

# Acute-Onset Bipolar Psychosis in an Adolescent Without Prodromal Symptoms: A Case Report

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## Abstract

Early-onset bipolar disorder often begins in adolescence and carries greater severity, comorbidity, and long-term impairment. Diagnosis is complicated by symptom overlap with normative developmental behavior, nonspecific prodromes, and conditions that can cause high-risk or impulsive behaviors, such as attention deficit/hyperactivity disorder (ADHD). This report examines the case of an adolescent with the acute onset of bipolar disorder and psychosis without an identifiable prodrome, after stimulant-treated ADHD, with emphasis on evaluation, treatment, and outcome. A 15-year-old male child with a history of anxiety and ADHD since childhood (well-controlled on dexamethylphenidate hydrochloride extended-release (XR)) presented after two weeks of abrupt restlessness, racing thoughts, decreased sleep, pacing, pressured speech, disorganization, echolalia, episodic disrobing/soiling, and paranoid delusions. He was admitted to the inpatient adolescent psychiatry unit for altered mental status with mania and psychosis. Risperidone was initiated and titrated to 1 mg in the morning and 2 mg nightly, with marked clinical improvement in manic and psychotic symptoms within one week, suggesting an original decrease in dosage may have precipitated psychosis. No organic etiology was revealed on magnetic resonance imaging (MRI), lumbar puncture, labs, or encephalitis panels. Given the patient's clinical improvement, he was discharged on risperidone with first-break psychosis clinic follow-up. The patient tolerated long-term dexamethylphenidate hydrochloride use without prior manic symptoms, and discontinuation did not improve his presentation, making stimulant-induced bipolar disorder less likely. An extensive workup, including MRI, cerebrospinal fluid (CSF), and infectious/autoimmune panels, ruled out encephalitis. The case documents an abrupt-onset severe bipolar episode with psychosis in the absence of a prodrome; the temporal risperidone reduction may have precipitated decompensation. This case highlights the need for thorough multidisciplinary evaluation to exclude medical etiology, structured medication-transition protocols, and close outpatient follow-up.

**Categories:** Psychiatry, Pediatrics

**Keywords:** bipolar disorders, child and adolescent mental health, child and adolescent psychiatry, first episode psychosis, lyne's disease

## Introduction

Early-onset bipolar disorder (BD), defined as onset before age 18, is associated with substantial psychiatric comorbidity and long-term functional impairment [1], in contrast to a typical peak age of onset of approximately 25 years [2]. Psychotic symptoms are particularly associated with bipolar I disorder and frequently occur during manic episodes or episodes with mixed features [3]. In pediatric BD, reported rates of psychotic features vary widely, ranging from 16% to 87.5% [3].

Diagnosis during adolescence can be challenging because symptoms may overlap with normative developmental behaviors and other psychiatric conditions, particularly attention-deficit/hyperactivity disorder (ADHD). ADHD is a common comorbidity in pediatric bipolar I disorder, with a lifetime prevalence of 61.2% reported among affected young children and adolescents [4]. Because ADHD frequently co-occurs with and often precedes pediatric BD, adolescents who develop BD may have a long history of stimulant treatment. Whether stimulant exposure influences the onset or course of BD remains uncertain. Retrospective data suggest an association between prior stimulant treatment and earlier age at BD onset, but these data cannot distinguish the effect of stimulants from an underlying shared vulnerability [5].

New-onset psychosis or mania in a pediatric patient also warrants evaluation for medical etiologies. Autoimmune encephalitis, particularly anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis, can present with prominent psychiatric symptoms in young people [6], and infectious causes such as Lyme neuroborreliosis have been reported to present as first-episode psychosis [7-9].

After acute symptoms improve, antipsychotic doses are often reduced because of adverse effects such as sedation, which is common with risperidone in children and young people [10]. In first-episode psychosis, antipsychotic dose reduction and discontinuation are associated with an increased risk of relapse [11], but evidence to guide dose reduction in adolescents recovering from acute mania with psychosis is limited.

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Prodromal features of BD are often nonspecific, and their nature and predictive value remain incompletely characterized [12]. Although emerging evidence suggests that many pediatric patients with bipolar I disorder experience a prodromal period of subthreshold mood symptoms and behavioral and functional changes before the first manic episode [13], less is known about adolescents who present abruptly without one, particularly after long-term stimulant treatment and recent antipsychotic dose changes. This case describes an adolescent with long-standing, stimulant-treated ADHD who developed abrupt-onset bipolar I disorder with psychotic features without an identifiable prodrome, with decompensation following a reduction in risperidone dose. We detail the extensive evaluation for organic etiologies, the treatment course, and implications for managing medication transitions in adolescents.

## Case Presentation

A 15-year-old White male patient with a history of ADHD (diagnosed at six years of age due to inattention; well controlled on dexamethylphenidate hydrochloride XR 10mg once a day), anxiety, and prior brief mood disturbance presented with marked behavioral disorganization characterized by recurrent disrobing, fecal smearing, echolalia, repetitive pacing, mania, and psychosis. Prior to the current and preceding psychiatric admissions, the patient's only documented psychiatric diagnosis was ADHD, which had been diagnosed primarily on the basis of inattentive symptoms.

At baseline, the patient was described as high functioning and academically successful, enrolled in accelerated classes, and actively involved in multiple sports. His medical history was notable for pericarditis diagnosed at nine years of age, which was in remission and routinely monitored by a cardiologist. Cardiac magnetic resonance imaging (MRI) and additional cardiac testing performed approximately one month prior to his first psychiatric admission were reportedly unremarkable. There was no previously documented history of other psychiatric disorders, developmental delays, substance use, or significant familial psychiatric illness. The patient also had positive relationships with both parents and his two siblings.

One month prior to the current admission, the patient underwent a three-week psychiatric hospitalization at a neighboring institution for new-onset confusion and decreased need for sleep. During that hospitalization, dexamethylphenidate extended-release (Focalin XR) 10 mg daily was discontinued due to concerns of it contributing to current insomnia and behaviors. The patient had been maintained on the same dose for approximately nine years without previously reported adverse effects, including severe insomnia, marked hyperactivity, or agitation. He was weaned off his stimulant for ADHD over the first week of admission. Risperidone was initiated at 1 mg daily and titrated to 1 mg twice daily during the hospitalization. This was associated with transient improvement in sleep, confusion, and social reciprocity. Given this clinical improvement, his symptoms were considered responsive to stimulant discontinuation and initiation of antipsychotic treatment; however, a definitive psychiatric diagnosis was not established at the time of discharge.

Approximately two weeks after discharge, risperidone was reduced by an outpatient provider because of his experienced sedation and inability to focus in school. Immediately following this titration, the patient experienced decreased need for sleep and was started on guanfacine 1 mg at night. Over the following week, the patient experienced recurrence of symptoms similar to those preceding his prior hospitalization, characterized primarily by restlessness, racing thoughts, decreased need for sleep, pacing, pressured speech, disorganization, echolalia, and episodic disrobing, prompting emergency room presentation and current psychiatric admission. Upon admission, the patient was restarted on his previous regimen of risperidone 1 mg twice daily. He demonstrated minimal response to this dosage and subsequently developed new behavioral symptoms, including fecal soiling and smearing, as well as repetitive quoting and imitation of specific movie characters.

Initial physical exam and vital signs were normal. Neurology was consulted given the possibility of an encephalopathic presentation. Neurological testing was positive for below-age cognitive functioning, which contrasted with baseline function. Parental report and lack of previous medical or chronic psychiatric issues warranted additional testing. The patient underwent brain MRI (Figure 1) that required diazepam 2 mg and lumbar puncture with involvement of the pediatric sedation team, neurology, pediatric intensive care unit (PICU) team, and general inpatient pediatrics. Imaging was negative.

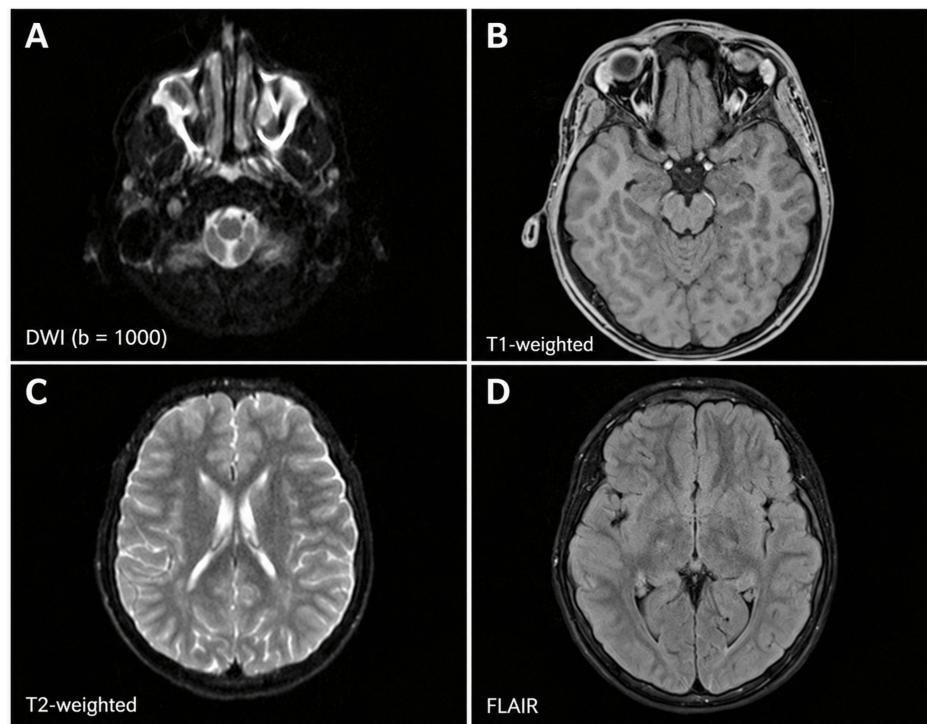


**FIGURE 1: Brain MRI with contrast**

A: Diffusion-weighted imaging (DWI),  $b = 1000 \text{ s/mm}^2$ ; B: T1-weighted axial MRI; C: T2-weighted axial MRI; D: Fluid-attenuated inversion recovery (FLAIR) axial MRI

Routine bloodwork including complete blood count (CBC), comprehensive metabolic panel (CMP), renal and liver panel largely unremarkable. Cerebrospinal fluid (CSF) demonstrated a mildly elevated lymphocyte count (Table 1). Urine drug screening was negative as well. An elevated serum mercury result was determined to be a false positive, confirmed by repeating testing. Screening Lyme serology returned positive; however, review by infectious disease physicians concluded this represented a false positive, as confirmed by repeating testing. Additional encephalitis panels were sent to the Mayo Clinic for further analysis and were negative. Electroencephalogram (EEG) displayed no signs of seizure or abnormal neurological activity.

| CSF Parameter                          | Result                         | Reference Range (if applicable) |
|----------------------------------------|--------------------------------|---------------------------------|
| Glucose (mg/dL)                        | 54                             | 40–70                           |
| Protein (mg/dL)                        | 44                             | 15–45                           |
| Red blood cells (cells/μL)             | 0                              | 0                               |
| White blood cells (cells/μL)           | 5                              | 0–5                             |
| Differential – Lymphocytes (%)         | 96 ↑                           | N/A                             |
| Differential – Monocytes (%)           | 4                              | N/A                             |
| CSF total nucleated cells (cells/μL)   | 46                             | 0–5                             |
| CSF color                              | Colorless                      | Colorless                       |
| CSF clarity                            | Clear                          | Clear                           |
| CSF electrophoresis/interpretation     | No oligoclonal bands detected  | No oligoclonal bands            |
| Immunofixation (CSF)                   | No monoclonal protein detected | No monoclonal protein           |
| N-methyl-D-aspartate receptor antibody | < 1:1                          | < 1:1 (negative)                |
| Tube number                            | Tube 1                         | N/A                             |

TABLE 1: Cerebrospinal fluid (CSF) results

While undergoing the medical workup, the patient was started on risperidone 1 mg nightly, and guanfacine was not restarted, as it may have contributed to sedation that was experienced previously. Due to insufficient response, the regimen was titrated to 1 mg risperidone in the morning and 2 mg nightly. Following titration, the patient showed progressive clinical improvement with reduced echolalia, improved orientation, paranoia of harm from others, and organization, decreased impulsivity, and improved clinical response over approximately one week. After a week of treatment on this risperidone regimen, his mental status largely stabilized, permitting more active participation in unit programming, which included art therapy and psychiatric occupational therapy as depicted in Table 2.

| Hospital Day | Clinical Course                                                                                                            |
|--------------|----------------------------------------------------------------------------------------------------------------------------|
| Day 1        | Admitted with altered mental status, disorientation, impulsivity, and psychosis. Started risperidone; neurology consulted. |
| Day 3        | Risperidone increased to 1 mg AM / 2 mg PM, with gradual improvement.                                                      |
| Day 8        | Brain MRI and lumbar puncture performed; workup largely unremarkable.                                                      |
| Day 11       | Cognitive assessment showed improvement toward baseline.                                                                   |
| Day 12       | Positive Lyme titer determined by infectious disease to be false positive.                                                 |
| Discharge    | Marked clinical improvement.                                                                                               |

TABLE 2: Clinical course timeline

The abrupt severe presentation without antecedent prodrome, in the context of chronic stimulant-treated ADHD, aligns with literature suggesting ADHD may precede BD but that stimulants more likely unmask or accelerate underlying vulnerability [6]. Given that this patient had been weaned off long-term stimulants over a two-week period before his first psychiatric admission without improvement in symptoms, stimulant-induced mania does not explain the symptoms with which he presented. As noted above, there was no medical or neurologic etiology discovered after thorough assessment. The patient was discharged on risperidone, 1 mg in the morning and 2 mg nightly.

Discussion

Retrospective research has examined whether stimulant exposure influences the trajectory of bipolar

disorder. DelBello et al. reported that adolescents hospitalized for BD who had received prior stimulant treatment had an earlier age of BD onset than those without prior exposure, consistent with the possibility that stimulants may unmask or accelerate an underlying bipolar vulnerability [5]. However, this association does not establish a causal relationship. In the present case, the patient had received dexamethylphenidate hydrochloride XR for approximately nine years without previously documented manic or psychotic symptoms. The medication was discontinued during his initial psychiatric hospitalization, but symptoms subsequently recurred while he remained off treatment. This course makes stimulant exposure alone a less compelling explanation for his presentation, although a contributory role cannot be definitively excluded.

The positive initial Lyme serology complicated the differential diagnosis, particularly given reports of neuropsychiatric manifestations associated with Lyme disease, including psychosis [7–9]. However, repeat testing and infectious disease consultation supported a false-positive result. Additional evaluation, including brain MRI, EEG, CSF studies, and infectious and autoimmune encephalitis testing, did not identify an alternative organic etiology. The patient improved without antimicrobial treatment, which was consistent with the conclusion that Lyme disease was unlikely to explain his presentation.

A further notable feature was the recurrence of symptoms following a reduction in risperidone dosage due to sedation and difficulty concentrating in school. Although relapse following antipsychotic discontinuation is well documented in first-episode psychosis, the evidence base remains less developed for adolescents recovering from mania with psychotic features [11]. In this patient, symptom recurrence after dose reduction and subsequent improvement following risperidone re-escalation raise the possibility that reduced medication exposure contributed to clinical decompensation. However, the temporal association does not establish causation, and the natural course of the illness may also have contributed. It is difficult to distinguish between true childhood BD prodrome and ADHD [12]. There has been limited investigation on this, and it warrants more targeted research [13].

As a single case, this report cannot establish a causal relationship between risperidone dose reduction and decompensation. Also, the history preceding admission relied on caregiver reports and outside records, which may have limited detection of prodromal symptoms. Future research should further investigate the relationship between stimulant exposure and the onset of BD in children and adolescents with ADHD, as well as the clinical characteristics of adolescents who develop mania without an identifiable prodrome. Prospective studies are also needed to determine optimal antipsychotic dose-reduction strategies following first-episode psychosis or mania in adolescents, including predictors of relapse and the role of structured monitoring during medication transitions.

## Conclusions

This case documents an abrupt, severe presentation of BD with psychosis in the absence of identifiable prodromal symptoms or an alternative organic etiology despite extensive neurologic and infectious evaluation. The temporal association between recent risperidone dose reduction and rapid clinical decompensation raises the possibility that pharmacologic adjustments contributed to relapse. The case underscores outpatient responsibilities in balancing relapse risk against medication adverse effects, the need for structured and closely supervised titration protocols, close monitoring and documentation of symptom relapse, and targeted psychoeducation for adolescents and caregivers to support informed shared decision-making when considering medication adjustments and treatment options. Psychotropic adjustments may prolong hospitalization, disrupt schooling and social development, and increase caregiver anxiety regarding kids' reintegration into the community. Further research is needed to guide medication transitions in this population.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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## References

1. Cirone C, Secci I, Favole I, et al.: What do we know about the long-term course of early onset bipolar disorder? A review of the current evidence. *Brain Sci.* 2021, 11:341-10.
2. Yatham LN, Kennedy SH, Parikh SV, et al.: Canadian Network for Mood and Anxiety Treatments (CANMAT) and International Society for Bipolar Disorders (ISBD) 2018 guidelines for the management of patients with bipolar disorder. *Bipolar Disord.* 2018, 20:97-170. [10.1111/bdi.12609](https://doi.org/10.1111/bdi.12609)
3. Birmaher B, Axelson D, Strober M, et al.: Clinical course of children and adolescents with bipolar spectrum disorders. *Arch Gen Psychiatry.* 2006, 63:175-83. [10.1001/archpsyc.63.2.175](https://doi.org/10.1001/archpsyc.63.2.175)
4. Tillman R, Geller B, Klages T, Corrigan M, Bolhofner K, Zimmerman B: Psychotic phenomena in 257 young children and adolescents with bipolar I disorder: delusions and hallucinations (benign and pathological). *Bipolar Disord.* 2008, 10:45-55. [10.1111/j.1399-5618.2008.00480.x](https://doi.org/10.1111/j.1399-5618.2008.00480.x)
5. DelBello MP, Soutullo CA, Hendricks W, Niemeier RT, McElroy SL, Strakowski SM: Prior stimulant treatment in adolescents with bipolar disorder: association with age at onset. *Bipolar Disord.* 2001, 3:53-7. [10.1034/j.1399-5618.2001.030201.x](https://doi.org/10.1034/j.1399-5618.2001.030201.x)
6. Cabrera-Maqueda JM, Planagumà J, Guasp M, Dalmau J: Autoimmune neuropsychiatric disorders manifesting with psychosis. *J Clin Invest.* 2025, 135:e196507. [10.1172/JCI196507](https://doi.org/10.1172/JCI196507)
7. Šegždaitė G, Aliukonytė O, Pociūtė K: Neuropsychiatric manifestations of Lyme disease: a literature review of psychiatric and cognitive impacts. *Acta Med Lit.* 2025, 32:6-21. [10.15388/Amed.2025.32.1.17](https://doi.org/10.15388/Amed.2025.32.1.17)
8. Mattingley DW, Koola MM: Association of Lyme disease and schizoaffective disorder, bipolar type: is it inflammation mediated?. *Indian J Psychol Med.* 2015, 37:243-6. [10.4103/0253-7176.155660](https://doi.org/10.4103/0253-7176.155660)
9. Farooq F, Farooqi M, Qadir T, Ferrando SJ: Psychosis triggered by Lyme disease and coronavirus disease 2019 (COVID- 19): a case report. *Ann Case Rep.* 2023, 8:1402. [10.29011/2574-7754.101402](https://doi.org/10.29011/2574-7754.101402)
10. Besag FM, Vasey MJ, Hollis C, Taylor D: Neurological adverse effects of antipsychotic medication in children and young people. *Paediatr Drugs.* 2026, 28:127-57. [10.1007/s40272-025-00730-5](https://doi.org/10.1007/s40272-025-00730-5)
11. Kishi T, Ikuta T, Matsui Y, Inada K, Matsuda Y, Mishima K, Iwata N: Effect of discontinuation v. maintenance of antipsychotic medication on relapse rates in patients with remitted/stable first-episode psychosis: a meta-analysis. *Psychol Med.* 2019, 49:772-9. [10.1017/S0033291718001393](https://doi.org/10.1017/S0033291718001393)
12. Hauser M, Correll CU: The significance of at-risk or prodromal symptoms for bipolar I disorder in children and adolescents. *Can J Psychiatry.* 2013, 58:22-31. [10.1177/070674371305800106](https://doi.org/10.1177/070674371305800106)
13. Luby JL, Navsaria N: Pediatric bipolar disorder: evidence for prodromal states and early markers . *J Child Psychol Psychiatry.* 2010, 51:459-71. [10.1111/j.1469-7610.2010.02210.x](https://doi.org/10.1111/j.1469-7610.2010.02210.x)