Case Report

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Severe Lithium Toxicity in an Elderly Patient: A Case Report on Management and Recovery

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Introduction

Lithium is used widely as a treatment for mood disorders [1]. Lithium with a narrow therapeutic index is very effective and powerful antimanic medication. Its pharmacodynamics and proposed mechanisms include brain inositol depletion leading to reduced responsiveness to alpha adrenergic stimulation, reducing neural responsiveness to neurotransmitters due to inhibitory effects of adenyl cyclase and G proteins vital for ion channel opening, stimulating serotonin release from hippocampus. It affects ion transport and cell membrane potential as it acts similar to potassium and sodium [2].

Lithium toxicity may be the result of accidental or intentional overdose or intoxication for several reasons, including reduced renal clearance Lithium toxicity has been divided into three patterns: acute, acute on chronic, and chronic with the latter two form being more dangerous because more time to distribute into CNS intracellular space. Mild lithium toxicity symptoms include diarrhea, mild ataxia, poor concentration, weakness, worsening tremors. With worsening toxicity patient can manifest as vomiting, development of gross tremors, slurring of speech, confusion and lethargy rare complication includes the syndrome of irreversible lithium-effectuated neurotoxicity (SILENT) involving persistent and predominantly cerebellar dysfunction [3,4]

Review of Literature

In 2014 Neil Patel and Carol Lee reported a patient of bipolar disorder with lithium toxicity presented with ECG suggestive of sinus bradycardia and T wave inversions on arrival and patient improved with sessions of hemodialysis and was discharged on day 4 [5]. Similarly, Jane Kobylianskii et al in 2021 observed a case of 54 year female known bipolar disorder with altered sensorium and lithium levels of 1.50mmol/L with ECG suggestive of normal sinus rhythm with T wave inversions and prolonged Qtc. Patients' sensorium improved with hemodialysis and recovered fully and was discharged after 1 week [6]. Dahiya B et al reported a 62-year-old bipolar male patient with syndrome of irreversible lithium effectuated neurotoxicity (SILENT) with toxic lithium levels with supportive treatment and drug modification [7].

Ahmad H. Almadani et al observed a patient on lithium with complaints of dysarthria, intermittent tremors and increased blood levels of lithium, treated in ICU with supportive treatment and discharged after 24 days of supportive treatment [8]. This current case adds to the evolving understanding by demonstrating that severe lithium toxicity can present with relatively mild symptoms initially, but may deteriorate later due to delayed renal clearance even when lithium serum levels appear deceptively low. It also reinforces the importance of clinical judgment over reliance on serum levels alone, and highlights how timely hemodialysis can lead to full recovery without long-term sequelae, even in elderly patients with multiple risk factors. This case therefore underscores the necessity for vigilant monitoring and personalized management, contributing valuable insight into the subtleties of lithium toxicity in geriatric psychiatric patients, and supporting the need for refined clinical guidelines in such complex scenarios.

Case

A 79-year-old elderly male patient, known case of mood disorder and reduced sleep for last 40 years, was brought to Emergency

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room with alleged history of consumption of 20 lithium tablets 5 days back, following which patient developed altered sensorium in form of reduced responsiveness and hypoactivity. He also had history of decreased urine output since 1 week and history of vomiting and nausea since 1 week. Past history of consumption of tablets includes Tablet Lithium300mg thrice daily and Tablet olanzapine 5mg once daily and Tablet alprazolam 0.25mg once daily at night for the last 35 years.

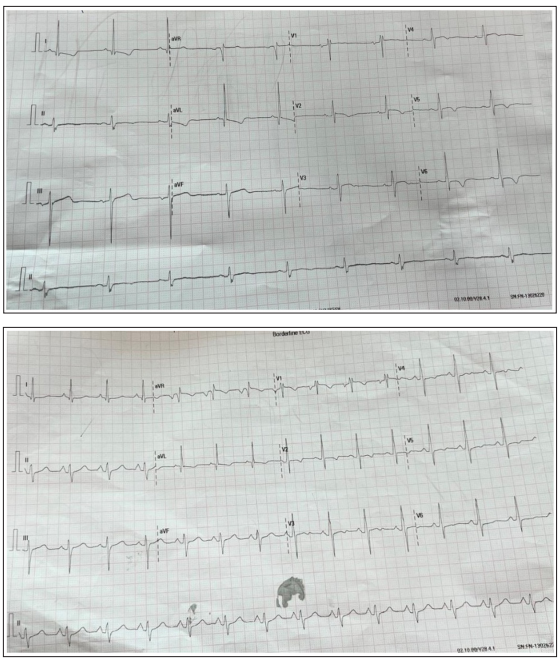
Patient was given supportive treatment at a local hospital for last 5 days and according to the attenders his condition improved in terms of responsiveness and activity but had persistent tremors of both hands and slurring of speech for last 2 days for which he was referred to tertiary care hospital. On initial assessment patient had patent airway and was communicating in one complete sentence but had slurring of speech. Respiratory rate was 18/min saturation was 99 % at room air and bilateral air entry was present, Heart rate was 86 beats per minute and blood pressure 130/80mmHg.

He had a Glasgow Coma Scale (GCS) score of 15/15 (eye-opening response (E) = 4; verbal response (V) = 5; motor response (M) = 6) with Blood sugar of 112mg/dl and bilateral pupils equal and reactive to light. He was Afebrile to touch and no injury marks or hesitation cuts were present.
Patient had normal Arterial blood gas analysis and Electrocardiogram showed normal sinus rhythm with no ischemic or arrhythmic changes. Point of care ultrasound suggestive of B/L A profile with IVC collapsing of 1cm and EF moderately reduced, with no regional wall abnormalities.

Detailed central nervous systemic examination was done MMSE scoring 26/30 with Power of 5/5, sensory system within normal limits Reflex's 2+ in bilateral upper limb and lower limb, plantar flexor and Cerebellar signs were absent like no ataxia, dysdiadokinesia, dysmetria, hypotonia, nystagmus, scanning or staccato speech, but had tremors in both hand in sitting position and other systemic examination were normal.

Patient was admitted to toxicology ward for observation, baseline investigation was sent in the form of kidney function test and liver function tests, NCCT head along with lithium levels and supportive treatment was started with iv fluids NS/RL at 70ml per hour, strict input/output charting and cardiac monitoring. Initially patient has creatinine of 1.57, BUN 54mg/dl suggested acute kidney injury as it gradually improved with fluid therapy. MRI brain was done and reported as chronic infarct in left temporal lobe. His thyroid function tests were normal.

On day 2 of admission patient had bradycardia with heart rate 53 beats per minute and repeat ECG showed sinus bradycardia with Twave inversion in V1to V6 and lead I and lead avL. Patient was shifted to intensive care unit and in view of history of lithium overdose with tremors and bradycardia with output of 750ml in last 24 hours, Hemodialysis catheter was secured in right internal jugular vein and Hemodialysis was initiated. Initially patient has creatinine of 1.57, BUN 54mg/dl suggested acute kidney injury as it gradually improved with fluid therapy.
Photo 1 ECG



Second session hemodialysis was given on day 3 following which patient heart rate returned to normal, ECG with sinus rhythm and tremors reduced and patient was better clinically.

He was further observed and was discharged after day 5 of admission after psychiatric counselling with normal speech, heart rate and no tremors.

Table 1

28/5/2024 Hb	14.2g/dl	29/5/24 Hb	11.9g/dl
Wbc	75200	WBC	5600cumm
Platelet	111000	Platelets	70000cumm
pH	7.30	Ph	7.35
Pco2	38	pCO2	39.3
pO2	25.2mmHg	pO2	29
Hco3	20.0mmHg	HCO3	20.6
Lactate	2.3mmol/l	Lactate	1.4
Glucose	132mg/dl	Glucose	142
Creatinine	1.57mg/dl	Serum creatinine	0.94mg/dl
Urea	110mg/dl	Serum urea	18.0mg/dl
Serum sodium	135mmol/L	Serum sodium	142mg/dl
Serum potassium	4.0mmol/L	Serum potasium	4.4mg/dl
Serum calcium	10.0mg/dl	Serum calcium	9.7mg/dl
serum uric acid	7.9mg/dl		
Total bilirubin	0.81mg/dl		
Direct bilirubin	0.21mg/dl		
SGOT	93.0mg/dl		
SGPT	84.0mg/dl		

ALP	178mg/dl
GGT	29.0mg/dl
TSH	1.902mcIU/ml
FT4	1.02ng/dl
FT3	2.26pg/ml

Magnesium 2.29mEq/L
USG KUB bilateral mildly raised renal cortical echogenicity
Serum Lithium 0.20 mmol/L

Discussion

Lithium is commonly used in bipolar and major depressive disorder cases but has a narrow therapeutic index and should be prescribed with utmost care to avoid toxic dosage in form of acute or even chronic poisoning.

Management of toxicity includes minimizing exposure time to toxic lithium levels by nasogastric tube placement and gastric lavage and whole bowel irrigation. There is no specific antidote for lithium detoxification, general approach to a lithium intoxicated patients includes airway management especially in case of altered mental status and intravenous hydration should be provided with isotonic saline in case dehydration signs are present followed by hemodialysis as Lithium is one of the readily dialyzable toxins [9]. Indications of hemodialysis

- Lithium serum levels greater than 4.0 mmol/l
- Lithium levels exceed concentration of 2.55mmol/l
- Patient suffers from severe signs of lithium intoxication,
- Renal impairment present,
- When there are other illness potentially deteriorating by excessive intravenous hydration [10].

In this patient the mild clinical features were evident on arrival but life-threatening manifestations started late after consumption mostly due to delayed excretion of lithium and in spite of normal lithium levels, treated with clinical judgement of symptoms and relevant history as mentioned, patient improved with hemodialysis and was discharged after a short span of hospital stay.

Acute lithium toxicity most frequently occurs during its chronic use due to the overdose or too large dose of the drug given to the person with renal failure/disorder. Sometimes it happens in patients on Lithium with lack of sodium (fever, diet, diuretic use, dehydration). It is more common in patients with underlying hyponatremia. Thus, this case report highlights the importance of the safe use of lithium include patient's selection/indication, careful screening procedure, examinations before and during lithium therapy and clinical and laboratory monitoring (including side effects).

In 2014 Neil Patel and Carol Lee reported a patient of bipolar disorder with lithium toxicity presented with ECG suggestive of sinus bradycardia and T wave inversions on arrival and patient improved with sessions of hemodialysis and was discharged on day 4 [5]. Similarly, Jane Kobylianskii et al in 2021 observed a case of 54-year female known bipolar disorder with altered sensorium and lithium levels of 1.50mmol/L with ECG suggestive of normal sinus rhythm with T wave inversions and prolonged Qtc. Patients' sensorium improved with hemodialysis and recovered fully and was discharged after 1 week [6].

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Conclusion

The extensive differential diagnosis of a patient presenting with change in mental status and neurologic symptoms should include lithium toxicity. After lithium toxicity is confirmed, management depends on the lithium level as well as the clinical presentation. Hemodialysis is the treatment of choice for severe lithium intoxication but still a cut off levels of lithium is not well defined. By respecting the principle that we should treat the patient, and not the level of lithemia, we contributed to the successful treatment, without sequelae. Lithium dosage needs to be individually tailored.

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