

## OCCURRENCE OF VENTRICULAR ECTOPICS IN A PATIENT WITH THERAPEUTIC LITHIUM LEVEL

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### ABSTRACT

*A young male with bipolar affective disorder, mania with psychotic features was started on lithium therapy. Four days later, he developed an irregular pulse on a lithium level of 0.45 mmol/L. An ECG showed multiple unifocal ventricular ectopics. The cardiac arrhythmia disappeared when lithium was discontinued.*

*Key words : Cardiac, arrhythmia, ventricular, ectopics, lithium*

Lithium is well known to cause cardiac arrhythmias in toxic doses (Baldessarini, 1996). However, it has been known to cause cardiac conduction abnormalities within the therapeutic range, especially in the elderly (Ayd, 1995).

We report here a case of a patient in whom therapeutic level of lithium resulted in ventricular ectopics which disappeared on discontinuation of lithium.

A 34 years old male was admitted with features of a manic episode with psychotic features. He had a past history suggestive of a similar episode about four years ago. At that time, he was treated in a local hospital with chlorpromazine, lithium and carbamazepine for about 10 days. Following discharge, he remained well and discontinued all medication about two weeks later. There was no history of substance abuse. Other than a history of asthma as a child he had no physical complaints.

At the time of his admission to St. John's, his physical examination was unremarkable. Initial blood pressure recording was 130/100 mm Hg, but subsequent recordings in the ward, done regularly over the next one month were normal. His pulse rate was regular. Investigations which included haemoglobin level, total and differential counts, ESR, blood urea, creatinine, electrolytes,

blood sugar, VDRL, T<sub>3</sub>, T<sub>4</sub>, TSH levels and a ECG were normal.

He was started on lorazepam 2 mg and lithium carbonate 600 mg per day. Four days later, while taking a routine pulse and blood pressure recording, it was noted that his pulse was 88/min and irregularly irregular in rate. He was asymptomatic at this time, having no complaint of palpitations. A repeat ECG showed a heart rate of 104/min and multiple unifocal ventricular ectopics (about 24/min). 12 hour serum lithium at this time was 0.45 mmol/L. During this time, the patient was not on any other medication other than lithium and lorazepam. Lithium was discontinued while lorazepam was continued. A cardiac ECHO done one day later and an ECG done two days later were both normal. A Holter recording done two weeks later was also normal. Five days after stopping lithium, the patient was started on carbamazepine and the dose increased to 600 mg a day. After one week, lorazepam was reduced and stopped. (Two weeks after starting carbamazepine its serum level was 9.9 µg/ml). The patient recovered uneventfully and an ECG done while the patient was on carbamazepine was normal.

During outpatient review, he had an exacerbation of asthma but recovered. A repeat

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ECG was normal and serum carbamazepine level was 7.6 µg/ml. At the time of writing this report, patient is euthymic and remains well.

The issue of the patient's irregular pulse on lithium was discussed with the patient and family members. At this time, his sister, who is a nurse, mentioned that about a year ago she had taken a routine pulse and blood pressure recording on him and remembered that his pulse was irregular in rate and blood pressure recording was about 120/100 mm. She did suggest that he see a doctor about this, but he had refused saying that he had no physical complaints. Interestingly, when the patient was treated with lithium, chlorpromazine and carbamazepine four years ago, there was no mention of any irregularity in the pulse.

### DISCUSSION

The most common ECG abnormality due to lithium is reversible T wave changes. Demers and Heninger (1971) reported T wave depression, unrelated to lithium levels in all six of his study patients. He noted that a single ECG was unlikely to pick up this finding.

The most clinically significant complication of lithium therapy is stated to be SA node dysfunction - even when lithium is in the therapeutic range (Wellens et al., 1975). Rosenquist et al. (1993), reported that depressed sinus node dysfunction was significantly more in lithium treated patients when compared to age matched population.

Lithium is also known to aggravate preexisting cardiac arrhythmias, particularly in patients with heart failure in whom renal clearance of this ion is impaired (Brunwald, 1997).

There are very few references to lithium induced ventricular arrhythmias. Tangedahl and Gau, (1972) reported lithium induced premature ventricular contractions (PVCs) in a patient with no previous symptoms or evidence of cardiac disease. This patient had chest discomfort and palpitations (lithium level 0.6 mEq/L). The patient recovered when lithium was discontinued.

Tilkian et al. (1976) assessed the effect of

long term lithium treatment on cardiac arrhythmias and cardiovascular performance on 12 patients. He found that 11 patients had some ventricular arrhythmia but only one had PVCs after starting lithium and not before (measured by ambulatory ECG monitoring). He also remarked that ventricular arrhythmia (PVC, couplets and ventricular tachycardia) during exercise and in the immediate recovery period reduced in three out of four patients during lithium therapy (measured by ECG monitoring). This conflicting finding in the same group of patients was rationalised by the authors as being a result of differing methods of testing. They also noted that lithium abolished premature atrial contractions during exercise in four patients and suggested that lithium may be beneficial in controlling atrial tachycardia.

In our patient, there is a possibility that he had an irregular pulse atleast on one occasion in the past (In view of the information subsequently obtained from his sister). However, at the time of admission to our hospital his pulse rate was regular. A therapeutic level of lithium resulted in an irregular pulse and ECG abnormalities, both of which reverted to normal after lithium was stopped.

In conclusion, in a review of the literature, Brady and Horgan (1988) point out that though lithium is implicated in a variety of cardiac conduction abnormalities, the prevalence and clinical significance of cardiac arrhythmias due to lithium are still unclear. Most of the published literature is based on small number of patients and uncontrolled studies.

It is recommended that repeat ECGs are done on high risk patients on lithium (Burggraf, 1997). As our patient illustrates the point that ventricular ectopics can be asymptomatic and occur when lithium level is well within the therapeutic range, it is important to routinely monitor atleast the pulse rate in all patients after starting lithium.

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