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CASE REPORT

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Lithium intoxication causing ST segment elevation and wandering atrial rhythms in an elderly patient

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Abstract

Lithium overdoses causing cardiotoxicity are uncommon and electrocardiographic changes suggesting myocardial ischemia are rare. However, some authors have specifically reported the occurrence of ischemic electrocardiography changes due to a lithium overdose. This paper describes a case where electrocardiography changes mimic inferior myocardial infarction during the course of chronic lithium treatment in an elderly patient. The patient's electrocardiography changes were partially resolved after hemodialysis. (Cardiol J 2009; 17, 4: 404–407)

Key words: electrocardiography, toxicity

Introduction

Lithium is commonly used to treat conditions such as depressive and bipolar affective disorders. The therapeutic index of lithium is narrow and its toxicity can cause multisystem dysfunction [1]. Lithium overdose is generally not associated with cardiac disorders, but it can occur in individuals who have underlying heart disease [2]. Lithium overdose is associated with variable electrocardiography (ECG) changes including QT prolongation, ST segment and T wave changes. Cardiac arrhythmias: junctional bradycardia, sinus node and atrioventricular (AV) dysfunction, including sinus bradycardia, sinoatrial block, and first-degree AV block, ventricular asystole [3, 4], and myocardial infarction (MI) have been documented [5]. We observed a case involving lithium intoxication that electrocardiographically mimicked inferior MI in an elderly patient.

Case report

A 66 year-old female suffering from diabetes, hypothyroidism and major depression was brought to the emergency department (ED) due to her altered mental state. The patient had received 300 mg of lithium twice daily for more than 20 years to treat her major depression.

Additional medication which the patient took on a daily basis included 20 mg of paroxetine, 100 mg of aspirin, 120 mg of gliclazide, and 100 μg of levothyroxine. Seven days prior to the presentation, the patient became confused and experienced a loss of appetite. Upon admission to the ED, the patient's lithium level was 1.9 mEq/L (therapeutic blood level 0.8 to 1.5 mEq/L). 'She appeared to be confused and somnolent', blood pressure: 80/50 mm Hg; pulse: 130 bpm; respiratory rate: 28; and oxygen saturation at 88%. Intravenous saline and

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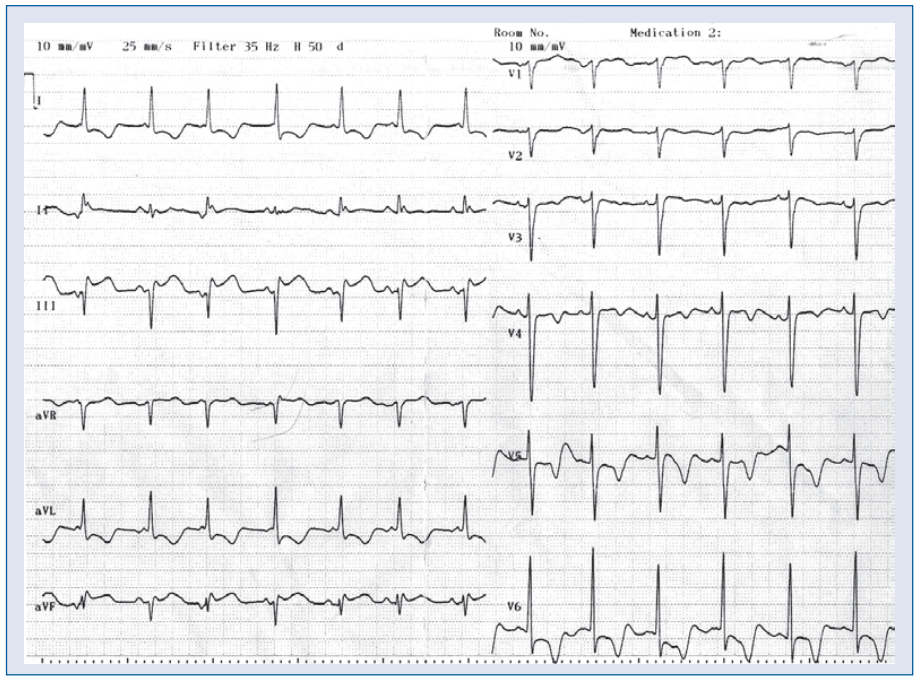

Figure 1. Electrocardiogram at admission revealed irregular rhythm with 78 bpm and first degree atrioventricular block (PR duration: 160 ms). P waves are variable in morphology. Wandering atrial pacemaker was revealed, QT interval and QRS duration were normal (QT: 320 ms and QRS duration: 90 ms, respectively).

dopamine infusion (5 μg/kg/h) were begun and supplementary oxygen was delivered. After administering IV fluids and inotropic support, the patient's blood pressure increased to 110/60 mm Hg and her pulse was reduced to 90 bpm. Her laboratory profile revealed serum sodium of 137 mEq/L, potassium of 5.0 mEq/L, urea of 74 mEq/L, and creatinine of 1.7 mg/dL. The results of the patient's thyroid function tests and chest radiography were within normal limits.

An ECG showed lead III with 2 mm of ST segment elevation, II with 1 mm of ST segment elevation, aVF with 2 mm of ST segment elevation, lead I, aVL with 1 mm of ST segment depression and T inversion in the anterior leads. Also, P wave morphologies were different, suggesting a wandering atrial pacemaker (Fig. 1). On admission, the patient's cardiac markers were within normal levels (CK-MB: 6.0 ng/dL and Trp I: 0.01 ng/dL). Serial cardiac enzymes were done without any significant change. An echocardiogram did not reveal abnormal wall motion. The ejection fraction of the left ventricle was calculated at 60% according to a modified Simpson's formula. The right ventricle sizes and estimated pulmonary artery pressure were within the normal range. The patient was admitted for hemodialysis with a presumptive diagnosis of lithium intoxication. After hemodialysis, her lithium level decreased to 1.09 mEq/L and ECG partially normalized. Deep T-waves in the apical-lateral wall still persisted (Fig. 2). Following a second session of dialysis to treat her hemodynamic instability, the patient's vital signs became normal. She was discharged several days later.

Discussion

Lithium causes toxicity through several mechanisms including competition with sodium, potassium, calcium, and magnesium ions, each of which plays an important role in cellular membrane physiology.

Because of these mechanisms, certain ECG changes, such as QT prolongation, non-specific ST segment and T wave abnormalities can occur. ECG changes are more likely to result from chronic rather than acute lithium overdoses [6]. Puhr et al. [5] wrote: 'Lithium intoxication can cause transient ST segment elevations suggesting of an acute myocardial infarction'. And Canan et al. [7] reported a single case with a 1 mm ST segment elevation in the inferior leads which was related to a lithium overdose. Three years previously Darbar et al. [8] demonstrated that lithium induced transient ST-segment elevation (type 1 Brugada pattern) in the right precordial leads at therapeutic concentrations in two patients with bipolar disorder. Lithium with-

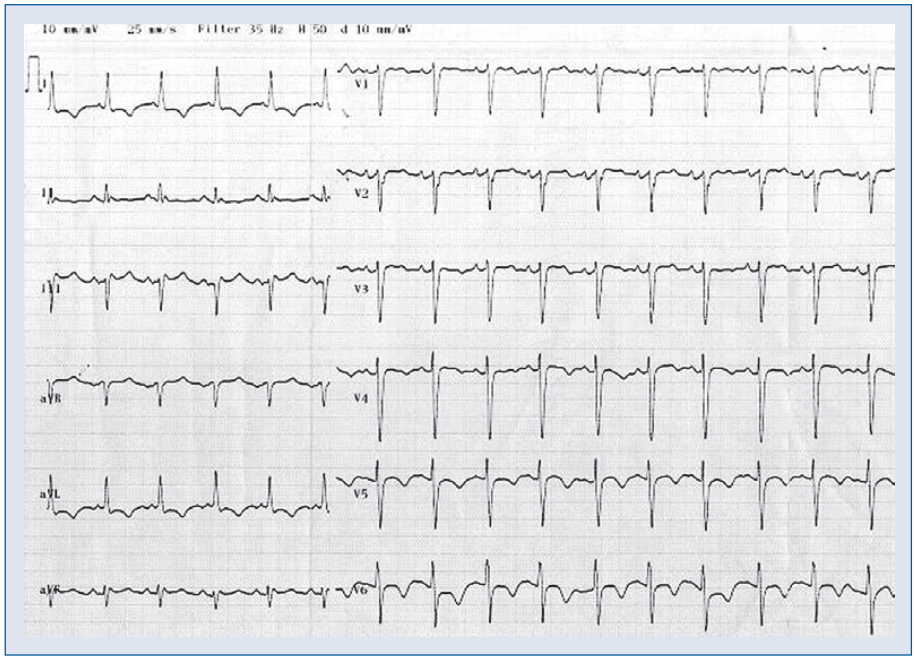

Figure 2. This electrocardiogram (ECG) represents an example of borderline sinus tachycardia with a QRS axis of about –15 degrees, T wave axis of about –30 degrees, and a P wave axis at about +60 degrees. PR interval is normal (PR < 200 ms), QT interval: 420 ms, QRS duration: 60 ms. Prolongation in S wave duration in anteroseptal leads of ECG (V1–V3) suggesting right parietal block (prolonged S wave upstroke). Parietal block, defined as a QRS duration in leads V1 through V3 that exceeds the QRS duration in lead V6 by 25 ms. There is also ST-T depression in leads 1, aVL, V5, V6, with a hint of ST elevation in leads III and aVF, and deep negative T waves are seen in apicolateral leads.

drawal in the patients resulted in reversion to type 2 or 3 Brugada patterns or resolution of ST-T abnormalities. We observed demonstrative ECG patterns in the patient who had significant ST segment elevation in the inferior leads and a wandering atrial pacemaker rhythm. To the best of our knowledge and research in the literature, a wandering atrial pacemaker due to lithium intoxication has not been reported previously.

A serum level between 0.8 mEq/L and 1.2 mEq/L is considered therapeutic, and is usually obtained with a dose of approximately 900 to 1200 mg/day [9]. Interestingly, our case was accustomed to lower doses of lithium; however, our patient developed severe clinical features despite only modestly elevated lithium concentrations. There is a large variation among patients as to what constitutes a toxic serum lithium level and it is generally recommended that elderly patients should remain at the lower end of the range for maintenance therapy. Lithium concentrations of > 5 mmol/L might be used to indicate a need for hemodialysis after acute lithium overdose, whereas a much lower treatment threshold is recommended for chronic or acute-on-therapeutic toxicity (and the clinical features are a much more important guide than blood levels for cases that involve chronic poisoning).

Our case involved an elderly patient who experienced comorbid conditions including hypertension, hypothyroidism, and diabetes mellitus. Probably the hypothyroidism was secondary to chronic use of lithium because the commonest endocrine disorder secondary to chronic toxicity is hypothyroidism. Lithium is taken up avidly by thyroid cells and blocks thyroid hormone release from thyroglobulin, which inhibits adenylate cyclase and prevents thyroid-stimulating hormone (TSH) from activating thyroid cells via the TSH receptor. It may also affect thyroid hormone synthesis. Myxedema coma has been reported as a complication of toxicity. According to our observations, it appears that advanced age and the other comorbid conditions may have contributed to the development of intoxication symptoms at the lower plasma levels and lower lithium dosages. The patient had received lithium treatment for 15 to 20 years. Age, hypertension and diabetes were also important factors which contribued to decreased creatinine clearance.

Despite the ECG changes, which suggested an acute inferior MI, we ruled this out with serial cardiac enzyme prosecutions and emergency troponin (Trp I: 0.01 ng/dL) and echocardiographic examination. Echocardiography may be especially useful

in ruling out a transmural MI via normal wall motion of the left ventricle. Because of the resolution of ST segment elevation after hemodialysis via extracorporeal elimination of lithium, we suspected that the ECG changes were associated with lithium overdose.

Hemodialysis was performed for six hours each day for four consecutive days. The efficacy of hemodialysis for resolving chronic lithium intoxication has been questioned because intracellular lithium diffuses slowly from cellular compartments [10] and 'rebound' can occur [11]. This indicates that hemodialysis is generally more effective in lithium clearance than other modalities. However, it appears that prolonged hemodialysis (> 16 h) is required in order to prevent the 'rebound' of lithium concentrations [11]. Because of the particulars of the technique, we were unable to prolong hemodialysis.

Conclusions

In conclusion, emergency physicians and cardiologists should be reminded that patients may occasionally present with ECG changes that mimic MI due to lithium toxicity, especially elderly patients. Furthermore, these ECG changes can be partially resolved after hemodialysis. These results differ considerably from other cardiac diseases.

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