Cambridge City Publishers

Case Reports in Clinical Cardiology Journal

Case Report | Vol 6 Iss 4

Unmasking of Brugada Syndrome Type 1 from Lithium Administration

Ethan Swartz[1*], Navid Radfar[1], Harsh Singh[1], Serin Varughese[1], Eugene Kim[1], Meet Shah[2], Eison de Guzman[2], Delia Cotiga[3] and Renjit Thomas[3]

[1]Department of Internal Medicine, Rutgers New Jersey Medical School, 185 South Orange Avenue, Newark, NJ, USA

[2]Division of Cardiology, Department of Medicine, Rutgers New Jersey Medical School, 185 South Orange Avenue, Newark, NJ, USA

[3]Department of Cardiology, VA New Jersey Healthcare System, 385 Tremont Avenue, East Orange, NJ, USA

[*]**Corresponding author:** Ethan Swartz, Department of Internal Medicine, Rutgers New Jersey Medical School, 185 South Orange Avenue, Newark, NJ, USA. E-mail: es1398@njms.rutgers.edu

Received: July 16, 2026; **Accepted:** August 03, 2026; **Published:** August 15, 2026

Abstract

Brugada syndrome (BrS) is an inherited cardiac channelopathy associated with characteristic electrocardiographic (ECG) changes and an increased risk of sudden cardiac death (SCD). These ECG findings are often concealed and may be unmasked by sodium channel blocking agents. Lithium, a first-line mood stabilizer for bipolar disorder, exerts clinically relevant cardiac sodium channel blockade, though its association with BrS is infrequently reported. We describe a case of a 28-year-old male with bipolar disorder type I who presented with chest pain and was found to have a type 1 Brugada ECG pattern while taking lithium therapy. Following discontinuation of lithium and transition to aripiprazole, repeat ECG one year later demonstrated resolution of the Brugada pattern. Given lithium's widespread use and the transient nature of drug-induced Brugada patterns, heightened clinical awareness is essential to avoid misdiagnosis and mitigate arrhythmic risk.

Introduction

Bipolar disorder is a pathological mood disorder characterized by significant mood shifts [1]. Mood stabilizers such as lithium continue to be the mainstay of treatment in preventing relapses [2]. In fact, prescription rates for lithium across the United States are nearly 17% [3]. Nevertheless, given its narrow therapeutic range, lithium is associated with many deleterious effects including the potential unmasking of BrS [4-5]. It is hypothesized that lithium leads to uncovering of BrS through cardiac sodium channel blockade [4].

BrS is an autosomal dominant disease associated with classic ECG changes according to diagnostic criteria. Given the risk for SCD, it is imperative for clinicians to accurately diagnose and manage such patients [6]. The ECG changes of BrS are often concealed but can be unmasked by sodium channel blockers such as flecainide and procainamide [7]. However, there is a paucity of literature describing the association of BrS and lithium usage. This case highlights lithium's potential to unmask latent BrS and underscores the importance of recognizing drug-induced Brugada patterns to guide management and reduce arrhythmic risk.

Case Presentation

A 28-year-old male with a history of bipolar disorder type I presented with sharp chest pain and shortness of breath during a manic episode. An initial ECG revealed a type 1 Brugada pattern. Laboratory tests showed normal troponin levels (<0.03 ng/mL), magnesium at 2.2 mg/dL, calcium at 9.6 mg/dL, CRP at 4.52 mg/L and lithium at 0.4 mmol/L. The following day, after learning about his abnormal ECG findings, the patient became tearful and experienced chest pain unrelieved by nitroglycerin. Repeat ECGs continued to display the type 1 Brugada pattern, with troponin levels remaining normal (Figure 1). He reported feeling overwhelmed by his new cardiac diagnosis.

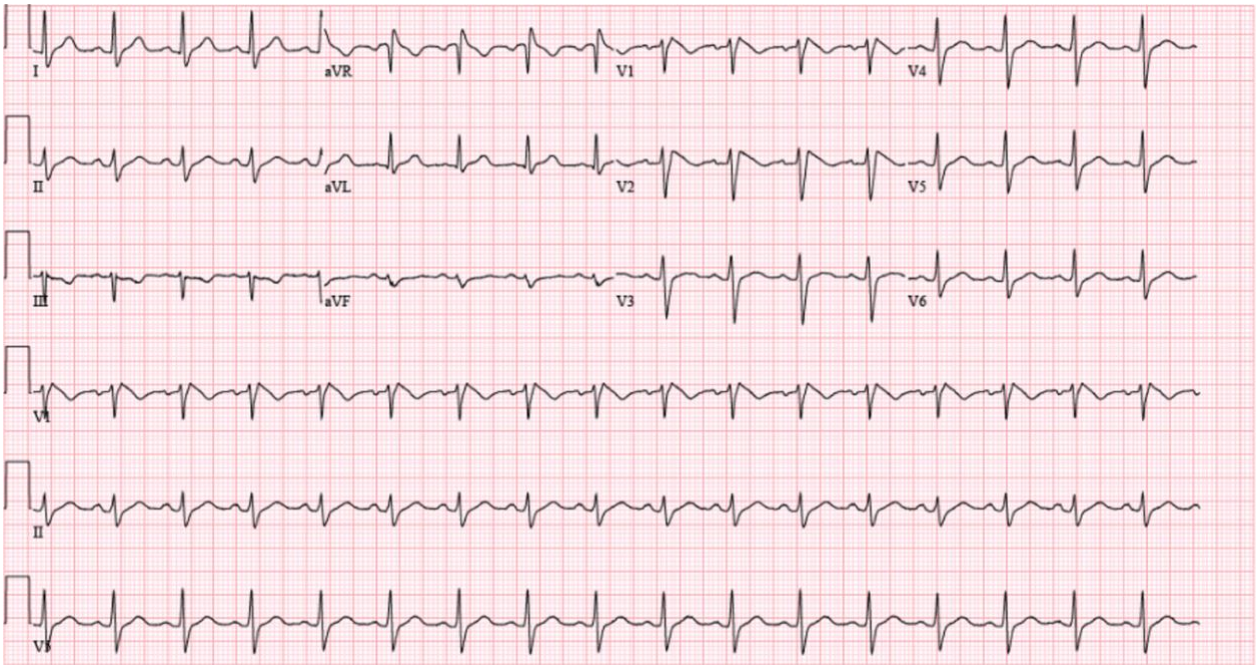

Figure 1: ECG initially obtained after patient complained of substernal chest pain. The patient was on lithium treatment at the time of this ECG. There are coved ST segments of about 3-4mm in leads V1-V2 with T wave inversions in both leads.

Given the potential pro-arrhythmogenic effects of lithium, it was discontinued, and the patient was transitioned to aripiprazole for continued management. An echocardiography performed during the inpatient hospitalization did not demonstrate signs of structural heart disease. The patient denied any history of syncope, presyncope, dizziness, or palpitations and reported an active lifestyle with good exercise tolerance. Notably, his family history was significant for a grandfather who died suddenly at the age of 31.

After discontinuation of lithium, the patient was discharged with a prescription for aripiprazole and scheduled for outpatient electrophysiology follow-up to assess the need for further intervention. Repeat ECG one year later demonstrated reversal of the coved ST-segment elevations in leads V1-V2 with a redemonstrated T wave inversion in lead V1 and normalization of the ST-segment with an upright T wave in lead V2 (Figure 2).

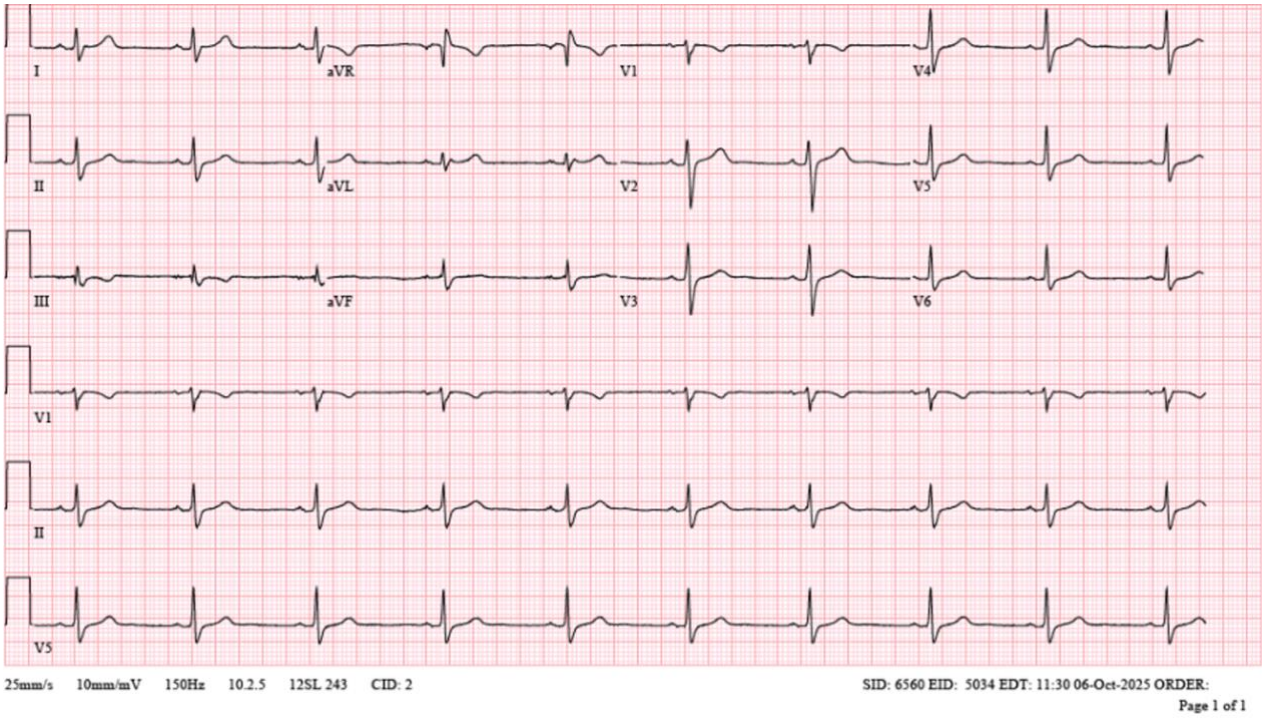

Figure 2: ECG obtained about 1 year after discontinuation of lithium. There is a loss of coved ST segments in leads V1-V2 with a redemonstrated T wave inversion in lead V1. There is normalization of the ST-segment and T wave axis in lead V2.

Discussion

BrS is an inherited cardiac channelopathy characterized by distinct electrocardiographic patterns and an increased risk of malignant ventricular arrhythmias and sudden cardiac death. The diagnostic hallmark of a type 1 Brugada ECG pattern is defined by coved ST-segment elevation ≥ 2 mm followed by T-wave inversions in the right precordial leads (V1–V3). These ECG changes may be transient and often concealed, presenting only under certain conditions, such as fever, electrolyte abnormalities, or exposure to sodium channel-blocking agents, as in this patient.

Lithium, a first-line mood stabilizer for bipolar disorder, has well-documented electrophysiologic effects, including inhibition of cardiac sodium channels. Although lithium is not traditionally classified as an antiarrhythmic agent, its sodium channel blocking properties are believed to mechanistically resemble those of class I antiarrhythmics, which are known to unmask latent Brugada ECG patterns. Prior reports describing lithium-associated Brugada patterns remain limited, underscoring the clinical importance of recognizing this association, particularly given lithium's widespread use and narrow therapeutic index.

Accurate differentiation of BrS from ECG mimics, such as incomplete right bundle branch block, early repolarization, or acute ischemia, is essential as management and prognostic implications differ substantially. ECG angles such as the β angle have been proposed as adjunctive tools to differentiate Brugada patterns from benign conduction abnormalities. However, once a clear type 1 Brugada pattern is identified, as in this patient, the diagnosis is established according to current consensus guidelines, and additional ECG metrics serve only as an adjunctive tool to support the diagnosis.

This patient's family history of sudden cardiac death at a young age further raises concern for an inherited arrhythmogenic substrate. While genetic testing was not reported in this case, it is notable that pathogenic variants, most commonly in the SCN5A gene, are identified in only about 30% of patients with BrS [8]. As such, the absence of a genetic diagnosis would not exclude the condition.

Management of BrS centers on risk stratification and avoidance of known triggers. In this case, prompt discontinuation of lithium was appropriate given its potential role in unmasking the Brugada pattern. Outpatient electrophysiology follow-ups remain essential for this patient for longitudinal risk assessment. Patient education regarding fever management and avoidance of contraindicated medications is also a critical component of care.

This case adds to the growing, but still limited body of literature associating lithium therapy to the unmasking of BrS. It highlights the importance of maintaining a high index of suspicion for BrS in young patients presenting with characteristic ECG findings while receiving sodium channel blocking psychotropic medications, even in the absence of overt arrhythmic symptoms.

Take Home Message

Lithium's sodium channel-blocking properties can unmask a latent type 1 Brugada ECG pattern, underscoring the importance of an initial ECG in young patients receiving mood stabilizers that have sodium channel-blocking properties. Prompt recognition and discontinuation of the offending agent, along with appropriate electrophysiology follow-up, are critical to mitigate the risk of malignant arrhythmias and sudden cardiac death.

REFERENCES

1. Nierenberg AA, Agustini B, Köhler-Forsberg O, et al. Diagnosis and Treatment of Bipolar Disorder: A Review. JAMA. 2023; 330: 1370-1380.
2. Machado-Vieira R, Manji HK, Zarate CA Jr. The role of lithium in the treatment of bipolar disorder: convergent evidence for neurotrophic effects as a unifying hypothesis. Bipolar Disord. 2009; 11 (Suppl 2): 92-109.
3. Sköld M, Rolstad S, Joas E, et al. Regional lithium prescription rates and recurrence in bipolar disorder. Int J Bipolar Disord. 2021; 9: 18.
4. Crawford RR, Higdon AN, Casey DB, et al. Multiple lithium-dependent Brugada syndrome unmasking events in a bipolar patient. Clin Case Rep. 2015; 3: 14-18.
5. Wright D, Salehian O. Brugada-type electrocardiographic changes induced by long-term lithium use. Circulation. 2010; 122: e418-e419.
6. Krahn A, Behr E, Hamilton R, et al. Brugada Syndrome. J Am Coll Cardiol EP. 2022; 8: 386-405.
7. Darbar D, Yang T, Churchwell K, et al. Unmasking of brugada syndrome by lithium. Circulation. 2005; 112: 1527-1531.
8. Doundoulakis I, Pannone L, Chiotis S, et al. SCN5A gene variants and arrhythmic risk in Brugada syndrome: An updated systematic review and meta-analysis. Heart Rhythm. 2024; 21: 1987-1997.