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 sequelae 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
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**