catatonia	Neurology admission; CT + LP normal; MRI: left hippocampal atrophy + MTS; Risperidone tapered; switch to eslicarbazepine
Mid-August 2024	Mutism, negativism, motor slowing, posturing, agitation, insomnia, echolalia	Catatonia; affective instability; suspected psychosis	Re-admission to CAP; lorazepam titrated to 2.5 mg TID; risperidone tapered; switched to aripiprazole
Late August 2024	Resolution of catatonia; hypervigilance, hyperfamiliarity, unprovoked laughter	Residual psychosis; emotional dysregulation in ID context	Quetiapine XR 50 mg added; aripiprazole 10 mg AM + 5 mg PM; eslicarbazepine 800 mg; lorazepam maintained
September 2024	Functional improvement; verbal communication resumed; self-care regained	Partial recovery; residual symptoms	Discharged with full medication plan; psychiatric follow-up + gynaecology referral for contraceptive planning

This table presents a chronological overview of the patient's clinical course, including the onset and progression of symptoms, inpatient and outpatient evaluations, and therapeutic interventions. It summarises key events from initial psychiatric and neurological assessments to pharmacological adjustments and response to treatment. Abbreviations used in the table include: CAP (Child and Adolescent Psychiatry), CT (Computed Tomography), EEG (Electroencephalography), ER (Emergency Room), ID (Intellectual Disability), LP (Lumbar Puncture), MTS (Mesial Temporal Sclerosis), MRI (Magnetic Resonance Imaging), XR (Extended Release), BID (twice daily), TID (three times daily), and AM/PM (morning/evening).

DISCUSSION

This case illustrates the intersection of psychiatric, neurological and developmental domains in adolescent-onset catatonia. The acute behavioural decompensation in the context of ID and epilepsy poses major diagnostic challenges. Catatonia often mimics seizure-related phenomena, psychosis, or behavioural dysregulation in ID.⁽⁷⁾

The symptom cluster of mutism, rigidity, posturing, and autonomic instability was initially misattributed to side effects or developmental regression. Only after the re-emergence of symptoms and neurological exclusion was catatonia correctly identified. This delay is common in paediatric populations, particularly with coexisting ID.^(3,5) The Bush-Francis Catatonia Rating Scale, used for systematic assessment of catatonic signs, is helpful but not widely validated in youth.⁽⁸⁾

Lorazepam remains the first-line treatment for catatonia, often requiring high doses titrated to clinical response and frequently leading to rapid reversal of motor symptoms.⁽²⁾ In our case, the response was evident within days of titration. Antipsychotic management was more complex. Risperidone led to motor worsening; aripiprazole, with its partial dopamine agonist profile, was better tolerated and effective in stabilising mood and psychosis.⁽⁹⁾

The switch from valproate to eslicarbazepine was based on concerns about valproate-induced parkinsonism, a known but under-recognised complication.⁽¹⁰⁾ Eslicarbazepine offered similar seizure control with less sedation and cognitive impact.⁽¹¹⁾ The patient's functional recovery followed this change, though causality is speculative.

Importantly, this episode coincided with menarche, raising the possibility of hormonal modulation of seizure threshold and affective stability.⁽¹²⁾ Literature shows that oestrogen-progesterone fluctuations can exacerbate psychiatric symptoms in epilepsy.^(4,12) This temporal link, alongside the emergence of mood lability and affective symptoms, suggests an underlying bipolar diathesis superimposed on neurodevelopmental vulnerability.

Behaviourally, the progression from catatonia to impulsivity, inappropriate laughter, and hyperfamiliarity mirrors findings in ASD and ID populations, where catatonia may precede or alternate with manifold symptoms.⁽⁶⁾ Environmental triggers, sensory overload, and communication barriers are often implicated.^(5,6)

Beyond pharmacology, rehabilitation focused on restoring routines, self-care, and engagement. The role of structured environments, occupational therapy, and behavioural consistency is critical to recovery.⁽⁷⁾

Finally, the case emphasises safeguarding in adolescents with ID. Poor risk awareness, impaired social cognition and hyperfamiliarity significantly increase the risk of sexual exploitation.⁽⁵⁾ The proactive referral to gynaecology was ethically appropriate and aligned with best clinical practice.

CONCLUSIONS

This case exemplifies the diagnostic and therapeutic complexity of catatonia in adolescents with epilepsy and intellectual disability. Early identification requires high clinical suspicion, careful exclusion of organic pathology, and awareness of catatonia's atypical presentations in neurodevelopmental contexts. Treatment success depended on benzodiazepine titration, antipsychotic rationalisation, mood stabilisation, and structured multidisciplinary care.

The patient's outcome reinforces the need for an integrated psychiatric-neurological approach, close follow-up, and attention to hormonal, environmental, and safeguarding factors. This case adds to the growing body of evidence on catatonia in paediatric neurodevelopmental populations and calls for greater awareness among clinicians.

AUTHORSHIP

Tiago Aguiar Soares - Conceptualization; Investigation; Data Curation; Writing – Original Draft; Writing – Review & Editing; Project Administration

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Ana Rita Rodrigues - Validation; Methodology; Writing – Review & Editing

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REFERENCES

1. Wilcox JA, Reid Duffy P. The syndrome of catatonia. *Behav Sci (Basel)* 2015; 5(4): 576–88. <https://doi.org/10.3390/bs5040576>.
2. Sienaert P, Dhossche DM, Vancampfort D, De Hert M, Gazdag G. A clinical review of the treatment of catatonia. *Front Psychiatry* 2014; 5: 181. <https://doi.org/10.3389/fpsyt.2014.00181>.
3. Consoli A, Raffin M, Laurent C, *et al.* Medical and developmental risk factors of catatonia in children and adolescents: a prospective case-control study. *Schizophr Res* 2012; 137(1–3): 151–58. <https://doi.org/10.1016/j.schres.2012.02.012>.
4. Kanner AM. Psychiatric comorbidities in epilepsy: should they be considered in the classification of epileptic disorders? *Epilepsy Behav* 2016; 64: 306–8. <https://doi.org/10.1016/j.yebeh.2016.06.040>.
5. Emerson E. Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *J Intellect Disabil Res* 2003; 47(Pt 1): 51–8. <https://doi.org/10.1046/j.1365-2788.2003.00464.x>.
6. Moore S, Amatya DN, Chu MM, Besterman AD. Catatonia in autism and other neurodevelopmental disabilities: a state-of-the-art review. *NPJ Ment Health Res* 2022;1: 12.

- <https://doi.org/10.1038/s44184-022-00012-9>.
7. Helmstaedter C, Witt JA. Epilepsy and cognition – a bidirectional relationship? *Seizure* 2017; 49: 83–9. <https://doi.org/10.1016/j.seizure.2017.02.017>.
 8. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardized examination. *Acta Psychiatr Scand* 1996; 93(2): 129–36. <https://doi.org/10.1111/j.1600-0447.1996.tb09814.x>.
 9. Kanner AM, Rivas-Grajales AM. Psychosis of epilepsy: a multifaceted neuropsychiatric disorder. *CNS Spectr* 2016; 21(3): 247–57. <https://doi.org/10.1017/S1092852916000250>.
 10. Brugger F, Bhatia KP, Besag FM. Valproate-associated parkinsonism: a critical review of the literature. *CNS Drugs* 2016; 30(6): 527–40. <https://doi.org/10.1007/s40263-016-0341-8>.
 11. Zhu LN, Chen D, Tan G, *et al*. The tolerability and safety profile of eslicarbazepine acetate in neurological disorders. *J Neurol Sci* 2020; 413: 116772. <https://doi.org/10.1016/j.jns.2020.116772>.
 12. Pennell PB. Hormonal aspects of epilepsy. *Neurol Clin* 2009; 27(4): 941–65. <https://doi.org/10.1016/j.ncl.2009.08.005>.

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