

Lithium Induced Sialorrhoea: A Distinctly Curious Phenomenon

Dr. Ankit Halder (2nd Year Junior Resident)

Dr. Navna Panchami Ravindran (1st year Junior Resident)

Dr. Devavrat Harshe *(Associate Professor)

Dr. Gurudas Harshe (Professor and Head of the Department)

*Corresponding author

Department Of Psychiatry , Dy Patil Medical College Hospital And Research Institute, Kolhapur

Abstract

Sialorrhoea (drooling or excessive salivation) is a common problem encountered in neuropsychiatry patients. It can cause a range of physical and psychosocial complications, including perioral chapping, dehydration, odor, and social stigmatization, which can be devastating for patients and their families. Mood stabilizer lithium has hypersalivation as a rare side effect. Here, a 46-year-old male presented with lithium induced hypersalivation with serum lithium level being in normal range. It subsided on addition of an anticholinergic agent. Case reports in this regard are sparse. Sialorrhoea due to lithium can occur in normal as well as toxic level of serum lithium and there is no fixed duration of onset. It is a rare side effect of lithium and is a clinical dilemma like its toxicity concern. Dose reduction, anticholinergic drugs as well as in severe case stoppage of lithium therapy is also warranted to overcome it.

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I. Introduction

Psychiatric medications have significant side effects that include extrapyramidal symptoms, metabolic^[1], cardiovascular, psychosexual^[1] side effects as well as tremors, gastrointestinal (GI) disturbances, Steven Johnson's Syndrome (SJS)^[2]. Sialorrhoea is also a side effect of many psychotropic drugs^[3].

Sialorrhoea (drooling or excessive salivation) is a common problem encountered in neuropsychiatry patients. Contributing factors include hypersecretion of saliva, dental malocclusion, postural problems, and an inability to recognize salivary spill. It can cause a range of physical and psychosocial complications, including perioral chapping, dehydration, odor, and social stigmatization, which can be devastating for patients and their families^[3].

Case reports of Sialorrhoea due to lithium has not been reported widely in literature.

II. Case Report

A 46 year old male vegetable vendor was brought to the psychiatry OPD in a florid manic state. He was admitted and treated with haloperidol 10 mg, trihexyphenidyl 4 mg and lithium carbonate 600 mg, and was given modified sessions of electroconvulsive therapy (ECT). He was discharged with complete resolution of his acute manic episode. His serum lithium level at the end of first month was 0.85 mEq/l.

During the second month, he reported excess salivation during the entire day.

At his next follow up, his drug compliance was assessed, which was satisfactory. He was not on any medication that would cause excess daytime drowsiness. His serum lithium levels were 0.99 mEq/L and apart from fine tremors, no other sign of a serious adverse effect due to lithium or haloperidol was observed. There was no focal neurodeficit and no symptoms or signs suggestive of an oral or a dental pathology. He was prescribed tab glycopyrrolate 1 mg once a day for a month. Patient reported for next three follow ups, with significant reduction in sialorrhoea episodes. The patient was well maintained since the last six months on haloperidol 10 mg, trihexyphenidyl 4 mg and lithium 600 mg with no manic symptoms, minimal sialorrhoea and no other significant adverse effects.

III. Discussion

-Lithium Levels:

Sialorrhoea was reported when serum lithium level was 0.99 meq/l. This correlates with another case report by Donaldson et al.^[4] where sialorrhoea started at serum lithium level of 0.85 meq/l as well as the case report by Khalil et al.^[5] where sialorrhoea started at serum levels of 0.9 meq/l i.e in both cases the levels were below the serum level of lithium toxicity. It has been found that sialorrhoea can occur below toxic serum levels of lithium and as well as in Lithium toxicity.^[6]

-Duration of onset:

In our case sialorrhoea occurred after 1 month of starting lithium and in the case report by Donaldson et al.^[4] it started after 10 days of initiation of lithium therapy whereas in the report by Khalil et al.^[5] it started after 3 days of starting lithium. Thus it suggests that there is no fixed time gap after which sialorrhoea can be predicted following starting Lithium therapy.

-Mechanism for lithium induced hypersalivation:

1. Lithium levels in the serum are directly proportional to severity of hypersalivation.^[7] Lithium levels affect the catecholamine metabolism in the central nervous system but has no effects on the peripheral nervous system.^[7,8] The secretion of lithium ions in the saliva is known to cause persistent localized irritation.^[7,8] It is also known to trigger the central chemoreceptors in the emetic zone. Lithium is reported to produce transient rise in urinary aldosterone which increases salivation.^[7] Another possibility is the direct effect of lithium on salivary gland secretion.^[7]

Coming to the reasons in our case: -Physical examination showed no persistent localized irritation, such as a tumor or dental disease. -Patient never complained of nausea. -Patient did not complain of metallic taste -Patient did not have any other signs of Lithium toxicity .

So any other organic causes as well as lithium toxicity were ruled out

Treatment

In one case report^[4] addition of anticholinergic medication caused reduction of sialorrhoea. In other case report^[6] dose reduction resulted in remission of hyper-salivation. In the third one^[5] stoppage of lithium therapy resulted in remission of excessive salivation. In our case addition of anticholinergic agent in the form of 1 mg glycopyrrolate provided partial relief of symptoms. Dosage of lithium was unaltered.

IV. Conclusion

Sialorrhoea due to lithium is a rare and embarrassing side effect that can impair personal, social and occupational domains. It has been reported in cases where serum lithium is in normal range and may or may not be associated with clinical signs of lithium toxicity. On presentation with sialorrhoea, patient should be monitored for clinical signs of lithium toxicity and serum lithium levels should be assessed and further treatment should be planned accordingly .Both dose reduction and addition of anticholinergic agent can significantly improve sialorrhoea induced by lithium.

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