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Letter to Editor

Psychiatry/Mental Health Section

Hyperthyroidism Mimicking Primary Psychiatric Illness: A Case of Acute Psychosis

AMIT KHUNE[1], MONISHA SERALATHAN[2]

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To editor,

Hyperthyroidism typically presents with classic signs such as palpitations, tremors, heat intolerance, and weight loss. Rarely, however, it may present with neuropsychiatric symptoms including anxiety, emotional lability, or frank psychosis. Such unusual presentations pose a significant diagnostic challenge and are often mistaken for primary psychiatric illness, leading to misdiagnosis and delay in appropriate medical intervention [1-3].

The authors describe a case of a 50-year-old married woman from a lower socioeconomic background with incomplete primary education, who presented with illness of acute onset and fluctuating course of 15 days of self-talking, aggression, decreased sleep, auditory hallucinations, and irrelevant speech. She was bathing 2-3 times a day and collecting rainwater for the purpose. She was also collecting hair and small stones and keeping them inside her house, sometimes she was rolling on the ground and dancing while claiming possession by a deity which lasted for 15-20 minutes. She reported hearing her deceased son's voice calling her name. There was no disturbance of consciousness and orientation. There was no prior psychiatric history, though a family history of psychosis and suicide was present. The patient also had diabetes mellitus and hypertension for last seven years and was on metformin 1000 mg, glipizide 10 mg, and amlodipine 5 mg.

On admission, she was conscious and oriented. Pulse rate was 107/min and blood pressure was 120/90 mmHg. Systemic examination revealed no significant abnormalities. Laboratory investigations showed HbA1c 6.43%, fasting blood glucose 171 mg/dL, and postprandial blood glucose 176 mg/dL. Thyroid function tests revealed Free Triiodothyronine (FT3) 2.34 pg/mL, Free Thyroxine (FT4) 1.1 ng/dL, and Thyroid-Stimulating Hormone (TSH) 0.01 mIU/L, suggestive of subclinical hyperthyroidism. Physician's opinion was obtained for managing the medical comorbidities. She was started on carbimazole 5 mg daily along with antipsychotics-chlorpromazine 100 mg, risperidone 4 mg, diazepam 10 mg, trihexyphenidyl 2 mg, and sodium valproate 800 mg. Chlorpromazine was prescribed for its sedative effects during the acute phase and was discontinued after 15 days once agitation resolved. Risperidone was continued and significant improvement in psychotic symptoms was noted within two weeks. On follow-up, her aggressive behaviour and psychotic symptoms were reduced. She took medicines without any follow-up for the next six months during which she developed extrapyramidal side effects while on risperidone 4 mg and trihexyphenidyl 2 mg. She had slowness of activities, excessive salivation, slowness of speech, tremors, slow gait and rigidity of both upper limbs. Hyperthyroidism may increase susceptibility to antipsychotic-induced parkinsonism through altered dopaminergic neurotransmission. In the present case, this, together with prolonged risperidone exposure, may have contributed to persistent drug-induced parkinsonism despite prophylactic trihexyphenidyl.

The patient was readmitted for the management of extrapyramidal symptoms. Her antipsychotic medications were gradually tapered and discontinued over a period of 15 days. She received a stat dose of intramuscular promethazine 50 mg, which was subsequently converted to oral promethazine 25 mg daily. The dose of trihexyphenidyl was increased to 2 mg twice daily. Over the following two weeks, her extrapyramidal symptoms, including rigidity, tremors, excessive salivation, and slowness of speech, improved significantly. Thyroid function tests during inpatient care showed FT3 of 3.27 pg/mL, FT4 of 1.63 ng/dL, and TSH of 6.16 mIU/L while on carbimazole 5 mg suggestive of possible over suppression. The physician advised repeat thyroid function testing after one month for adjusting the dose of carbimazole. Ultrasonography of the thyroid revealed no abnormalities. At follow-up after 15 days, she had no psychotic symptoms. She was maintaining well with no psychotic symptoms or aggressiveness for the next six months without antipsychotics.

Psychosis secondary to thyrotoxicosis is an uncommon but well-recognised phenomenon, reported in approximately 1% of hyperthyroid cases. Proposed biological mechanisms include the impact of excess thyroid hormones on brain metabolism and the regulation of various neurotransmitter systems, specifically dopamine, serotonin, norepinephrine, and Gamma-Aminobutyric Acid (GABA). The sudden onset of hallucinations, delusions, and aggression in the present mirrors established literature describing acute psychotic states, disorganised behaviour, and thyroid-storm emergencies where psychosis is a manifesting symptom [1-3].

Similar case reports have emphasised that Graves' disease and other forms of thyrotoxicosis can mimic primary psychiatric disorders like schizophrenia or mania [2,4,5]. This report underscores the critical necessity for routine thyroid screening in patients presenting with atypical or first-episode psychosis. Clinicians should be particularly cautious when psychosis presents with abrupt onset of symptoms, a fluctuating course, or the absence of a prior psychiatric history. Concurrent physical symptoms-weight loss, tremors, palpitations, or heat intolerance-should further raise suspicion of an underlying endocrine aetiology. Early identification of organic causes can lead to complete recovery and potentially eliminate the need for long-term psychiatric medications [3,5].

Hyperthyroidism can present with severe psychiatric manifestations. Clinicians must maintain a high index of suspicion for thyroid dysfunction, as early recognition facilitates an integrated multidisciplinary approach combining endocrinology and psychiatry-that significantly improves patient prognosis and quality of life [1,2].

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PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Psychiatry, Sree Balaji Medical College and Hospital, Chromepet, Tamil Nadu, India.
2. Assistant Professor, Department of Psychiatry, Sree Balaji Medical College and Hospital, Chromepet, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Monisha Seralathan,
Assistant Professor, Department of Psychiatry, Sree Balaji Medical College and Hospital, Chromepet, Chennai-600044, Tamil Nadu, India.
E-mail: dr.monishapsychiatrist@gmail.com

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