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Multisystemic Side Effects of Lithium in Older Adults: A Case Report

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Abstract

We report a case of a 73-year-old male with bipolar affective disorder. Three years prior to this admission the patient was diagnosed with lithium induced posterior reversible encephalopathy syndrome (PRES) and lithium was discontinued. This year he presented with mania and later delirium. Investigations revealed a delayed presentation of multiple lithium-associated side effects emerging including hypercalcemia, hyperparathyroidism, and nephrogenic diabetes insipidus (NDI). Healthcare professionals should be cognizant that lithium-related side effects might trigger or exacerbate each other and may present late in the elderly. Therefore, close follow-up and clinical supervision are important for the early diagnosis and treatment of these side effects..

Keywords: *Lithium, Side effects, Bipolar affective disorder (BPAD), Posterior reversible encephalopathy syndrome (PRES), Nephrogenic diabetes insipidus (NDI)*

Introduction

Lithium is a widely used and effective treatment for mood disorders. It is one of the first treatment options for bipolar affective disorder and it has been used in modern psychiatry since 1949 [1]. Although its efficacy has been proven as a prophylactic in the relapse and recurrence of unipolar depression, hypomania, mania, short-term mortality, and suicidal risk, it has also many side effects [1,2].

Patient Information

DR is a 73-year-old male, previously diagnosed with bipolar affective disorder, who was admitted to the acute psychiatric unit for a manic episode marked by agitation, paranoia, and reduced oral intake. His past history was significant for controlled essential hypertension.

DR had a history of multiple manic episodes and had been on lithium 1200 mg per day for more than twenty years with no adverse effects. Three years ago, he was admitted to an acute medical unit with delirium and episodes of unresponsiveness. MRI revealed findings consistent with posterior reversible encephalopathy syndrome (PRES). Therefore, lithium was stopped and sodium valproate and quetiapine were initiated. He returned to baseline and received regular community follow up. In community a history of polyuria and polydipsia was noted.

His inpatient stay was complicated by reduced oral intake since the start of the manic episode and subsequent delirium. Clinical examination was unremarkable apart from confusion and signs of dehydration. He developed significant dysphagia shortly after and was declared NPO due to risk of aspiration. Despite being initially responsive to IV fluid therapy, his hypercalcaemia persisted (Table 1).

Table 1: Laboratory Values.

Serum Na+	147 mmol/L (135-145)
Serum K+	5.3 mmol/L (3.5-5.2)
Serum Cl-	109 mmol/L (95-108)
Urea	22 mmol/L (2.8-8.1)
Creatinine	134 μmol/L (53-106)
Serum Ca+	2.95 mmol/L (2.05-2.55)
Serum Osmolality	294 mmol/kg (275-295)
Urine Osmolality	199 mOsm/kg (400-1000)
Thyroid stimulating hormone	1.21 mU/L (0.27-4.20)
Parathyroid hormone	50 pg/mL (15-65)
Sodium Valproate level	45 mg/L (50-100)

Endocrinology input was sought and after investigation he was diagnosed with nephrogenic diabetes insipidus secondary to lithium. He was managed with intravenous fluid replacement via a peripheral line and was allowed to drink as desired. Delirium was managed with adjusted doses of quetiapine. After 4 weeks, he did remarkably well and blood parameters returned to normal levels with his hypercalcaemia managed by a fluids guideline of 3L/day before discharge home.

Discussion

Lithium therapy is the most common cause of nephrogenic diabetes insipidus (DI), occurring in as much as 10-15% of patients. Lithium's impact on renal function is well known, likely through several mechanisms still under exploration [3,4].

Our case had an atypical presentation that delayed treatment due to the temporal disparity between the causative medication and the presentation of symptoms. DI typically presents with marked hypernatremia and concurrent hypercalcaemia [5]. The proposed chain of events was that long term lithium induced nephrogenic diabetes insipidus that lead to parathyroid hyperplasia and hypercalcaemia, which was then compensated by his polydipsia in community. However, when he became manic, his oral intake decreased and the subsequent electrolyte imbalance led to a delirium, with a dysphagia secondary to the rising calcium that then worsened the pre-existing imbalance.

It may benefit clinicians to be aware that lithium induced DI may present slowly with significant time delay from the period of lithium treatment and symptom presentation and that the presentation can be masked by more prominent major mental health disorders. Additionally patients that may be on maintenance therapy for long periods are still vulnerable to uncommon and serious adverse events.

Conclusion

We report an elderly patient who presented with acute hypocalcaemia and dysphagia during a manic episode three years after cessation of lithium. Lithium was stopped due to PRES, which occurred after two decades of uneventful lithium therapy. Therefore, rare adverse side effects are a concern even in previously stable patients and the development of NDI is not always overtly evident after lithium therapy.

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Informed consent

Informed consent was obtained from the patient prior to publication.

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