Case Report

# A Rare Case of Lithium Induced Quadriparesis in a Patient with Bipolar Disorder

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## Abstract

Lithium is one of the first line drugs used for bipolar disorder associated with various side effects owing to its narrow therapeutic range. A patient of bipolar disorder stable on lithium for past fifteen years gradually developed weakness of all limbs along with facial palsy and quadriparesis. The serum lithium levels were raised. Sodium valproate was concomitantly prescribed to manage manic attacks. We report this case where lithium was responsible for the quadriparesis.

**Keywords:** Lithium, bipolar disorder, polyneuropathy, quadriparesis.

## INTRODUCTION

Lithium is a commonly used drug in the treatment of bipolar disorder. However, the treatment is associated with various side effects owing to its narrow therapeutic range.[1] It is used for bipolar disorder which affects one to two percent of the total population typically between 20 and 30 years of age.[2] National Institute for Health and Care Excellence (NICE) guidelines have recommended lithium for first-line treatment along with valproate, olanzapine and quetiapine as the most frequently prescribed maintenance treatments.[1] Facial palsy and quadriparesis are rare adverse effects associated with lithium but are highly morbid and debilitating conditions.[3] Similarly polyneuropathy is associated with lithium toxicity, but very few cases are reported without lithium toxicity.[4] Hence, this case is reported.

## CASE DESCRIPTION

A 63 year old male patient with bipolar disorder, who was stable on lithium 250 mg twice daily for the fifteen years presented to the emergency department with quadriparesis. History revealed that he

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had been developing gradual weakness of all limbs for the last two and a half years. He also had a history of forgetfulness and difficulty in maintaining posture. He had history of diabetes mellitus for the past nineteen years. ADR Monitoring Centre was notified. Causality assessment of this individual safety case report entitled 'lithium induced neurotoxicity' was done using WHO UMC causality assessment and Naranjo probability scales.[5,6] The causality assessment for association between lithium and neurotoxicity was "possible" on both WHO and Naranjo probability (score: four) scales. The severity of reaction was assessed by Hartwig adverse drug reaction assessment scale[7] which classified the adverse drug reaction as 'serious and potentially life threatening'. After evaluation he was diagnosed as a case of lithium induced polyneuropathy. Lithium was stopped. Serum lithium was has higher (1.69mEq/L) two days after stopping lithium (therapeutic serum levels of 0.6–1.2 mEq/L).

## DISCUSSION

Lithium-induced polyneuropathy is characterized predominantly by the motor symptoms and signs. Symptoms may range from slight paresis to complete quadriplegia with symmetrical involvement. Leg muscles have a propensity to be affected, albeit the weakness is generalized and more pronounced distally.[4] The patient was on sodium valproate 50 mg twice daily for four years for manic symptoms. The concomitant sodium valproate administration with lithium has been documented to elevate serum lithium levels.8 Long-lasting neurological sequelae has been reported following acute lithium poisoning.[9] Possible risk factors predisposing to high serum levels include presence of fever, concomitant use of other drugs (e.g. antipsychotics, tricyclic antidepressants and anticonvulsants), rapid correction of hyponatremia and coexistent illnesses such as hypertension, renal failure, heart failure, acute gastroenteritis and epilepsy.[10] In this case, drug-drug interaction of lithium and sodium valproate may be responsible for the presenting illness.

## CONCLUSION

Clinicians should avoid coadministration of lithium and sodium valproate. To ensure patient safety it recommended to estimate serum lithium levels periodically.


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## Conflict of Interest

There is no conflict of interest


## REFERENCES


1. Maddala RN, Ashwal AJ, Rao MS, Pad¬makumar R. Chronic lithium intoxication: Varying electrocardiogram manifestations. Indian J Pharmacol 2017;49:127-9
2. Meyer JM. Pharmacotherapy of psychosis and mania. In: Brunton L, Chabner B, KnolIman B, editors. Goodman and Gill¬man's: The Pharmacological Basis of Therapeutics. 12th edition. 2011 McGraw Hill: New Delhi, p 417-56.
3. Hayes JF, Marston L, Walters K, Geddes JR, King M, Osborn DPJ. Adverse Renal, Endocrine, Hepatic, and Metabolic Events during Maintenance Mood Stabi¬lizer Treatment for Bipolar Disorder: A Population-Based Cohort Study. PLoS Med 2016;13:e1002058.
4. Mohapatra S, Sahoo MR, Rath N. Lithium ¬Induced Motor Neuropathy: An Unusual Presentation. Indian J Pyschol Med 2016;38:252-53.

5. Naranjo CA, Busto U, Sellers EM. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther 1981;30:239-45.
6. Edwards IR, Arsonson JK. Adverse drug reactions: Definitions, diagnosis and man-agement. Lancet 2000;356:1255-9.
7. Hartwig SC, Siegel J, Schneider PJ. Pre¬ventability and severity assessment in re¬porting adverse drug reactions. Am J Hosp Pharm 1992;49:2229-32.
8. Vargas C, Tannhauser M, Tannhauser SL, Barros HM. Lithium and valproate com¬bined administration: acute behavioural ef¬fects and drug plasma levels. Pharmacol Toxicol 1996;9:87-91.
9. Banwari G, Chaudhary P, Panchmatia A, Patel N. Persistent cerebellar dysfunction following acute lithium toxicity: A report of two cases. Indian J Pharmacol 2016;48:331-33.
10. Porto FH, Leite MA, Fontenelle LF, Mar¬rocos RP, Szczerback NF, de Freitas MR. The syndrome of irreversible lithium ¬effectuated neurotoxicity (SILENT): One year follow-up of a single case. J Neurol Sci 2009;277:172-3.