

SITTING IN THE EYE OF THE STORM: WHEN PSYCHOTIC MANIA MASKS A METABOLIC CRISIS – A CASE REPORT

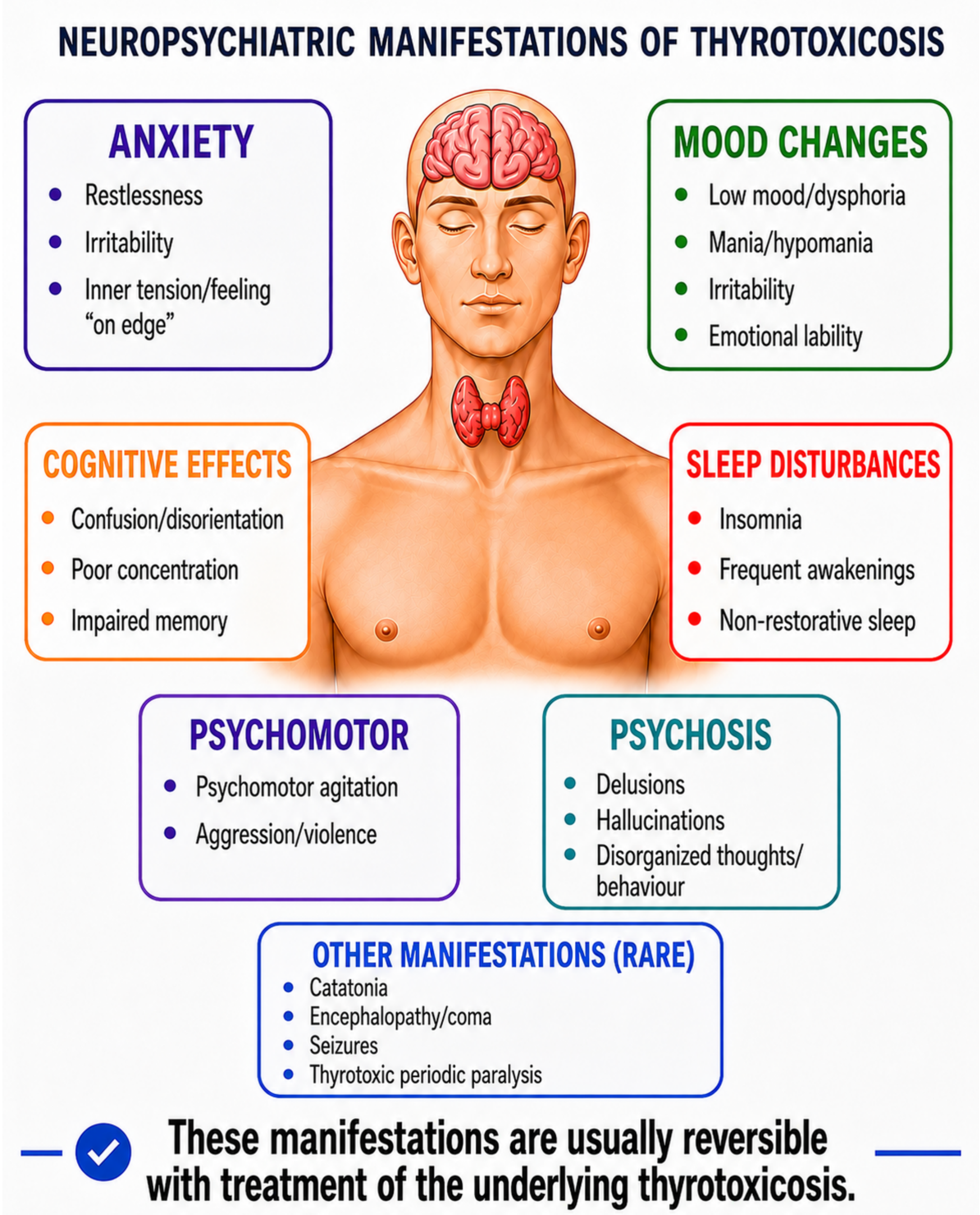
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Introduction

Severe thyrotoxicosis may present with neuropsychiatric manifestations, such as agitation, psychosis, and mania due to hyperadrenergic and hypermetabolic states.

Existing evidence suggests that these resolve with improvement or normalisation of thyroid levels (Taha et al. Health Sci Rev 2025; 16: 100237). At present, there is limited literature on managing refractory neuropsychiatric presentations while treating thyrotoxicosis, and no established treatment algorithm exists.



Objectives

To demonstrate successful use of lithium as a dual therapy for thyrotoxic mania.

Method

A 28-year-old female presented with one week of altered mental state, manifesting in excessive talkativeness, disruptiveness, and disorganised behaviour. She was noted to have been previously prescribed Methimazole for hyperthyroidism, and Risperidone for an unknown psychiatric illness.

Mental state examination showed expansive mood, labile affect, disinhibition, overfamiliarity, agitation, and was noted to be talking to herself though she denied hallucinations and delusions. Physical examination revealed a **goitre with an auscultated bruit**, and she had a **Burch-Wartofsky Point Scale score of 30** for agitation and tachycardia (heart rate of 116-136) which suggested **impending thyroid storm**. The rest of her vitals were otherwise normal.

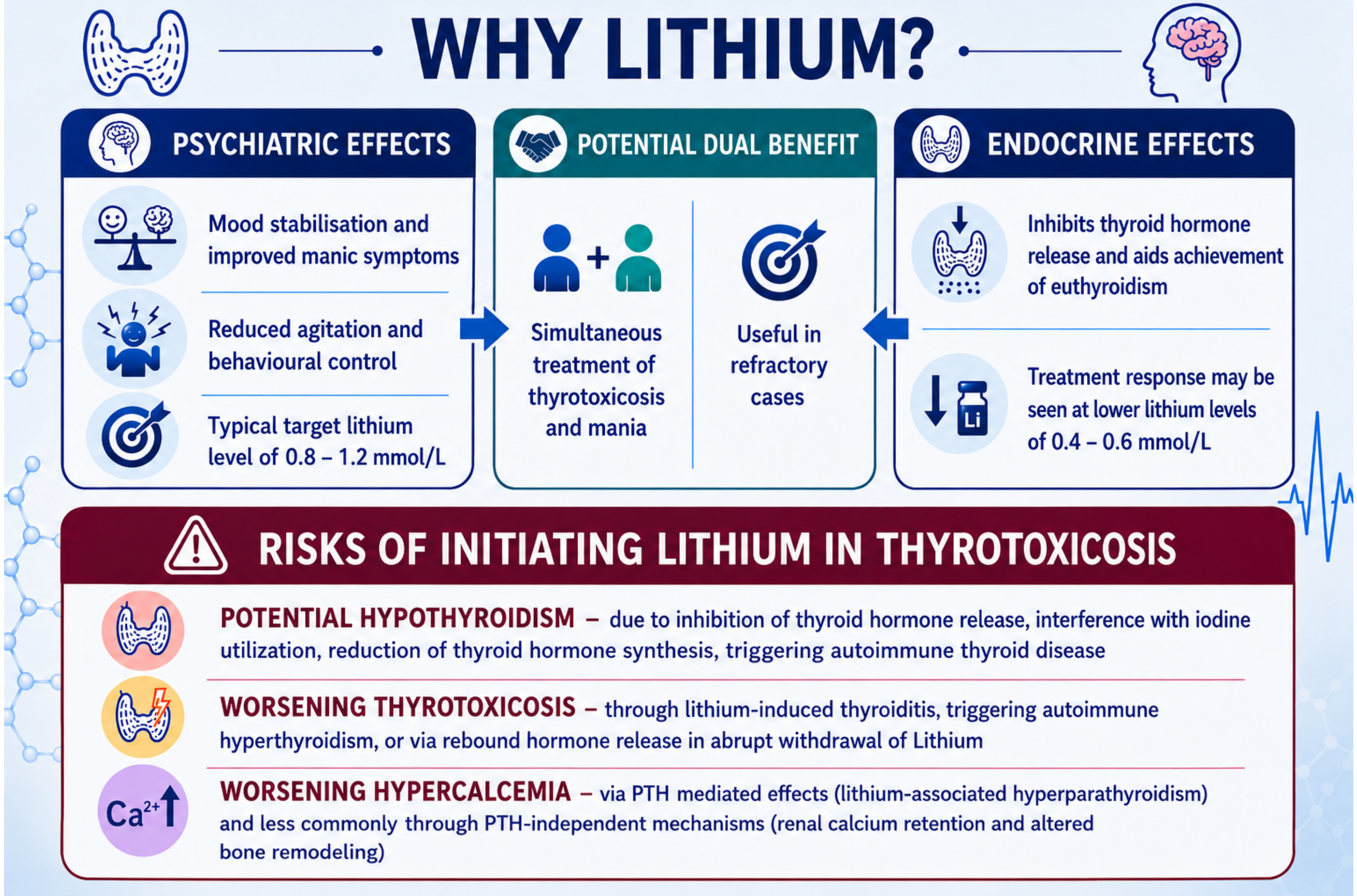
Investigations showed **severe primary thyrotoxicosis (free thyroxine >70pmol/L, thyroid stimulating hormone <0.01mIU/L)**, and hypercalcemia (peak calcium 2.8mmol/L).

Results

She was diagnosed with Grave’s Disease and mania, and received high dose Thiamazole and Propranolol. However, her mania persisted with limited response to a week of antipsychotics Olanzapine 20mg/day and adjunctive Haloperidol 4.5mg/day. In view of significant agitation in her manic state, she required regular Clonazepam during this period of time.

Failing first-line options, Lithium has evidence for treating thyrotoxicosis at levels of 0.4-0.6mmol/L (Prakash et al. Case Rep Endocrinol 2015; 2015: 869343). These levels are lower in comparison to the target lithium levels used in mania (0.8-1.2mmol/L). Important considerations of initiating Lithium included potential hypothyroidism, worsening thyrotoxicosis and hypercalcemia.

Lithium was eventually initiated with careful monitoring of thyroid, calcium and lithium levels. Lithium doses of 400mg/day was given (achieving a lithium level of 0.3mmol/L) for dual therapy of thyrotoxicosis and mania. Within twelve days from initiation, free thyroxine decreased to 26pmol/L and her mental state was calm and mood was euthymic post-treatment



Conclusion

This case highlights the diagnostic and therapeutic complexities posed by severe thyrotoxicosis presenting with prominent neuropsychiatric manifestations. Psychotic mania may obscure the underlying endocrine pathology of an impending thyroid storm. Hence, clinicians should maintain a high index of suspicion for thyrotoxicosis in such patients. While restoration of a euthyroid state remains the cornerstone of management, neuropsychiatric symptoms may persist despite initiation of appropriate antithyroid therapy and antipsychotic treatment. In this patient, adjunctive lithium was associated with biochemical improvement and resolution of manic symptoms, supporting its potential role as a dual therapeutic agent in selected refractory cases. There is a need to closely monitor the potential medical complications of initiating Lithium therapy.

Further research is required to establish therapeutic guidelines for the use of Lithium in thyrotoxicosis presenting with severe neuropsychiatric manifestations.

References

- Taha H, Abdel Wahed S, Berggren V. Psychosis secondary to thyrotoxicosis: An educational review. Health Sci Rev. 2025;16:100237.
- Prakash I, Nylen ES, Sen S. Lithium as an alternative option in Graves thyrotoxicosis. Case Reports in Endocrinology. 2015;2015:869343. doi:10.1155/2015/869343.