

## CASE REPORT

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# Lithium and endocrine dysregulation in decompensated bipolar disorder with psychotic features

Noah Begley, Zuha Tariq, Maggie Yip, Saad Sadaf, Mia Rajan

## ABSTRACT

**Introduction:** Bipolar disorder (BD) is a mood disorder characterized by manic episodes, depressive episodes, mixed states, and psychotic symptoms. It affects over 2% of the global population and is associated with higher suicide rates than the general population. Treatment options include antipsychotics, anticonvulsants, and lithium, which are effective for long-term management and suicide prevention. Lithium has long been the gold standard for BD treatment; however, it can cause thyroid and parathyroid dysfunction. Patients receiving medications for medical comorbidities can be at higher risk for side effects.

**Case Report:** In this case, a 66-year-old female with bipolar I disorder and multiple endocrine abnormalities was admitted during a manic episode with psychotic features that followed their discontinuation of lithium.

**Conclusion:** This case illustrates the complexities of managing BD with concurrent endocrine disorders and the importance of collaboration between specialty providers to maintain patient medical and psychiatric safety.

**Keywords:** Bipolar disorder, Endocrinology, Pharmacology, Psychiatry

## How to cite this article

Begley N, Tariq Z, Yip M, Sadaf S, Rajan M. Lithium and endocrine dysregulation in decompensated bipolar disorder with psychotic features. *Case Rep Int* 2025;14(2):12–16.

Article ID: 100135Z06NB2025

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doi: 10.5348/100135Z06NB2025CR

## INTRODUCTION

Bipolar affective disorders consist of marked and extended changes in mood such as mania, hypomania, and depression. Collectively, bipolar disorders affect more than 2% of the global population and are one of the leading causes of suicide attempts and functional impairment [1, 2]. Treatment for bipolar disorder (BD) includes antipsychotics, anticonvulsants, and antidepressants. According to the World Health Organization, essential medicines for BD include anticonvulsants like carbamazepine, valproic acid (VA), and lithium [3]. Antipsychotic drugs are more effective in treating acute manic episodes than anticonvulsants and lithium [4]. Studies have shown that anticonvulsants, such as lamotrigine and valproate, have shown modest, yet not statistically significant effects in treating BD [4]. Lithium, whose specific mechanism of action is still debated, has been shown to have the most benefit in the long-term maintenance treatment of BD and is the only known treatment to prevent suicidal behavior in patients with BD [5].

Common adverse side effects of lithium include thyroid disorders, parathyroid disorders, and renal impairment. Hypothyroidism is the most common thyroid disorder associated with chronic lithium use. Up to 30% of patients, frequently women over 45 years of age, develop hypothyroidism within two years of lithium therapy and an equal amount develop hyperparathyroidism with

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Received: 18 March 2025

Accepted: 21 August 2025

Published: 18 October 2025

hypercalcemia [6, 7]. The effects of lithium on prolactin levels remain unclear; studies have shown that short-term lithium use can increase prolactin levels while long-term use more can lead to decreased prolactin levels [8]. Given these results and the common endocrine side effects of lithium, therapy in patients with BD and concurrent endocrine disorders can pose a unique challenge.

Patients with BD and prolactinoma should be monitored closely. Bromocriptine is a dopamine D2 agonist that is often used in the treatment of pituitary prolactinomas. This medicine effectively reduces prolactin levels, minimizing the effects of hyperprolactinemia on sexual dysfunction and fertility issues [8]. However, bromocriptine treatment should be cautiously used in patients with BD or a history of psychosis due to its dopamine agonism. In these cases, bromocriptine treatment could heighten psychotic disorders and increase impulsivity [9, 10].

In this case, we present a patient with a history of bipolar I disorder who was admitted to the inpatient psychiatry unit for a manic episode. Her BD symptoms were previously stable on lithium therapy for more than 20 years, but due to the incidental discovery of a benign parathyroid nodule and minimally elevated calcium levels lithium therapy was discontinued.

## CASE REPORT

Our patient was a 66-year-old female with the diagnosis of bipolar I disorder and hypothyroidism who was admitted to the inpatient psychiatry unit after being brought by police after leaving the scene of an accident. Per the patient's son, the patient has experienced a dramatic shift in her mood and energy within the last three months and reports that symptoms have progressively worsened. In addition, the patient presented to the emergency department two months prior, where she demonstrated a flight of ideas, distractibility, irritability, and poor insight. At that time, they did not meet the criteria for mania, psychosis, or involuntary admission and refused the offer of voluntary admission.

From available records, the patient had been diagnosed with Bipolar I disorder in 2002. Her medical history included hypothyroidism, prolactinoma, parathyroid adenoma, hypercalcemia, and obesity. The patient had been on lithium for the past 24 years and had also been taking Bromocriptine since 1986 due to hyperprolactinemia. Three months prior, the patient was tapered off lithium 600 mg and titrated on Lamictal to 450 mg after the endocrinology provider palpated a nodule in the neck and detected an elevated parathyroid hormone (PTH) of 194 (10–65 pg/mL) with a calcium level of 10.8 (8.5–10.5 mg/dL), phosphorous 4.1 mg/dL (2.5–4.5 mg/dL), and vitamin D level of 33.3 ng/dL (20–50 ng/dL). A subsequent thyroid ultrasound revealed a "hypoechoic nodule in the left thyroid gland" (Figure 1). The patient was then sent for scintigraphy which

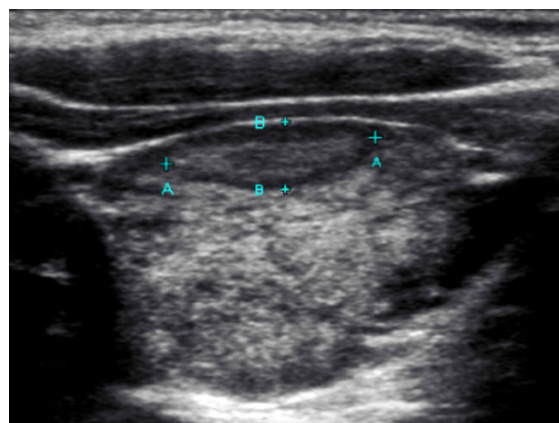


Figure 1: A hypoechoic nodule in the left thyroid gland.

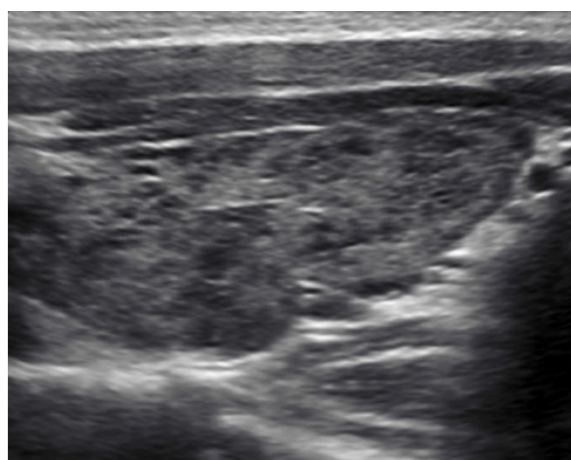


Figure 2: 14 months later; patchy infiltrates, hypoechoic in nature, consistent with autoimmune infiltrate in the left thyroid gland.

revealed no abnormal uptake throughout the thyroid or parathyroid. At that time, her PTH and calcium levels remained elevated at 158 and 10.5, respectively. Despite these inconclusive findings, lithium was discontinued by the patient's providers as a precautionary measure and titrated on lamotrigine. During this taper, the patient's psychiatrist of 20 years retired, and by the time of presentation, she was still in the process of establishing a new psychiatrist.

While in the psychiatric emergency department, the patient appeared disheveled and demonstrated rapid, pressured speech, labile mood, delusional thought content, decreased appetite, flight of ideas, irritability, increased goal-directed activity, hypersexual behavior, paranoid delusions, and hallucinations. Delusions were persecutory in character and centered around her previous co-workers conspiring against her to suppress large-scale fraud. The patient claimed to have all the evidence necessary but declined to share it due to "an ongoing investigation." Moreover, the patient accused hospital staff of working for the Nazi regime and stated that the FBI was planted amongst the other patients in the milieu. Despite a longstanding BD diagnosis, the patient

seemed to have no insight into her condition or the need for treatment. The patient refused all blood draws and EKGs but was willing to take medication and expressed a preference for Lamictal stating that her husband seemed to like her personality and increased libido after being taken off lithium. On hospital Day (HD) 1, the patient received Olanzapine 5 mg nightly for psychosis, lamotrigine 25 mg daily for mood stabilization, and her home medications bromocriptine 2.5 mg for prolactinoma, and valacyclovir 500 mg daily for herpes suppression treatment. The following day, she provided vital signs, electrocardiogram (EKG), and labs including TSH, free T4, parathyroid hormone (PTH), Prolactin, complete blood count (CBC), and comprehensive metabolic panel (CMP). All results were within normal limits except PTH and prolactin at 151.5 pg/mL and 22 ug/L (<20 ug/L), respectively.

During the first week of her hospitalization, this patient was titrated up to 20 mg of olanzapine with minimal clinical response. The patient had also started to refuse lamotrigine after three days and showed no response while taking it or appreciable worsening after discontinuation. Next, the patient was offered haloperidol and VA for psychosis and mania, respectively. Over the following four days, our patient showed moderate improvement in her mood lability, impulsivity, and hyperactivity. However, psychotic symptoms were remarkably unaltered. The patient insisted that the VA solution was, in fact, fentanyl and quit taking it despite a negative urine drug screen. On hospital day 8, records from an outside hospital were received, and more insight was provided regarding the patient's endocrinological history. The patient's pituitary microadenoma was never captured on imaging nor reimaged after 30 years of treatment with bromocriptine. Moreover, the elevated prolactin was a coincidental finding during an infertility workup. With this information, we attempted to obtain imaging of the patient's brain. Again, the patient's psychotic symptoms prohibited the appropriate medical workup as she accused staff of plotting to kill her in the imaging suite so that her investigative results would never be released. Despite the absence of further imaging, the patient's intermittent compliance and persistent symptoms prompted a different approach to treatment.

On hospital day 15, refractory symptoms led us to discontinue potentially contributory medications. Specifically, bromocriptine was discontinued. Within 48 hours the patient showed dramatic improvement in her psychotic symptoms. She began participating in shared decision-making with her providers and was able to confirm that a magnetic resonance imaging (MRI) obtained at the time of bromocriptine initiation revealed no adenoma. After discussing risks and benefits with the patient it was decided to restart lithium therapy. Over the following week, our patient showed remarkable improvement. The patient started sleeping more than 2–3 hours a night, irritability converted to congeniality, they no longer engaged in sexual behaviors with other patients, and they no longer felt that the nursing staff was

administering fentanyl or attempting to commit homicide. Repeat imaging and labs after one week of lithium therapy revealed a stable PTH level, normal calcium and thyroid infiltrate consistent with the patient's known history of autoimmune hypothyroidism (Figure 2).

## DISCUSSION

This case demonstrates the delicate balance required in managing a patient with bipolar I disorder alongside complex endocrine issues. In treating these patients, it is essential to weigh the risks and benefits of therapeutic interventions carefully. This becomes particularly important when treatments for one condition could exacerbate the other. Surgeons at Vanderbilt and Baylor University have recommended strategies for hypercalcemia in patients on Lithium therapy (Figure 1) [11]. Their recommendations acknowledge the risks of withdrawing maintenance therapy and the ability to resume lithium post-operatively with a low likelihood of recurrent hyperparathyroidism or hypercalcemia [11, 12]. For this patient, the sequelae of her treatments for comorbidities resulted not only in a worsening of her BD but also provoked psychosis for the first time in her life. Moreover, the removal of lithium after 20 years of maintenance did not normalize calcium levels and likely contributed to the development of mania. This patient's decompensation underscored the need for caution in treating medical conditions in patients with BD as the psychiatric side effects may outweigh the endocrinological benefits and acute risks for morality.

## CONCLUSION

This case exemplifies the delicate balance in managing patients with BD and comorbid medical conditions. In treating these patients, it is imperative that clinicians carefully assess the risks and benefits of therapeutic interventions. It is vital to consider whether the treatment of one disorder can provoke or worsen another. For this patient, the use of bromocriptine did lower prolactin levels; however, the same treatment provoked psychotic symptoms for the first time. In addition, the withdrawal of the patient's mood stabilizing medication, Lithium, ultimately did not influence a change in calcium levels. This change resulted in a recurrent episode of mania for the first time in nearly 20 years. Our patients' abrupt decompensation highlighted the need for mindful assessment of treatment plans and the acute risks for mortality and irreparable damage to the patient's quality of life.

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## Acknowledgments

The authors would like to thank our attending physician Michelle Oyeka, MD for her teaching, mentorship, and

encouragement throughout the writing and publication of this report.

Grammarly Inc. generative AI was used in both the introduction and conclusion to correct grammatical errors and summarize information.

### Author Contributions

Noah Begley – Conception of the work, Design of the work, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Zuha Tariq – Conception of the work, Acquisition of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Maggie Yip – Conception of the work, Acquisition of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Saad Sadaf – Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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### Guarantor of Submission

The corresponding author is the guarantor of submission.

### Source of Support

None.

### Consent Statement

Written informed consent was obtained from the patient for publication of this article.

### Conflict of Interest

Authors declare no conflict of interest.

### Data Availability

All relevant data are within the paper and its Supporting Information files.

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