

# Lithium toxicity induced complete heart block: A case report

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## ARTICLE INFO

### Article history:

Received  
21 March 2025  
Revised in revised form  
13 August 2025  
Accepted  
13 October 2025  
Published  
1 September 2026

### Keywords:

Lithium,  
Cardiotoxicity,  
heart block,  
haemodialysis,  
B  
bipolar disorder

### DOI:

10.24191/jchs.v11i2.13414

## ABSTRACT

Lithium is traditionally used as a mood stabiliser for the treatment of bipolar disorder. Despite its proven effectiveness, it has a narrow therapeutic index. Lithium intoxication presents with a spectrum of clinical manifestations that are directly proportional to the serum lithium level. It ranges from mild gastrointestinal symptoms to more severe cardiovascular manifestations. We report a case of a 79-year-old male with underlying bipolar disorder who presented with unstable complete heart block following lithium intoxication. He was started on transcutaneous pacing in the emergency department and later underwent haemodialysis. The complete heart block resolved with the normalisation of the serum lithium levels. Lithium causes cardiotoxicity through various mechanisms, including competition with electrolytes involved in cellular membranes. Haemodialysis remains the mainstay treatment for severe lithium toxicity. Prompt recognition is crucial as patients can present with various cardiac manifestations after lithium toxicity, particularly in elderly patients, due to their underlying comorbidities.

## 1. INTRODUCTION

Lithium has traditionally been used as a first-line mood stabiliser for the treatment of bipolar disorder [1]. Despite its proven effectiveness in protecting against manic and depressive episodes in bipolar disorder, lithium has a narrow therapeutic index. Therefore, it predisposes patients to develop lithium toxicity. Possible complications of lithium overdose range from mild symptoms of nausea, vomiting, diarrhoea, and muscle weakness to a more severe spectrum, such as altered mental status, seizures, nephrogenic diabetes insipidus, and cardiac arrhythmias [2]. We report a case of a 79-year-old male with lithium intoxication who presented to the emergency department (ED) with an unstable complete heart block (CHB).

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## 2. CASE PRESENTATION

This is a case of a 79-year-old male with history of type II diabetes mellitus, dyslipidaemia and bipolar disorder on oral lithium. He presented with a week history of altered mental status in which he became progressively less verbal and entirely dependent. The psychiatry team recently increased the oral lithium dose from 300 mg daily to 600 mg because his manic symptoms were not adequately controlled. Two days before admission to the ED, he had a history of falls in the toilet and sustained a laceration wound over the nose. He was brought to a private medical centre, where a plain computed tomography (CT) scan of the brain was done and revealed normal findings. He was discharged after he underwent toilet and suturing of the laceration wound. Given the declining symptoms and the progressively deteriorating altered sensorium, he was brought to the ED for further treatment. On arrival at the ED, the patient was drowsy with a Glasgow coma scale of 12/15 (E4V2M6). His vital signs were significant for bradycardia with heart rate of 28 beats per minute. His blood pressure was 110/80 mmHg, oxygen saturation of 95% on room air, and temperature of 37 °C. The cardiorespiratory and abdominal examinations were unremarkable. The neurological examination revealed no focal deficits despite a depressed level of consciousness. Electrocardiogram (ECG) revealed CHB with infrahisian ventricular escape rhythm with P wave rate of 70 bpm and wide QRS complex with the rate of 20 bpm as shown in Figure 1. Focused cardiac ultrasound revealed good ejection fraction with no regional wall motion abnormalities. His blood investigations are notable for prerenal acute kidney injury with hypernatremia, the latter being consistent with nephrogenic diabetes insipidus secondary to lithium. His full blood count and liver function tests were within normal limits.

Repeated plain CT of the brain was done and revealed normal findings. Due to the unstable CHB, he was started on transcutaneous pacing, which eventually increased the heart rate. The initial level of lithium upon admission was 2.3 mEq/L, which is above the normal therapeutic level (0.8 to 1.5 mEq/L). He was admitted to the cardiology care unit for further treatment and was started on intermittent haemodialysis. The complete heart block completely resolved with the normalisation of the serum lithium levels. His oral lithium was stopped and changed to oral sodium valproate. He was discharged well with outpatient psychiatry follow-up.

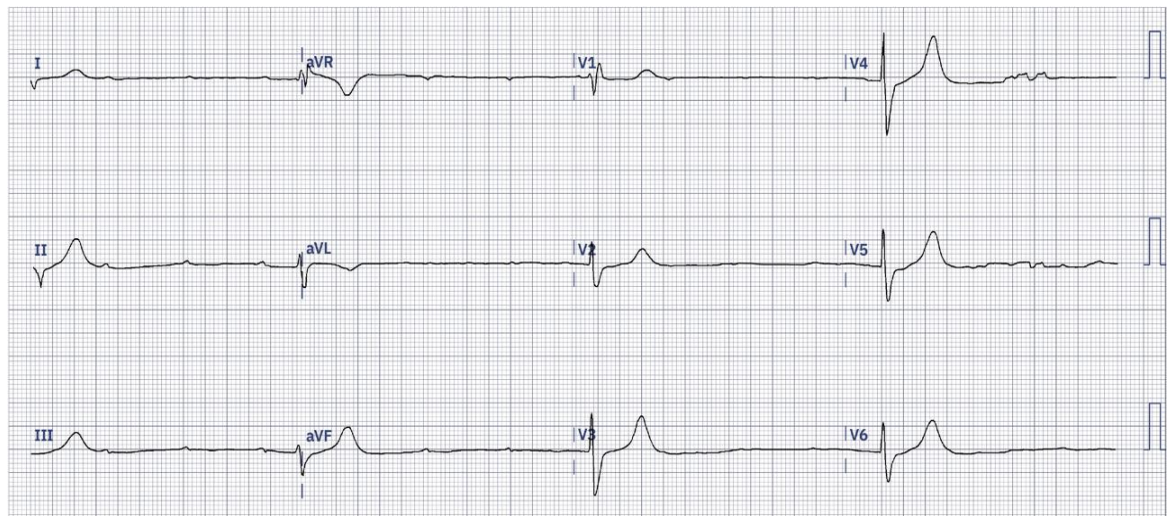


Fig. 1. ECG revealed a complete heart block

Source: Muhammad Nur Azmi Baharuddin (2026)

### 3. DISCUSSION

Lithium is a naturally occurring salt in the form of an alkali element, which was discovered serendipitously to treat bipolar mood disorder and introduced into clinical practice in the mid-20<sup>th</sup> century. It is widely used as a first-line treatment for bipolar affective disorder in the general population and older adults. It is also used for the treatment of other nonpsychiatric diseases, such as cluster headaches and migraines [1]. Despite its therapeutic benefits, lithium has a narrow therapeutic index, making toxicity a significant risk even at near-therapeutic levels [2]. Patients with lithium intoxication presented with a spectrum of clinical manifestations, and the severity is directly proportional to the degree of elevation of the serum lithium levels. The manifestation of lithium toxicity varied, with neurotoxicity being the most common, followed by renal and cardiovascular toxicity [3]. Several risk factors predisposed the patient to develop lithium toxicity, including advanced age and a rapid increase in serum lithium level following certain conditions such as dehydration and renal dysfunction [3]. Cardiac manifestations of lithium toxicity can range from benign ECG changes to severe arrhythmias, including CHB [2].

CHB, also known as third-degree heart block, is a severe cardiac condition where the electrical signals from the atria do not reach the ventricles, leading to a complete dissociation of atrial and ventricular rhythms [4]. While CHB can result from various etiologies, including coronary ischemia, congenital conditions, and certain medications, lithium toxicity is a notable but rare cause [4]. The exact mechanisms by which lithium induces cardiac toxicity are not fully understood. However, it is known that lithium can interfere with the normal function of cardiac ion channels and electrogenic pumps, affecting the distribution and passage of sodium, potassium, and calcium ions across the cardiac cell membrane. This disruption can lead to a spectrum of cardiac effects, ranging from benign nonspecific ST segment and T wave abnormalities to severe manifestations such as atrioventricular block, atrial flutter, sinus node dysfunction, ventricular tachycardia, ventricular fibrillation and CHB [2]. This patient also developed hypernatremia due to nephrogenic diabetes insipidus, which can occur with lithium toxicity. It induced nephrogenic diabetes insipidus by its action on renal tubular function and caused lithium nephropathy [2]. The diagnosis of nephrogenic diabetes insipidus was made clinically based on the presence of hypernatremia in the context of lithium use, as a formal water deprivation test was contraindicated given the patient's unstable condition. According to our observation, it appears that advanced age and his comorbidities may have contributed to the development of toxicity, in which the patient had received lithium treatment for years. Age and diabetes mellitus were also important factors that led to decreased creatinine clearance, which predisposed the patient to develop lithium toxicity.

Management of lithium-induced CHB typically involves the immediate cessation of lithium, supportive care, and interventions to normalise serum lithium levels. Haemodialysis remains the mainstay of treatment in severe lithium toxicity, as it is one of the most readily dialysable toxins due to its small atomic weight and negligible protein [5]. Pacemaker implantation may be necessary for patients with persistent bradyarrhythmia or CHB [3]. Prognosis varies depending on the severity of toxicity and the timeliness of intervention, but many patients recover fully with appropriate treatment. The duration of haemodialysis is guided by the serial serum lithium level [5]. As in this patient, he requires intermittent haemodialysis until the normalisation of the lithium level.

This case highlights the crucial need for vigilant monitoring in patients on lithium therapy, particularly in elderly patients with comorbidities. The patient's toxicity developed shortly after his lithium dose was doubled. A more gradual titration dose with frequent serum level checks might have mitigated this adverse event. For clinicians, a key learning point is to maintain a high index of suspicion for lithium toxicity in patients presenting with new-onset cardiac symptoms. This case demonstrates that regular screening of both lithium levels and renal function is paramount to ensure patient safety.

#### 4. CONCLUSION

Lithium toxicity is a rare but serious cause of CHB. Prompt recognition is crucial, as patients can present with various cardiac manifestations after lithium toxicity, particularly in elderly patients, due to their underlying comorbidities. It is also essential to carefully review the patient's drug history in patients with altered consciousness and arrhythmia, as drug intoxication can be one of the culprits. This case demonstrates that lithium therapy should be used with extreme caution and frequent monitoring, especially in elderly patients with comorbidities, due to its narrow therapeutic window.

#### 5. ACKNOWLEDGEMENTS

The author would like to thank the patient for providing clinical information for this case and for permission to publish this case report.

#### 6. GENERATIVE AI DISCLOSURE STATEMENT

During the preparation of this manuscript, the author used Grammarly to assist with the language editing. The author has reviewed and revised all AI-assisted output and takes full responsibility for the content of this publication.

#### 7. CONFLICT OF INTEREST

Author declare none.

#### 8. AUTHORS' CONTRIBUTIONS

MNAB drafted the case report and contributed to its critical review.

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