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Case Report
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New Definition of Irreversibility in Neurological Side Effects of Lithium Intoxication: ACase Report

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Abstract

Background: Lithium carbonate is a commonly used drug in psychiatric therapy with frequently associated neurological side effects following intoxication. Syndrome of irreversible lithium-effectuated neurotoxicity (SILENT) is defined as neurological sequels induced by lithium toxicity persisted for at least 2 months after the discontinuation of the drug in the absence of previous neurological impairment. There are few reports of delayed improvement of neurologic signs following lithium toxicity but to our knowledge it is the first case of severe lithium toxicity who have had a more protracted course of neurological improvement even after 2 years.

Case presentation: A 60-year-old man with history of bipolar disorder and regular use of 1500mg/day lithium carbonate admitted to our hospital with progressive loss of consciousness and imbalance 6 days after a mild head trauma. Lab data revealed serum lithium level of 8.3mEq/L which decreased to 0.8mEq/lit after 2 dialysis sessions. Although severe disabling cerebellar signs were prominent during the first month after intoxication, he has experienced a protracted improvement course which is continuing even after 2 years.

Conclusion: The underlying mechanism of reversibility is suggested to be complete remyelination after the initial demyelinating insult and in the case of incomplete remyelination or degeneration, neurological deficits would be persistent. We suggest that the diagnosis of "SILENT" should be used more cautiously and be limited to chronic cases (at least 12months after intoxication) or in existence of obvious atrophy.

Keywords: Lithium;Intoxication;Reversibility;Demyelination; Creatinine

Abbreviations: SILENT: Syndrome of Irreversible Lithium-Effectuated Neurotoxicity; MRI: Magnetic Resonance Imaging; BUN: Blood Urea Nitrogen; Cr: Creatinine

Introduction

Lithium carbonate is a commonly used mood stabilizer with high possibility of neurological side effects following intoxication[1].

Although the neurological adverse effects are generally reversible, there are reports of long-lasting neurological sequels after 2 months that is called the syndrome of irreversible lithium-effectuated neurotoxicity (SILENT). The persistent neurological symptoms and signs are mostly related to cerebellar dysfunction which causes severe disability[2-4].

In this paper we have reported a patient with lithium toxicity after head trauma who have had a slowly remitting course of cerebellar symptoms even after 24 months.

Case Report

A 60-year-old man presented to our hospital with progressive loss of consciousness and imbalance for 3 days before. He had been diagnosed with bipolar disorder for 15 years and was on regular use of 1500mg/day lithium carbonate and 1mg/day clonazepam. He had a history of head trauma following car accident, with a brief loss of consciousness for about 5 minutes, 6 days before admission, which since then, he had experienced headache, vertigo and vomiting, despite normal brain computerized tomography scan. After 3 days, he gradually experienced progressive lethargy and imbalance. At the time of admission, he was febrile (oral temperature:38.1˚C) and dehydrated. Neurological examination revealed Glasgow Coma Scale of 11/15, dysarthria with slurred and scanning speech, generalized weakness, incoordination and increased deep tendon reflexes.

Serum laboratory tests showed elevated blood urea nitrogen (BUN:88 mg/dL), creatinine (Cr: 2.8mg/dL) and also serum lithium level of 8.3mEq/L (therapeutic range, 0.5-1.0mEq/L). Other metabolic and electrolyte tests were reported to be

normal and blood and urine cultures were negative. Brain Magnetic Resonance Imaging (MRI) showed no abnormal finding. Body temperature returned to normal after rehydration and a short course of antibiotic therapy. The patient underwent dialysis in day 2 and 4 after admission and serum lithium level decreased to 0.8mEq/lit after the second dialysis session. During the first month after intoxication a clear improvement in his level of consciousness was observed. However, severe disabling cerebellar signs became prominentwith SARA score [5] of 38. Follow up examinations after rehabilitation therapy in month 6,12,18 and 24 following intoxication revealed SARA scores of 33, 27, 20 and 17. Repeated brain MRIs, 6 and 12 months later were normal without any evidence of cerebellar atrophy.

Discussion

Although lithium is suggested to have neurotrophic and neuroprotective properties by reversing pathophysiological changes such as increased oxidative stress, apoptosis, inflammation and excitotoxicity [6,7] long-lasting neurological sequels occasionally occur following lithium intoxication. Common predisposing factors for lithium-induced neurotoxicity are acute infection, hyperthermia, hyponatremia, renal failure, drug interactions and dehydration, as vomiting induced dehydration was the underlying factor in our patient.

Ataxia, tremor, dyskinesias, dysarthria, hyperreflexia and muscle weakness are reported in mild lithium intoxication [8,9] while higher levels accompany with encephalopathy, severe cerebellar dysfunction, seizures and coma. The most common manifestation of persistent neurological dysfunction is cerebellar symptoms which as in our case, usually become apparent after the improvement of level of consciousness or remission of the encephalopathic state[2,3]. SILENT is defined as neurological symptoms induced by lithium toxicity persisted for at least 2 months after the discontinuation of the drug in the absence of previous neurological impairment [2]. But delayed recovery from neurological sequels is rarely reported with a more protracting course[10]. The mean plasmatic lithium levels in several cases of SILENT reported in the literature is found to be 2.29±1.67mEq/L[2]. In our patient, despite the severe neurological symptoms and high serum level of lithium which is suggested to be a risk factor of irreversibility[10], improvement of cerebellar signs has been continued in a more prolonged mode even after 2 years.

In a systematic review of SILENT cases Adityanjee et al [2]. have suggested demyelination as a probable mechanism for persistent neurological signs. It seems that in the case of mild demyelination or early remyelination, neurological symptoms rapidly improve. However, in setting of incomplete remyelination and occurrence of cerebellar degeneration and atrophy, persistent neurological deficits happen.Overall, due to possible elongated duration of remyelination process and reports of reversible neurological deficits in much more than 2 months after intoxication, we suggest that the diagnosis of "SILENT" should be used more cautiously and be limited to chronic cases (at least 12 months after intoxication) or in existence of obvious atrophy.

References

1. Timmer RT, Sands JM (1999) Lithium Intoxication. J Am SocNephrol 10(3): 666-674.
2. Adityanjee, Munshi KR, Thampy A (2005) The syndrome of irreversible lithium effectuated neurotoxicity. ClinNeuropharmacol 28(1): 38-49.
3. Niethammer M, Ford B (2007) Permanent lithium-induced cerebellar toxicity: three cases and review of literature. Mov Disord 22(4): 570-573.
4. Porto FH, Leite MA, Fontenelle LF, Marrocos RP, Szczerback NF, et al. (2009) The Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT): one-year follow-up of a single case. J Neurol Sci 277(1-2): 172-173.
5. Schmitz-Hübsch T, du Montcel ST, Baliko L, Berciano J, Boesch S, et al. (2006) Scale for the assessment and rating of ataxia: development of a new clinical scale. Neurology 66(11): 1717-1720.
6. Marmol, F (2008) Lithium: bipolar disorder and neurodegenerative diseases, possible cellular mechanisms of the therapeutic effects of lithium. ProgNeuropsychopharmacol Biol Psychiatry 32(8):1761-1771.
7. Machado-Vieira R, Manji HK, Zarate CA Jr (2009) The role of lithium in the treatment of bipolar disorder: convergent evidence for neurotrophic effects as a unifying hypothesis. Bipolar Disord 11(Suppl 2): 92-109.
8. Jaeger A (2007) Lithium. Medicine 35: 535-6.
9. Sadosty AT, Groleau GA, Atcherson MM (1999) The use of lithium levels in the emergency department. J Emerg Med 17(5): 887-891.
10. Tesio L, Porta GL, Messa E (1987) Cerebellar syndrome in lithium poisoning: a case of partial recovery. J NeurolNeurosurg Psychiatry 50(2): 235.

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