Lithium-Induced Motor Neuropathy: An Unusual Presentation

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ABSTRACT

Peripheral neuropathy secondary to lithium is under-recognized. Most cases of polyneuropathy were reported with lithium intoxication. However, very few cases were reported without lithium toxicity. We present a case of motor neuropathy due to the use of lithium in a 26-year-old male with a therapeutic lithium level.

Key words: Lithium, motor, neuropathy

INTRODUCTION

Lithium has been used for over 60 years for treatment of various psychiatric disorders. Though a large number of pharmacological agents are now available for treatment of bipolar disorder in particular, but lithium continues to be the most effective and best-tolerated treatment option for many patients. Its use is sometimes limited by its narrow therapeutic index, and it has been reported that over 75% of patients on long-term lithium therapy will experience some form of toxicity.[1] The overall incidence of side effects on the nervous system ranges from 35% to 50%, with lithium serum levels within the therapeutic range.[2] Most cases of polyneuropathy were reported with lithium intoxication.[3,4] Very few cases of peripheral nervous system damage secondary to lithium (with normal serum lithium levels) are reported.[5] We present a case of motor neuropathy due to the use of lithium in a 26-year-old male with a therapeutic lithium level.

CASE REPORT

A 26-year-old Hindu male reported to psychiatry outpatient department with a complaint of difficulty in walking, weakness of both lower extremities from last 1-month. He was receiving treatment from our institution since last 2 years with a diagnosis of the bipolar affective disorder. He was maintained on lithium carbonate 800 mg and olanzapine 10 mg/day from last 14 months. He was euthymic since last 3 months. Hence, he was maintained only on lithium 800 mg/day from last 3 months. On examination, the patient was conscious, oriented, power and tone of both lower limbs reduced, deep tendon reflexes were reduced and plantar responses remaining flexor. No other abnormal finding was elicited on physical examination. Routine laboratory tests including liver functions test, serum B12 and folate assays, thyroid function tests, serum
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electrolytes, electroencephalogram, magnetic resonance imaging brain were within normal limits. Serum lithium level was 0.84 mEq/L. Nerve conduction studies revealed large fiber axonal degeneration predominantly motor type. He had no previous medical history of hypertension, diabetes, heavy metal exposure, alcohol intake, epilepsy, neurological deficits, or any other drug intake. No features consistent with lithium toxicity, that is, confusion and lethargy, slurred speech, urinary incontinence, worsening tremor were present. There is no family history of any neuropsychiatric illness. The lithium carbonate was discontinued. He was started on quetiapine 400 mg/day. After a stoppage of lithium, the patient improved gradually within next 4 weeks. Over the next 6 weeks difficulty in walking, weakness of both lower extremities disappeared completely. There was no deterioration in affective symptoms.

DISCUSSION

In lithium-induced neuropathy, the motor symptoms and signs are predominant. They vary from slight paresis to complete quadriplegia. Both upper and lower limbs are symmetrically involved. Although the weakness is generalized and most pronounced distally, proximal leg muscles can preferentially be affected. Acute denervation activity with larger motor units and higher fiber density later on, together with only moderately slowed nerve conduction velocities indicate axonal neuropathy.\cite{3,6} A possible mechanism of lithium-induced neuropathy would be an intracellular accumulation of lithium and interference with the propagation of action potentials.\cite{7} Lithium-induced neuropathy should be distinguished from neurological features associated with lithium intoxication. In lithium intoxication muscle tone is usually increased, tendon reflexes are hyperactive, Babinski sign usually present, fasciculations and paresthesias may occur, but the sensation is normal. Previously most cases of lithium-induced neuropathy are reported in association with lithium toxicity. In our patient, only motor nerve fibers of both lower limbs are involved, and neuropathy is elicited with a therapeutic lithium level. Hence, peripheral neuropathy should be looked for in all patients on chronic lithium therapy. A systematic clinical search for symptoms and signs as well as an electrophysiological search for subclinical neuropathy in patients on lithium therapy is needed.

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Conflicts of interest
There are no conflicts of interest.

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