

## CASE REPORTS

# A Bizarre Electrocardiographic Pattern Due to Chronic Lithium Therapy

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Cardiotoxicity that results from lithium overdose is uncommon and electrocardiographic (ECG) changes are rarely reported. However, some authors have specifically reported the occurrence of ischemic ECG changes due to a lithium overdose. This article describes a case that is demonstrating ECG changes that mimic inferior myocardial infarction during the course of chronic lithium treatment and showing QTc prolongation in this patient. The patients' ECG changes were partially recovered after hemodialysis.

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electrocardiography; lithium; bipolar disorder

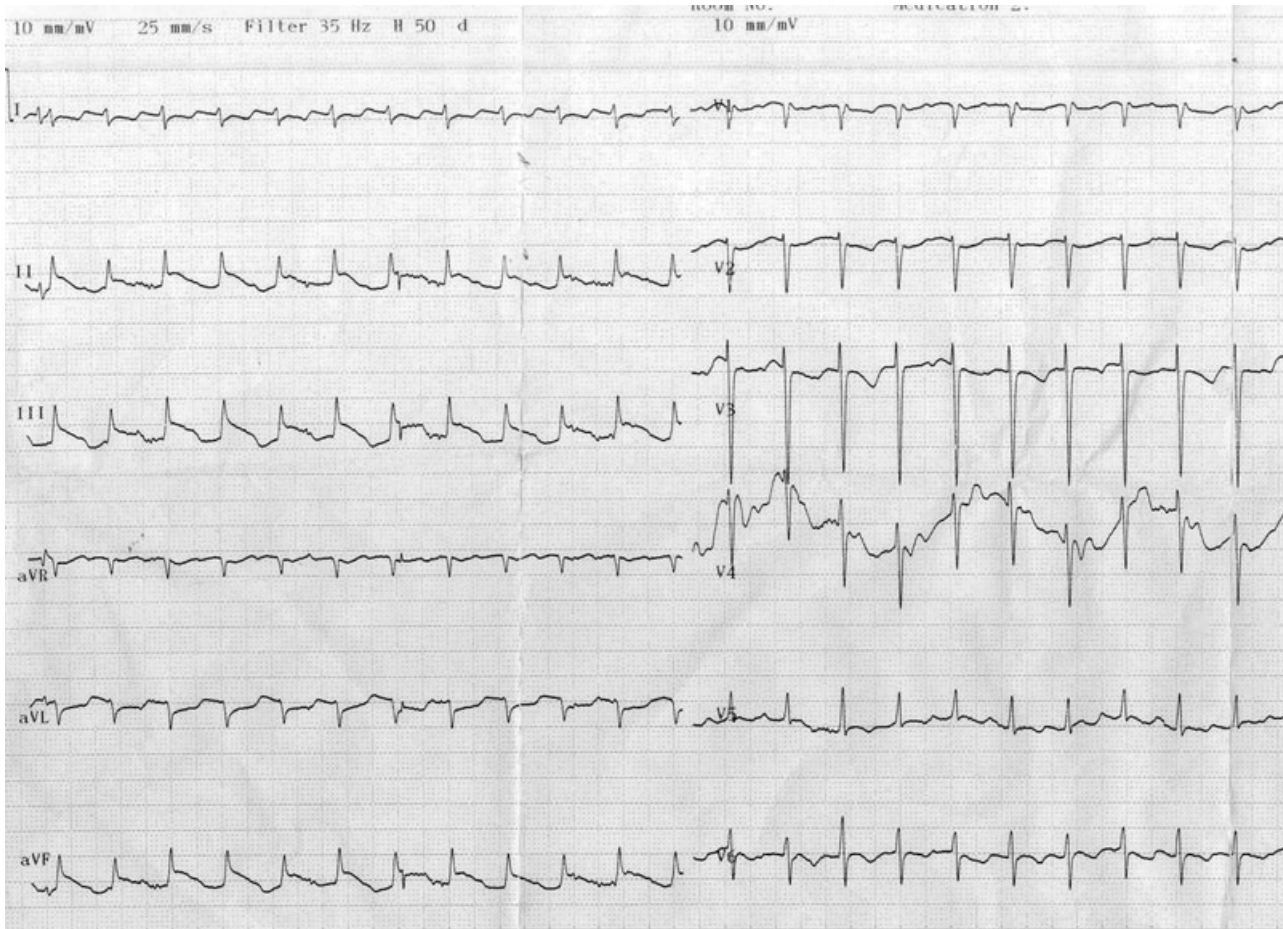
### CASE

A 70-year-old female who suffered from hypertension (HT) and bipolar disorder presented to the emergency department (ED) with dizziness, somnolence, and disorientation of time and place. She reported that she had taken a daily dose of 300 mg of lithium for over 15 years. The patient's antihypertensive therapy also required her to take 16 mg of candesartan cilexetil and 12.5 mg of hydrochlorothiazide each day. She had no relevant cardiac history and did not complain of chest pain or shortness of breath. Seven days prior to the patient's visit to the ED, her psychiatrist had increased her lithium dose to 300 mg twice per day. Upon admission to the ED, the patient's vital signs were abnormal: according to her medical records, "she appeared to be confused and agitated," and presented cool peripheries, a pulse of 135 bpm, and blood pressure of 90/60 mmHg. She was awake but her speech was incoherent. A cranial CT was conducted to further explore the patient's confused and ataxic state. The test did not reveal any areas of hemorrhagic infarct or mass. Her lithium level was 3.85 mEq/L (therapeutic blood level: 0.8 to

1.5 mEq/L). The patient's laboratory profile revealed a serum sodium level of 134 mmol/l, potassium at 3.5 mmol/l, and creatinine at 1.7 mg/dl. The patient's basic metabolic panel showed a white blood cell count of 8000/mm<sup>3</sup>. Furthermore, at her arrival, her plasma levels of both troponin I (Trp I) and creatine kinase-MB (CK-MB) were within the normal range (0.03 ng/dl and 2.7 ng/dl, respectively). Enzyme prosecutions were normal over 48 hours (CK-MB: 3.8 ng/dl and Trp I: 0.04 ng/dl).

An electrocardiogram showed ST elevations in leads II, III, and aVF, accompanied by ST depressions in leads I, aVL, V<sub>5</sub>, and V<sub>6</sub>. Details from the electrocardiogram revealed V<sub>5</sub> with 1 mm of ST-segment depression, V<sub>6</sub> with 2 mm of ST-segment depression, DI with 1 mm ST-segment depression, aVL with 1 mm ST-segment depression, DIII with 2 mm ST-segment elevation, and aVF with 1 mm ST-segment elevation. The PR duration was 160 ms; the QT duration was 400 ms; and QTc (corrected QT duration by Bazett formula) was prolonged before and after hemodialysis (QTc: 480 and 481 ms, respectively) (Fig. 1).

An echocardiogram showed minimal pericardial effusion, moderate mitral and mild aortic



**Figure 1.** Twelve-lead electrocardiogram at admission (heart rate: 135 bpm, PR duration: 210 ms, QT duration: 320 ms, QTc: 480 ms, QRS duration: 60 ms).

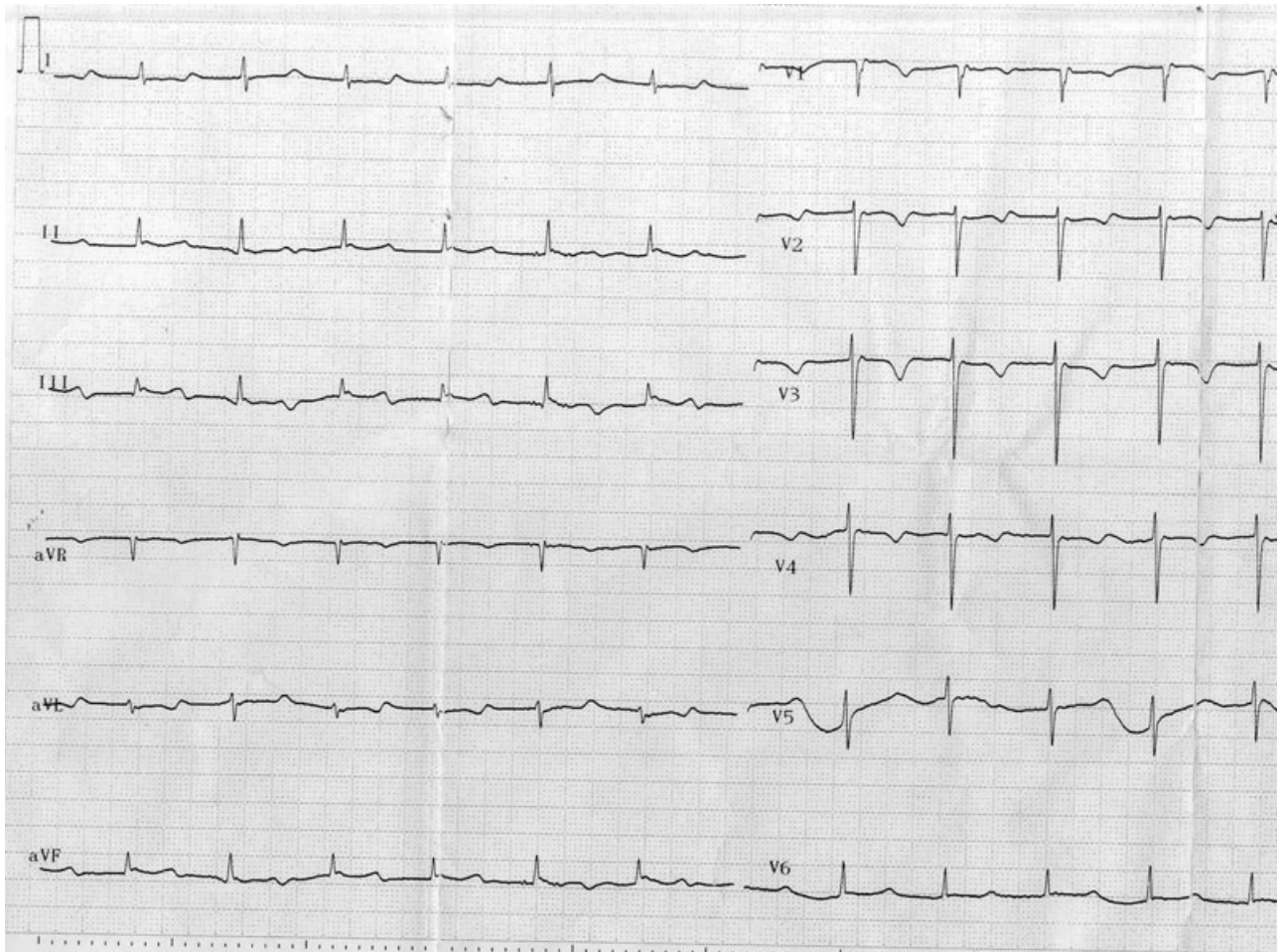
regurgitation, and normal wall motions. The ejection fraction of the left ventricle was calculated at 60% according to a modified Simpson's formula. Pulmonary systolic pressure was measured at approximately 30 mmHg. After two courses of hemodialysis for a total of 7 hours, the lithium level decreased to 0.87 mEq/L. The electrocardiogram showed partial recovery, but deep T inversions persisted in leads V<sub>1</sub>-V<sub>4</sub> (Fig. 2).

## DISCUSSION

Lithium toxicity may be associated with variable electrocardiographic (ECG) changes including QT prolongation, sinus node dysfunction, ST-segment changes, and T-wave changes.<sup>1-5</sup> Darbar et al. showed lithium-induced transient ST-segment elevation (Brugada type 1 pattern) in the right precordial leads in therapeutic concentrations in 2 pa-

tients with bipolar disorder.<sup>6</sup> In these 2 patients, ST elevation was seen in leads V<sub>1</sub> and V<sub>2</sub>. Lithium causes toxicity through several mechanisms including competition with sodium, potassium, calcium, and magnesium ions, each of these ions playing an important role in cellular membrane physiology.

We present a case of lithium toxicity that shows an unusual ECG pattern mimicking inferior ST-segment elevation myocardial infarction (MI). ST-segment changes suggesting ischemia due to lithium toxicity were first described by Puhr et al.<sup>7</sup> and Canan et al.<sup>8</sup> Puhr et al. reported that ST segments were elevated in the anterior leads with downward concavity, and T waves were biphasic. More specifically, V<sub>1</sub> showed 1 mm of ST-segment elevation, V<sub>2</sub>, 2 mm of ST-segment elevation, and V<sub>3</sub>, 1.5 mm of ST-segment elevation.<sup>6</sup> Canan et al. showed ST-wave depressions in leads V<sub>2</sub> and V<sub>3</sub> and a common T-wave inversion. But, our case was



**Figure 2.** Twelve-lead electrocardiogram after hemodialysis (heart rate: 79 bpm, PR duration: 260 ms, QT duration: 420 ms, QTc: 482 ms, QRS duration: 60 ms).

focused on ST elevations in leads II, III, and aVF accompanied by ST depressions and deep T inversions in leads V<sub>1</sub>-V<sub>4</sub>.

With an electrocardiogram suggesting an acute MI, cardiac enzymes and an emergency echocardiogram were used to rule out MI. Resolution of the ECG ST-segment changes with hemodialysis suggested that ECG changes associated with lithium toxicity can be misinterpreted as cardiac ischemia.

The electrocardiogram also revealed QTc prolongation in this patient. Among the many psychotropic drugs, antipsychotics and antidepressants have proved to be determinants of QT prolongation.<sup>9</sup> Mamiya et al. reported that lithium concentration correlates with QTc intervals in patients with psychosis.<sup>10</sup>

Our case involved an elderly female who experienced comorbid conditions including HT, hypo-

volemia, renal dysfunction, and candesartan and hydrochlorothiazide use. Our observations indicate that advanced age and the other comorbid conditions may have contributed to the development of intoxication symptoms. Age and HT were also important factors contributing to decreased creatinine clearance. In addition, reversible increases in serum lithium concentrations and toxicity have been reported during concomitant administration of lithium with angiotensin converting enzyme inhibitors, and with some angiotensin II receptor antagonists. Also, lithium generally should not be given with diuretics. Diuretic agents (such as hydrochlorothiazide) reduce the renal clearance of lithium and add a high risk of lithium toxicity.

In conclusion, emergency physicians, internal medicines, cardiologists, and also nurses should be reminded that patients may occasionally present

with ECG changes that mimic MI because of lithium toxicity.

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