Lithium Induced Goitre

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Abstract: Lithium is a well known anti-psychotic drug given mostly in bi-polar affective disorder. During lithium therapy, thyroid dysfunction, parathyroid dysfunction, renal tubular dysfunction, nephrogenic diabetes insipidus and cardiac toxicity can occur. Among these disorders, thyroid disorder is the most common one. Here we are presenting a case of lithium induced goitre.

Keywords: Lithium, Hypothyroidism, Goitre

1. Introduction

Lithium is an important drug in the long term therapy of bipolar affective disorder. Despite recent advances in pharmacotherapy of psychiatric illness, this drug is still preferred because of its efficacy and low cost. It is also proved as a prophylactic agent against relapse or recurrence of abnormal mood episodes in unipolar depression and mania. Thyroid dysfunction is a well known entity during lithium therapy. It can present as hypothyroidism or goitre or autoimmune hypothyroidism or very rarely hyperthyroidism. Lithium per se can induce autoimmune hypothyroidism or it can unmask the already existing autoimmune hypothyroidism. The hypothyroidism maybe clinical or sub clinical. In our case the patient developed clinical (overt) hypothyroidism after 3 years of lithium therapy and subsequently after 6 months she developed goitre.

2. Case Report

A 45 year female presented with a swelling in the thyroid region of 6 months duration. On eliciting the history, she was on lithium therapy 400 mg twice daily for the past 4 years for bi-polar affective disorder. 3 years after starting lithium therapy, patient developed hypothyroidism and there was no goitre at that time and she was started on Thyroxin 100mcg /day. 6 months later, she developed goitre with no history suggestive of pressure symptoms. On examination pulse 50/min, BP- 120/80mm, goitre was present which was non-tender. All systems were clinically normal.

On investigations, ECG showed bradycardia, CXR PA was normal, thyroid function test revealed patient was in euthyroid state as the patient is on Thyroxine therapy. Anti TPO antibodies were negative. USG- thyroid showed multi modular goitre. Thyroxine was continued in the same dosage. Lithium was also continued as per the psychiatrist opinion.

3. Discussion

Lithium is an effective antipsychiatric drug being used for the management of unipolar, bipolar depressive disorder and also for acute mania. It has also been used for long term prophylaxis of bipolar disorder. Thyroid abnormalities associated with lithium therapy have been widely reported for the past few decades. These include hypothyroidism, hyperparathyroidism, unmasking or induction of autoimmune thyroidities and goitre.

Lithium though it is an effective antipsychiatric drug, as it has low therapeutic index, toxicity is more especially during long time therapy. Hence, to prevent the occurrence of toxic effects periodic monitoring of serum lithium level will be ideal.

Lithium is concentrated in thyroid gland, 3 to 4 times more than plasma. Hypothyroidism develops in 5 - 35% of patients on lithium therapy usually 6 - 18 months of initiation and mostly subclinical and more common in females above 40. Our patients is also a female aged 45 and she developed hypothyroidism after 3 years of lithium therapy and subsequently developed goitre after 6 months

The exact mechanism for hypothyroidism is not known. Probable causes are

- 1) Lithium increases intrathyroidal iodine content.
- 2) It inhibits the coupling of iodotyrosine residues to form T4 and T3.
- 3) It inhibits the release of T4 and T3.

The magnitude of lithium induced inhibition of thyroid hormone release is sufficient to use lithium for hyperthyroidism and thyroid cancer.

Incidence of goitre on lithium therapy has been reported as 40-50%, it usually occurs 2 years after starting the treatment. Thyroid enlargement occurs due to alteration in insulin like growth factor, tyrosine kinase, and wnt/beta catenin signalling. The goitre is usually diffuse and nodular. In our case goitre developed after 3 1/2 years after starting of lithium and it was diffuse and nodular.

In view of thyroid dysfunction, before starting lithium therapy, base line thyroid hormone assay and also presence of anti thyroid antibodies should be evaluated. Even if thyroid dysfunction is detected, lithium can be started along with the treatment for hypothyroidism. TFT should be done 3 months after starting lithium and then periodically every 6 – 12 months. Although lithium induced hypothyroidism is

usually reversible after cessation of lithium, it can be continued with simultaneous treatment of hypothyroidism.

4. Conclusion

Before starting lithium treatment baseline thyroid function test to be done and when lithium therapy is started periodic TFT is to be done. If clinical or subclinical hypothyroidism is made out, Lithium need not be stopped and it can be continued along with the treatment of hypothyroidism.

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