Periodic Catatonia in an Adult With Autism Spectrum Disorder Responding to Lithium: A Case Report

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Catatonia is a complex neuropsychiatric syndrome that may occur across a spectrum of psychiatric and medical conditions.[1] Periodic catatonia (PC), a rare and underrecognized subtype, is characterized by recurring episodes of catatonia interspersed with periods of normal functioning.[2] It is commonly reported in association with mood disorders, particularly bipolar disorder, which may account for its cyclical nature. We report a rare case of PC in an adult with autism spectrum disorder (ASD), in the absence of any other identifiable psychiatric or neurological comorbidities, that responded to lithium prophylaxis.

Case Report

Mr S, a 30-year-old man, presented to the psychiatry outpatient department with a 2-year history of 15- to 28-day–long episodes marked by abrupt-onset mutism, posturing, staring, repetitive hand rubbing, reduced food intake, and rigidity. These symptoms fluctuated in intensity throughout the day, and during more severe phases, he even experienced bowel and bladder incontinence. Food intake and sleep were markedly reduced during these episodes, causing significant distress. Each episode was followed by a period of complete spontaneous symptomatic recovery lasting 3–4 weeks. Over 2 years, he experienced 10–12 episodes despite receiving multiple psychotropics for adequate durations (Table 1).

At the current presentation, his Bush-Francis Catatonia Rating Scale (BFCRS)[3] score was 21, which decreased to 11 after an intravenous 2-mg lorazepam challenge. His previous medications were discontinued, and he was maintained on lorazepam (up to 12–16 mg/day). Despite treatment, symptoms persisted for 2 weeks with BFCRS scores fluctuating between 10 and 20 before spontaneous remission, similar to previous episodes. Electroconvulsive therapy was not considered given the periodic nature and the consistent pattern of

Table 1.
Treatment Course[a]

Timeline	Medications/interventions	Clinical course and outcome
February 2023–June 2023	Tablet lorazepam up to 8 mg/d (only during the acute episodes) Tablet olanzapine up to 15 mg/d	Mild symptomatic improvement during episodes, but catatonic episodes recurred every 4 wk.
July 2023–October 2023	Tablet lorazepam up to 10 mg/d (only during the acute episodes) Tablet aripiprazole up to 20 mg/d	No significant change in periodicity or duration of episodes. Episodes recurred every 3–4 wk despite taking the medication regularly.
November 2023–February 2024	Tablet lorazepam up to 6–8 mg/d (started during acute episodes and continued even during interepisodic period) Tablet venlafaxine up to 150 mg/d	No prevention of recurrence; episodes continued at similar frequency.
March 2024–May 2024	Tablet lorazepam up to 6 mg/d (only during episodes) Tablet risperidone up to 4 mg/d (given for only 3 wk and then discontinued due to extrapyramidal symptoms) Tablet fluoxetine up to 60 mg/d	Minimal benefit during the episodes; episodes persisted.
June 2024–September 2024	Tablet fluoxetine 60 mg/d Tablet duloxetine up to 80 mg/d	No sustained improvement; episodes recurred at the same frequency.
October 2024–December 2024	Tablet lorazepam 4 mg/d (continuous) Tablet clozapine up to 50 mg/d	No clinical response; recurrent episodes persisted.
January 2025 (most recent episode)	Injection lorazepam (up to 12–16 mg/d)	First contact with the patient (current presentation). Treatment and assessments done as inpatient. Partial and transient improvement; symptoms persisted for ~3 wk followed by spontaneous remission.
Postremission February to September 2025	Tablet lithium 600 mg/d	No recurrence for 8 mo of follow-up; periodic pattern halted.

[a]Dates have been changed to protect patient anonymity.

spontaneous recovery observed in prior episodes. There was no history of psychotic or mood symptoms in any of the episodes including the current episode. Detailed developmental history revealed delayed language milestones, poor social reciprocity, restricted interests, emotional detachment, and an excessive need for routine—features suggestive of ASD. Diagnosis was confirmed using the Indian Scale for Assessment of Autism.[4] Neurological workup, including neuroimaging and autoimmune profile, was unremarkable.

A diagnosis of catatonia associated with ASD (*ICD-11* codes: 6A40 and 6A02) was considered most appropriate, although a definitive cause for the periodicity could not be determined. Given the cyclicity and lack of response to prior treatments, lithium was initiated prophylactically at 600 mg/day following spontaneous remission of the most recent episode. He was maintained on lithium monotherapy with serum levels of 0.47 mmol/L. At 8-month follow-up, the patient remained free of catatonic episodes and functionally stable.

Discussion

PC was first described by Kraepelin (1908) in schizophrenia and later investigated by Gjessing (1938), who proposed nitrogen balance changes as a possible contributor.[5] Since then, literature on PC has remained limited to a few case reports, primarily in the context of mood and psychotic disorders.[5–7] It has also been reported in a few cases of frontal lobe epilepsy[8] and encephalitis.[9] A review of the literature for the treatment of PC revealed only 5 case reports supporting lithium's efficacy in managing PC prophylactically,[10–14] including one with a 9-year symptom-free follow-up indicating its long-term efficacy.[10] Evidence for other agents remains even more limited, with isolated reports involving antipsychotics like olanzapine[15] and risperidone,[16] both ineffective in our patient, as well as mirtazapine[17] and lamotrigine.[18] While catatonia is well recognized in ASD,[19] to the best of our knowledge, PC in this population has not been previously reported. The British Association for Psychopharmacology guidelines to manage catatonia (2023) acknowledged PC as a distinct subtype and recommended lithium for prophylaxis based on a few case reports.[2] Our case supports this recommendation, demonstrating that lithium, even at low serum levels, can be effective and well tolerated in preventing recurrence of PC at least in the short term, even in the presence of a neurodevelopmental condition like ASD. Early recognition of such catatonic patterns in primary care can prevent unnecessary medication trials and facilitate timely interventions.

Article Information

Published Online: February 3, 2026. https://doi.org/10.4088/PCC.25cr04066

Prim Care Companion CNS Disord 2026;28(1):25cr04066

Submitted: August 21, 2025; accepted October 30, 2025.

To Cite: Bakam A, Raina S, Reddy YCJ. Periodic catatonia in an adult with autism spectrum disorder responding to lithium: A case report. *Prim Care Companion CNS Disord*. 2026;28(1):25cr04066.

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Financial Disclosure: None.

Funding/Support: None.

Patient Consent: Consent was received from the patient and family to publish this case report, and information, including dates, has been de-identified to protect patient anonymity.

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