

Thyrotoxicosis presented with Psychosis – Basedow's Psychosis

Y Silvester, D Wijesinghe

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Introduction

Though thyrotoxicosis can present with a wide range of clinical symptoms, psychosis as a presenting problem is a rare presentation (1,2). High degree of clinical suspiciousness is important to recognize the condition. We present a case of a 64-year-old female with no history of psychiatric illnesses presented with psychotic symptoms and ultimately found to have psychosis secondary to hyperthyroidism.

Case presentation

Mrs. X, a 64-year-old female, was admitted to the mental health unit due to behavioural disturbances for 3 months duration. She had a history of hypertension and was investigated for a multi-nodular goitre in past but has not engaged with further care. Aside from the recent suicide of a distant relative, there was no significant psychiatric history in her family.

Mrs. X had a poor rapport with the mental health team. According to the co-lateral information from her family members, she had been experiencing intermittent forgetfulness, lack of energy, and wandering behaviour over the past 3 months. Prior to her admission, she had been treated with escitalopram 10mg and olanzapine 2.5mg in the community, but there was no improvement. Her

medications were adjusted to include sertraline 50mg in the morning (later increased to 100mg), risperidone 0.5mg/day (later increased to 4mg/day), and clonazepam 0.25mg as needed, with a working diagnosis of agitated depression.

During the in-ward stay, her condition deteriorated, with intermittent confusion and the development of visual hallucinations. She reported seeing fire on the bed and floods on the floor, reacting to these perceptual disturbances.

A thorough investigation was conducted to rule out other potential causes, including Lewy Body Dementia.

Basic tests, including renal function, liver function, and serum electrolytes, were normal. Electroencephalography did not reveal any significant ictal changes, and a non-contrast computed tomography scan showed only mild subcortical white matter ischemia bilaterally. Her Vitamin B12 levels were normal and thyroid peroxidase enzyme was 2.4 (<5.61). However, her Thyroid Stimulating Hormone (TSH) level was very low at 0.016, suggesting thyrotoxicosis (serum T4 was not available prior to treatment due to limitations in the government sector). An ultrasound scan of the neck depicted a multinodular goitre.

Table 1. Mrs. X's thyroid functions tests results during subsequent clinic visits

Date	TSH (0.40-4.50)	T4 (0.89-1.7)	Carbimazole dose	Psychotic symptoms
August 2024	0.016	Not available	10mg BD	significant
September 2024	<0.005	0.8	15mg N	mild
October 2024	1.460	1.123	15mg N	no
November 2024	6.130	0.36	10mg N	no
December 2024	4.279	0.47	10mg N	no

In collaboration with an endocrinologist, Mrs. X was started on 10mg of carbimazole daily. Over time, her psychotic symptoms began to improve.

During the follow up visits, she continued to show anxiety symptoms but no more psychosis. A repeat TSH was less than 0.005 (normal range: 0.3-4.2), and her T4 level was 0.8 (normal range: 0.9-1.7). Her carbimazole dose was increased to 15mg. Sertraline dose was raised to 150mg/m gradually due to anxiety symptoms. Risperidone was discontinued but she remained stable without re-appearance of psychotic symptoms. Mrs. X's Thyroid Function Tests (TFT) levels during her follow-up visit are listed in Table 1.

Discussion

Thyrotoxicosis is a condition characterized by excess thyroid hormone activity with a clinical presentation ranging from asymptomatic to life-threatening thyroid storm (3). Anxiety, irritability and insomnia are common presenting features of thyrotoxicosis (2). Psychosis as the presenting symptom in thyrotoxicosis, which is also known as Basedow's psychosis is very rare with an estimated incidence of 1% (1, 2).

There are multiple hypotheses to explain the occurrence of psychosis in thyrotoxicosis. One such hypothesis suggests dopaminergic activity found in thyroid hormones as the reason for psychosis in thyrotoxicosis (4). Another hypothesis suggests that the TSH receptor antibodies seen in Graves' Disease, activate TSH receptors in the hippocampus and cerebrum, producing neuropsychiatric symptoms (5). Literature has shown altered glucose metabolism in limbic structures in patients with hyperthyroid disease affecting the limbic system possibly leading to manifestation of psychiatric symptoms (5, 6).

Though screening for thyroid diseases is advised as a routine practice in first episode of psychosis these tests are not always available in the peripheral government hospitals and most clients from such rural areas of Sri Lanka cannot afford the tests in the private sector (7). In this background it is very easy for such conditions to go undiagnosed unless there is a goiter raising the suspiciousness.

In literature most of the patients presented with thyrotoxicosis and psychosis were managed by prescribing a combination of antipsychotics, antidepressants, mood stabilizers with adequate management of thyroid illness(2,8-12). We started treatment with antidepressants, antipsychotics and carbimazole and were able to successfully discontinue antipsychotics making the client stable only on carbimazole and antidepressants. When there are cases with rebound psychosis even after

thyroidectomy Mrs. X remained stable without any psychotic symptoms after discontinuation of the antipsychotics (13).

Conclusion

Though psychosis is a rare presentation of thyrotoxicosis it should be considered as a differential diagnosis for psychosis.

Y Silvester, District General Hospital, Ampara, Sri Lanka

D Wijesinghe, District General Hospital, Ampara, Sri Lanka

Corresponding author: Y Silvester

E-mail: yajithasilvester94@gmail.com

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