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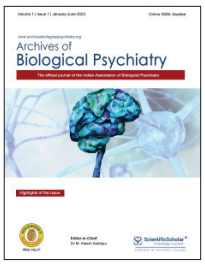

Case Report *Psychiatry*

Lithium precipitated acute kidney injury: A rare case report

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ABSTRACT

Lithium is known to cause nephrogenic diabetes insipidus, chronic tubule-interstitial nephropathy, and partial distal renal tubular acidosis. We are describing a rare instance of lithium precipitating acute renal injury within a week in a patient receiving long-term nonsteroidal anti-inflammatory drugs (NSAIDs) for medically unexplained symptoms. The patient presented with fluctuating orientation, cerebellar signs, and brisk deep tendon reflexes. Citing associated lithium toxicity discontinuation of lithium and NSAIDs brought back kidney function test to normal. Lithium toxicity was treated with intravascular fluids. We conclude that lithium may precipitate severe but reversible renal failure.

Keywords: Lithium, Nonsteroidal anti-inflammatory drugs, Acute kidney injury

INTRODUCTION

Lithium is an effective medication to treat psychiatric illnesses such as bipolar affective disorder (BPAD) and recurrent depressive disorder and has proven to decrease suicidality.[1] Lithium-ion is not metabolized in the body and is excreted through the kidney. Lithium is known to cause nephrogenic diabetes insipidus, chronic tubule-interstitial nephropathy, and partial distal renal tubular acidosis.[2] Rare case reports exist on lithium-precipitated acute kidney injury (AKI).[3,4] We present a rare case of lithium-induced AKI on long-term nonsteroidal anti-inflammatory drugs (NSAIDs) in a patient who had no reported evidence of pre-existing renal disease, which had a prompt reversal after lithium discontinuation.

CASE REPORT

A 45-year-old male presented to the outpatient department with chief complaints of decreased interaction with others, feelings of guilt, frequent crying spells, decreased sleep, multiple somatic complaints, and suicidal ideations since the past 25 days, along with lethargy and irrelevant talks which started 3 days back.

Medical history includes a major depressive episode 1.5 year back which lasted for 3 months, for which treatment details were not available and were subsequently followed by a hypomania episode which lasted for a week. The patient was receiving quetiapine 300 mg HS, lorazepam 2 mg HS, propranolol 60 mg OD, and indomethacin 75 mg OD daily. As per prior treatment records, the patient was on continuous medications. Alcohol intake was also reported, around

40–80 mL daily, abstinent for 1.5 years. Lithium was added 8 days before presentation to the outpatient department in the dose of 900 mg daily, in three divided doses.

On admission, the patient was afebrile, with blood pressure of 110/70 mm of mercury, heart rate of 72/min, respiratory rate of 20/min, and oxygen saturation of 98% on room air. The patient was not oriented to time, place, and person which was fluctuating throughout the interview. A general physical examination revealed bilateral pedal edema. The rest of the respiratory, abdominal, and cardiovascular examinations were within normal limits. Neurological examination revealed ataxic gait, Romberg's positive, past pointing in finger nose test, dysdiadochokinesia, intentional tremors, and brisk deep tendon reflex. Blood investigations revealed serum urea 83 mg/dL, serum creatinine 2.2 mg/dL, serum uric acid 8.8 mg/dL, serum potassium 5.8 mmol/lt, and serum alkaline phosphatase 191 U/L. Complete blood counts revealed hemoglobin 10.9 gm/dL while other parameters were within normal limits. The thyroid profile revealed thyroid-stimulating hormone to be 8.21 mIU/lt and serum lithium levels of 2.1 mmol/lt. Magnetic resonance imaging report had mild diffuse cerebral and cerebellar atrophy.

Naranjo scoring was applied and was found to be 5. A diagnosis of lithium-induced AKI was made and all the medications were withdrawn. Acute management of raised potassium was done in liaison with the medicine department and was given an injection of insulin with glucose and nebulization with salbutamol. The patient reported improvement in fluctuating orientation, lethargy, and tremors after 3 days of treatment except for the increased frequency of micturition which lasted for 6 days.

Kidney function test (KFT) attained normal range by the 7th day. Subsequently patient has discharged on tablet Quetiapine 25 mg HS, tablet Mirtazapine 7.5 mg HS, and tablet Lopez 1 mg BD. Lithium was not restarted again. On follow-up, the patient again presented with difficulty in sleep due to guilt about his past wrongdoings. The hospital anxiety and depression scale (HADS) came out to be 20. The patient is now being maintained on the tablet Desvenlafaxine 37.5 mg and tablet Mirtazapine 22.5 mg. He has regained his work functioning and has been on regular monthly follow-up since the past 1 year. Most recent HADS score is 9 (reduced by 55%).

DISCUSSION

Lithium-precipitated AKI is a possibility in our case with a Naranjo score of 5. A score range of 5–8 on the Naranjo algorithm is suggestive of probable adverse drug reaction which follows a reasonable temporal sequence after a drug. Lithium intoxication results from an inadvertent decrease in the renal elimination of lithium either through dehydration, hyponatremia, or drug-drug interactions. Chronic NSAID use could be another contributing factor in our case. Toxic drug interaction between lithium and NSAIDs is rare but only occurs in the setting of a strong prostaglandin inhibitor which was indomethacin in our case.[5] Although baseline renal function tests were not available, the patient had no symptoms suggestive of renal compromise before the addition of lithium. Lithium was started at a dose of 900 mg/day and serum lithium levels were 2.1 mmol/lt on 8 days which are higher than the expected level suggesting lithium toxicity which again points toward renal insufficiency. Furthermore, we agree acute on chronic renal insufficiency cannot be ruled with competence in this case. A similar case report by Jamaluddin *et al*., discusses a case of a young female diagnosed with BPAD with a family history of renal failure, who was given lithium along with NSAIDs. She showed worsening renal function after adding lithium.[3] In one more case study, it was seen that patients were already maintaining well on lithium and did not report any signs of lithium toxicity before the addition of NSAID, also their deranged KFT parameters returned to normal after prompt treatment and stopping the causative agent.[4,6]

CONCLUSION

In this case report, we want to focus on the importance of meticulous renal monitoring in cases in which a patient receives lithium along with NSAIDs, citing the risk of AKI.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Fenves AZ, Emmett M, White MG. Lithium intoxication associated with acute renal failure. South Med J 1984;77:1472-4.

2. Lavender S, Brown JN, Berrill WT. Acute renal failure and lithium intoxication. Postgrad Med J 1973;49:277-9.
3. Jamaluddin R, Ooi YT, Nazli AA. Lithium toxicity: Lessons learned. Curr Psychiatry 2023;22:e1-3.
4. Hassan S, Khalid F, Alirhayim Z, Amer S. Lithium toxicity in the setting of nonsteroidal anti-inflammatory medications. Case Rep Nephrol 2013;2013:839796.
5. Stein G, Robertson M, Nadarajah J. Toxic interactions between lithium and non-steroidal anti-inflammatory drugs. Psychol Med 1988;18:535-43.
6. Patel N, Lee C. A Case of Lithium Toxicity. Vol. 18. In: Proceedings of UCLA Healthcare; 2014.

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