Case Report

Lithium-induced Thyroiditis in a Patient Having Bipolar Affective Disorder - A Rare Case Report

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ABSTRACT

A 22-year-old female, previously diagnosed with bipolar affective disorder on lithium therapy, presented to us with manic symptoms. The blood investigations revealed elevated thyroxine and thyroid peroxidase antibodies and reduced thyroid-stimulating hormone with poor tapping function of thyroid on technetium thyroid scintigraphy indicating lithium-induced thyroiditis.

Key words: *Bipolar affective disorder, lithium, thyroiditis*

INTRODUCTION

Lithium is the gold standard agent used in bipolar affective disorder. One of the clinical adverse effects of lithium is goiter in up to 40% due to inhibition of thyroid hormone secretion in significant number of patients receiving the drug for treatment of bipolar disease.[1] Clinically, there is thyroid enlargement and low triiodothyronine (T3), thyroxine (T4) levels. Cases of hyperthyroidism have also been associated with its treatment but are rare compared with hypothyroidism and goiter. Silent thyroiditis has been rarely associated with lithium therapy. The following case presents silent thyroiditis where serum T4 was elevated with low thyroid-stimulating hormone (TSH) value.

CASE REPORT

Ms L, a 22-year-old single female, educated till the 12th standard is a known case of bipolar affective disorder for 17 years of age with two episodes of mania in the years 2013 and 2015. She was maintaining well on lithium (800 mg/day). Serum lithium levels and thyroid function tests were within normal limits even 3 months before the current episode. She had a family history of hypothyroidism in maternal grandmother who was on thyroid supplementation.

In April 2017, she was brought to the psychiatry department by her parents with a history of increased activity, reduced sleep, over-talkativeness, and anger outbursts toward family members for 1 week. After admission, detailed assessment was done. On physical examination, proptosis was evident. Mild lid lag was present. She had a fine tremor of hands and warm extremities. There was an increased volume in her speech and her affect was irritable. However, she

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had normal goal-directed psychomotor activity and cognitive functions were intact. She had grade 2 insight. The clinical features were suggestive of manic episode. On initial investigations, her serum electrolytes, hemogram, hepatic, and renal function tests were normal. Serum lithium quantitative analysis was done and levels were within therapeutic range (0.65 mEq/L). Her serum T4 was elevated (3.10 ng/dl) and TSH was low (0.0049uIU/ml). Endocrine consultation was sought, and thyroid peroxidase antibody was evaluated, which showed elevated values (594.80 U/ml). Technetium thyroid scintigraphy was suggested for further evaluation. Poor tapping function of the thyroid gland was evident in scintigraphy, and a diagnosis of iatrogenic thyroiditis was made by the endocrinologist. Propranolol 40 mg was started by the endocrinology department for hyperthyroidism. She was continued on lithium 800 mg/day as mood stabilizer and amisulpride 200 mg for control of acute manic symptoms. By discharge 10 days later, the patient had improved significantly in regard to the manic symptoms.

In the subsequent follow-up after 1 month, the results of her thyroid function tests were as follows T3, 4.90 ng/dl (1.31–3.71); TSH, 0.0018 μIU/ml serum (0.35–4.9); and thyroid peroxidase antibody (TPO) 594, which were suggestive of a diagnosis of iatrogenic thyroiditis (lithium induced). The patient was started on carbimazole 10 mg by the endocrinology. She was continued on lithium 800 mg, amisulpride 200 mg, and propranolol 40 mg. Her TPO levels reduced after initiation of carbimazole therapy. In the subsequent review after 3 months, her TPO levels normalized.

DISCUSSION

Lithium is effective in acute mania and in prevention of recurrence. The main indication for lithium is in prophylaxis of bipolar affective disorder where it reduces both the number and severity of relapses. The minimum effective plasma level for prophylaxis is 0.4 mmol/L with optimal range being 0.6–0.75 mmol/L. The drug has multiple effects on thyroid function. Lithium blocks the release of thyroid hormone from the thyroid gland which leads to goiter and hypothyroidism. The reported prevalence of hypothyroidism is estimated to be about 20%.[2,3] There have only been sporadic cases of hyperthyroidism among the patients treated with lithium.[3,4] Lithium-associated thyroiditis has rarely been described in the literature.[1,5] In the clinical setting, thyroiditis after initiating lithium therapy is rare, and only three cases of lithium-associated thyroiditis and four cases of autoimmune thyroiditis have been reported.[6] Lithium induces sporadic thyroiditis by direct toxic effects. It directly damages thyroid cells, with consequent release of thyroglobulin and thyroid hormones into the circulation; therefore, thyrotoxicosis caused by silent thyroiditis might be associated with lithium use.[1]

The differential diagnosis for hyperthyroidism included Grave's disease. The classical scintigraphy pattern of Grave's disease is homogeneous increase in radiotracer uptake in both lobes of the thyroid gland. In contrast, the distribution pattern of radiotracer is reduced in silent thyroiditis where the follicles are disrupted suggestive of iatrogenic thyroiditis (lithium induced). Radioactive iodine uptake is high in Grave's disease and low in thyroiditis. Histologically, painless thyroiditis resembles autoimmune thyroiditis, but according to Mizukami *et al*.[4] stromal fibrosis and Hurthle cells are rare in the former.

Before initiating prophylaxis with lithium in bipolar affective disorders, the assessment of thyroid function is recommended for all patients; the assessment should include plasma levels of TSH and antithyroid antibodies.[7] Determination of free T4 should be added in cases in which TSH is suppressed (to rule out central hyperthyroidism or hypothyroidism) or elevated (to confirm the presence of hypothyroidism). Once treatment with lithium salt has commenced, the analytic determinations to study thyroid functions should be repeated every year. It is necessary to re-establish the euthyroid state so that secondary psychiatric symptoms remit.

CONCLUSION

It becomes imperative to rule out thyroid disorders in all bipolar affective disorder patients as thyroiditis and mania shows similar symptoms. For every episode of mania in bipolar affective disorder, thyroid function tests must be carried out. Proper therapy helps control the progress of thyroid disorder and prevents relapse in bipolar affective disorder patients on lithium therapy.

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Conflicts of interest
There are no conflicts of interest.

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