Archives of ISPRM 2026;1(1):92-97

Case Report

OPEN ACCESS

Neurological inpatient rehabilitation in chronic lithium toxicity: A case report

Mitchell D Evans[1], Krystal Song[1,2,3]

[1]Department of Rehabilitation Medicine, Royal Melbourne Hospital (RMH), Victoria, Australia
[2]Department of Medicine (RMH), University of Melbourne, Melbourne, Australia
[3]Australian Rehabilitation Research Centre, RMH, Victoria, Australia

Abstract

Chronic lithium toxicity frequently leads to a range of neurological sequelae. Although rare, it may pose a risk of permanent neurological disabilities. A 45-year-old female patient was admitted with a history of bipolar disorder, previously stable on lithium. She presented with confusion, worsening tremor, dysarthria, and gait ataxia. Her prolonged neurological impairments, with the rare complication of foreign accent syndrome, required an interdisciplinary rehabilitation team approach, provided by a rehabilitation physician, physiotherapist, occupational therapist, nutritional therapist, speech therapist, psychologist and social worker. The rehabilitation program consisted of muscle strengthening, balance and coordination exercises, gait retraining, functional task retraining, cognitive remediation strategies, nutritional support, targeted communication strategies, psychosocial and psychiatric support to assist her transition toward community reintegration. In conclusion, timely delivery of neurological rehabilitation interventions is of utmost importance in helping maximize the patient's functional independence, psychosocial outcomes and quality of life.

Keywords: Ataxia, foreign accent syndrome, interdisciplinary rehabilitation, lithium toxicity, neurotoxicity.

Lithium is commonly used as a mood stabilizer for bipolar disorder since being introduced into therapeutic clinical practice in 1949.[1] Due to its narrow therapeutic index, it frequently causes toxicity and various neurological sequelae including confusion, lethargy, ataxia, tremor, myoclonus, dysarthria, psychological changes, seizures and coma.[2,3] The common risk factors for lithium neurotoxicity include older age, abnormal thyroid function, impaired renal function and arginine vasopressin V2 resistance (previously nephrogenic diabetes insipidus).[4,5] In this article, we report a 45-year-old female patient with chronic lithium toxicity and discuss the impact of neurological manifestations upon function and societal participation and the importance of an interdisciplinary neurorehabilitation approach toward disability management.

CASE REPORT

A 45-year-old female patient presented to the emergency department with progressively decreasing level of consciousness, confusion, worsening tremor, dysarthria, gait ataxia, vomiting and diarrhea. Her psychiatric history included bipolar affective disorder and generalized anxiety

Submitted: January 12, 2026
Accepted: January 30, 2026
Published: February 24, 2026

Correspondence: Mitchell Evans. MD. Department of Rehabilitation Medicine, Royal Melbourne Hospital, Royal Park Campus, Parkville, Victoria, Australia.

E-mail: mitchell.evans@mh.org.au

Doi: https://doi.org/10.5606/archisprm.2026.12

Citation:
Evans MD, Song K. Neurological inpatient rehabilitation in chronic lithium toxicity: A case report. Arch ISPRM 2026;1(1):92-97. https://doi.org/10.5606/archisprm.2026.12.

© 2026 The Author(s). This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (CC BY-NC-ND 4.0), which permits non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited and is not modified. https://creativecommons.org/licenses/by-nc-nd/4.0/

disorder and the patient was under treatment with venlafaxine, mirtazapine and lithium 1,575 mg daily for the past nine years. Her medical history consisted of migraine and hypothyroidism. Four months prior to hospital admission, the patient developed bilateral tremors and was initially treated with low-dose gabapentin at the discretion of her general practitioner without significant clinical improvement. Over time, her tremors progressively worsened and accompanied by functional decline, increasing drowsiness, and cognitive-communication and swallowing difficulties. Within the two weeks before admission to hospital, she attended two medical follow-up visits due to worsening tremors and reduced oral intake; however, serum lithium levels were not assessed. There was no prior history of lithium toxicity, substance abuse or intentional overdose.

On initial presentation, the patient was severely dehydrated and had a Glasgow Coma Scale (GCS) score of 10 with an intact airway. She was hypotensive (98/59 mmHg) and had an oxygen saturation of 98% on room air, with a normal heart rate, respiratory rate and temperature. Her neurological examination demonstrated bidirectional lateral nystagmus, dysarthria with unintelligible words, coarse tremors and hyperreflexia of bilateral upper and lower extremities. Assessment of coordination could not be performed due to somnolence and confusion.

An electrocardiogram revealed bradycardia and QTc prolongation. The lithium concentration level was significantly elevated at 5.3 mmol/L (0.6 to 1.2 mmol/L). Additional laboratory investigations demonstrated acute kidney injury (glomerular filtration rate 22 mL/min/1.73 m^2, creatinine 222 μmol/L) and elevated white cell count of 16.4×10^9 /L (4.0 to 12.0 × 109 /L). Thyroid function test result was unremarkable, with a thyroid stimulating hormone level of 0.71 mU/L (0.35 to 4.94 mU/I). An initial cranial computed tomography (CT) showed no acute intra-cranial pathology.

The patient was admitted to the intensive care unit for four days for treatment of lithium toxicity. She required central venous catheter insertion, intravenous fluid resuscitation with normal saline, nasogastric tube placement, and intermittent hemodialysis over two days until serum lithium levels normalized. The consultation-liaison psychiatry team was involved early in the patient's care and recommended temporary cessation of all psychotropics until resolution of her delirium. Although her lithium serum levels decreased, the patient demonstrated ongoing neurological deficits including tremors, cognitive impairment, dysarthria, dysphagia and gait ataxia in the context of a prolonged delirium state. The patient also had communication deficits including word-finding difficulties, ataxic dysarthria and a new foreign American accent. A cranial magnetic resonance imaging (MRI) was performed to evaluate for SILENT syndrome (Syndrome of Irreversible Lithium Effectuated Neurotoxicity); however, no abnormalities were identified. A persistent peripheral neuropathy including reduced light touch sensation was investigated with nerve conduction studies which demonstrated length dependent sensorimotor axonal neuropathy (Table 1).

Interdisciplinary rehabilitation

Following the resolution of her initial delirium, the patient had persisting upper limb tremors, gait ataxia, fatigue, cognitive deficits (reduced attention, organization, planning, sequencing, and insight), oropharyngeal dysphagia (requiring initial nasogastric feeding), and communication impairments (word-finding difficulties, foreign American accent, and dysarthria), which affected her ability to walk and perform her activities of daily living independently. The patient initially required two-person assistance with her transfers and mobility up to 20 m, limited by impulsivity and difficulty following instructions. Her initial Revised Functional Autonomy Measurement Scale (SMAF-R) total adjusted score was -57/-87 on Day 15 (Table 2), indicating her dependence for most functional tasks.

The patient underwent inpatient rehabilitation after two weeks following her initial hospital presentation. An interdisciplinary rehabilitation approach was employed toward addressing her impairments, activity limitations and participation restrictions. On average, she received a physiotherapy one-hour session five days a week tailored to her symptoms and fatigue levels.

Table 1. Nerve conduction studies demonstrating length dependant, sensory more than motor, non-specific axonal neuropathy

Motor NCS

	Nerve/Sites	Rec. Site	Lat. ms	Amp.1-2 mV	Dist cm	Dur. ms
R median - APB						
Wrist	APB	3.65	8.7	7		6.29
Elbow		7.79	8.1	23	55.5	6.46
R common peroneal - EDB						
Ankle	EDB	4.46	1.3			7.79
FibHead		12.33	1.1	30.8	39.1	8.15
Knee		14.38	1.0	7.8	38.2	8.10
L common peroneal - EDB						
Ankle	EDB	4.44	1.2			6.42
FibHead		12.90	1.0	32	37.8	6.71
R tibial malleolus - AH						
Ankle	AH	3.81	8.0			6.31
Knee		13.77	6.8	38	38.2	7.90
L tibial malleolus - AH						
Ankle	AH	4.73	6.5			6.42

F Wave

Nerve	Fmin ms
R Common peroneal	54.95
R Tibial malleolus	53.70
L Common peroneal	54.43
L Tibial malleolus	55.31
R Median	28.07

Sensory NCS

Nerve/sites	Rec. Site	Onset ms	Peak ms	Amp.2-3 µV	Amp.1-2 µV	Dist. cm	Vel. m/s
R leg sensories							
Sural	Lat Mall	NR	NR	NR	NR		
Superficial peroneal	Ankle	NR	NR	NR	NR		
L leg sensories							

Nerve/Sites

Nerve/sites	Rec. Site	Onset ms	Peak ms	Amp.2-3 µV	Amp.1-2 µV	Dist. cm	Vel. m/s
Sural	Lat Mal	NR	NR	NR	NR		
Superficial peroneal	Ankle	NR	NR	NR	NR		
R radial - antidromic							
Forearm	Snuff box	1.96	2.67	11.1	12.0	10	51.1
R antidromic sensories							
Median wrist	Dig II	2.58	3.52	20.0	12.5	13	50.3
Ulnar wrist	Dig V	1.92	2.69	25.8	19.8	11	57.4

NCS, nerve conduction studies; APB, abductor pollicis brevis; EDB, extensor digitorum brevis; AH, abductor hallicus; NR, no result.

Table 2. Pre- and post-rehabilitation outcome measurement scores from day of initial hospital admission

	Day 15	Day 23	Day 32	Score range	Interpretation
SMAF-R	–57	-	–3	0 to -87	A SMAF-R score closer to 0 indicates greater functional autonomy.
FGA	-	14	25	0 to 30	A FGA score below 15 indicates severely impaired balance.
FIM	39	-	113	18 to 126	A greater FIM score indicates greater functional independence.
FAB	-	-	17	0 to 18	A lower score indicates worsening frontal lobe dysfunction.

SMAF-R Revised Functional Autonomy Measurement System; FGA: Functional gait assessment; FIM: Functional independence measure; FAB: Frontal assessment battery.

Her Functional Gait Assessment (FGA) including balance and postural stability assessment, scored 14/30 on Day 23 which indicated severely impaired balance. These findings correlated with The Clinical Test of Sensory Interaction on Balance (CTSIB) measure, where the patient demonstrated forwards and backward postural sway and inability to tandem walk. The focus of physical therapy was, thus, targeted at coordination, balance and gait retraining. These exercises included static exercises targeting her postural sway and multiple sets of dynamic exercises with tandem walking, backward walking, hurdles and side stepping in rails. The patient also received functional task retraining to optimize her independence with activities of daily living.

Other core components of her rehabilitation program included cognitive rehabilitation focusing on task sequencing and planning, nutritional optimization, sleep hygiene strategies, targeted communication strategies with drill and vowel tasks for functional phrases, falls prevention strategies, energy conservation strategies, equipment prescription and aids, and psychological support for adjustment issues during her illness trajectory. The consultation-liaison psychiatry team was involved throughout the patient's hospitalization, including inpatient rehabilitation stay.

After one month of inpatient rehabilitation, the patient demonstrated clinical and functional improvements. She was more alert, orientated to her surroundings and demonstrated cognitive improvement, although she had residual self-reported short-term memory impairment. During her program, the patient developed low mood and emotional dysregulation, which was treated with low-dose, slow-release quetiapine. In collaboration with the psychiatry team, it was decided that the patient would continue community-based treatment for her bipolar affective disorder and would not resume lithium or her previous psychotropics. She experienced less tremors, reduced bidirectional nystagmus and improved signs of peripheral neuropathy. She was eventually transitioned to full ward diet and thin fluids, with improved oral intake. She demonstrated improvements in balance, gait and speech intelligibility. Her foreign American accent reverted to her native accent. She made functional gains, improving in her Functional Independence Measurement (FIM) scores from admission (39) to discharge (113), discharge SMAF-R score -3/-87 (independent), FGA of 25/30 and Frontal Assessment Battery (FAB) score of 17/18 (Table 1). Upon discharge from inpatient rehabilitation, she was independent with her transfers, unaided mobility up to 100 meters and was independent with stairs. She was independent in all of her personal care tasks, medication management and light domestic activities of daily living. A written informed consent was obtained from the patient.

DISCUSSION

Chronic lithium toxicity can cause various neurological sequelae including confusion, lethargy, ataxia, neuromuscular excitability and dysarthria, and is associated with longer length of hospital stay compared to an acute overdose.[3,4] While the patient was on a stable lithium dose, she had poor oral intake and vomiting before hospitalization, likely precipitating an acute kidney injury, which contributed to the worsening of chronic lithium toxicity and various neurological effects. Although the patient had

medical comorbidities (anxiety, migraine, and hypothyroidism) that could been potential contributing factors, these were excluded clinically. The potential complication of SILENT syndrome was explored in this patient. In general, SILENT syndrome presents with manifestations of cerebellar and cognitive dysfunction, with MRI scan commonly showing cerebellar gliosis and atrophy.[6] This was not demonstrated on cranial MRI scans of our patient. Although the foreign accent syndrome (FAS) that the patient demonstrated has not been reported as a complication of chronic lithium toxicity, it can occur with other neurological disorders such as stroke.[7] Foreign accent syndrome is often seen in anatomically heterogeneous brain lesions affecting cortical and subcortical regions (left superior temporal and medial frontal structures, bilateral subcortical structures, thalamus, left cerebellum), representing areas critical to motor speech control.[8] The disruption of this speech motor network can result in changes to articulation and prosody that can be perceived as foreign accent.[7] The phenomenon of FAS represents a complex neuropsychiatric diagnosis with a high comorbidity profile, underscoring the need for an interdisciplinary rehabilitation approach involving speech therapy and psychological support.[9]

From a rehabilitation perspective, there have only been rare case reports documenting the potential benefits of rehabilitation in patients with acute or chronic lithium toxicity.[9,10] To date, there is currently a lack of controlled studies and available evidence-based clinical guidelines for rehabilitation approaches in the management of patients with acute or chronic lithium toxicity, making treatment approaches more challenging. This case report adds valuable data to the existing literature, highlighting the importance of having timely access to neurological rehabilitation interventions in helping maximize this patient's functional independence, psychosocial outcomes and quality of life.

In conclusion, while lithium is a medication that has been used for more than seven decades, its mechanism of action still remains to be elucidated. This report highlighted the various neurological manifestations of lithium toxicity, including the rare case of FAS. To the best of our knowledge, this is one of the few case reports of chronic lithium toxicity that outlines the importance of interdisciplinary rehabilitation in maximizing functional, cognitive, communication, psychosocial and quality of life outcomes.

Declaration of Conflicting Interests

The authors declare that there are no conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

M.D.E., K.S.: Designed research, wrote the paper; M.D.E.: Had primary responsibility for final content. All authors read and approved the final manuscript.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

AI Disclosure

The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

REFERENCES

1. Geddes JR, Miklowitz DJ. Treatment of bipolar disorder. Lancet 2013;381:1672-82. doi: 10.1016/S0140-6736(13)60857-0.
2. Ott M, Stegmayr B, Salander Renberg E, Werneke U. Lithium intoxication: Incidence, clinical course and renal function - a population-based retrospective cohort study. J Psychopharmacol 2016;30:1008-19. doi: 10.1177/0269881116652577.
3. Dennison U, Clarkson M, O'Mullane J, Cassidy EM. The incidence and clinical correlates of lithium toxicity: A retrospective review. Ir J Med Sci 2011;180:661-5. doi: 10.1007/s11845-011-0712-6.
4. Oakley PW, Whyte IM, Carter GL. Lithium toxicity: An iatrogenic problem in susceptible individuals. Aust N Z J Psychiatry 2001;35:833-40. doi: 10.1046/j.1440-1614.2001.00963.x.
5. Hlaing PM, Isoardi KZ, Page CB, Pillans P. Neurotoxicity in chronic lithium poisoning. Intern Med J 2020;50:427-32. doi: 10.1111/imj.14402.

6. Marmol S, Beltre N, Margolesky J. Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT): A preventable cerebellar disorder. Cerebellum 2024;23:1733-5. doi: 10.1007/s12311-024-01668-z.
7. Higashiyama Y, Hamada T, Saito A, Morihara K, Okamoto M, Kimura K, et al. Neural mechanisms of foreign accent syndrome: Lesion and network analysis. Neuroimage Clin 2021;31:102760. doi: 10.1016/j.nicl.2021.102760.
8. McWhirter L, Miller N, Campbell C, Hoeritzauer I, Lawton A, Carson A, et al. Understanding foreign accent syndrome. J Neurol Neurosurg Psychiatry 2019;90:1265-9. doi: 10.1136/jnnp-2018-319842.
9. Nobematsu A, Wakabayashi H, Hanada T, Watanabe N, Tachibana K. Post-acute rehabilitation for Ataxia Associated with acute lithium toxicity: A case report. Prog Rehabil Med 2018;3:20180010. doi: 10.2490/prm.20180010.
10. Motru B, Iyer D. Syndrome of irreversible lithium induced neurotoxicity due to chronic lithium use (SILENT) - Effect on functional status - a case report. Australas Med J 2018;17:1217-9. doi: 10.21767/AMJ.2024.4031.