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CASE STUDY

UNCOMMON PRESENTATION OF HYPERTHYROIDISM AS ACUTE PSYCHOSIS IN A MIDDLE-AGED PATIENT: A DIAGNOSTIC CHALLENGE AND MANAGEMENT APPROACH

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Abstract

Introduction: Hyperthyroidism commonly presents with hypermetabolic symptoms such as weight loss, tachycardia, and tremors. While neuropsychiatric manifestations like anxiety and insomnia are frequently observed, the occurrence of overt psychosis without prior psychiatric history is rare, affecting less than 1% of hyperthyroid patients.

Methods: We report the case of a 47-year-old female who presented with acute-onset psychosis, including persecutory delusions, second-person auditory hallucinations, and severe agitation. A detailed physical and biochemical evaluation was conducted alongside psychiatric and endocrinological assessments.

Results: The patient exhibited fine distal tremors, a low-grade fever (37.8 °C), and tachycardia (heart rate 128 bpm). Laboratory findings showed elevated free thyroxine (56 pmol/L; reference: 9–20), triiodothyronine (19 pmol/L; reference: 3–6), and suppressed TSH (<0.01 mIU/L). Thyroid-stimulating immunoglobulins and ultrasound findings confirmed Graves' disease. She was treated with carbimazole (20 mg/day), propranolol (40 mg TID), and low-dose haloperidol (5 mg/day), resulting in full resolution of psychiatric symptoms within two weeks.

Conclusion: This case highlights the importance of considering thyroid dysfunction in the differential diagnosis of acute psychosis.

Keywords: Hyperthyroidism, Psychosis, Graves' disease, Thyrotoxicosis, Neuropsychiatric symptoms

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BACKGROUND/INTRODUCTION

Hyperthyroidism, characterized by excessive circulating levels of thyroxine (T4) and triiodothyronine (T3), affects approximately 1–2% of the global population, with a higher female predominance (female:male ratio ~5:1) and peak incidence between ages 30–50 years [1,2]. Graves' disease accounts for 60–80% of cases in adults and represents an autoimmune-mediated hyperthyroid state triggered by thyroid-stimulating immunoglobulins (TSIs) acting on TSH receptors [3].

Classic clinical features include weight loss despite normal or increased appetite, heat intolerance, resting tachycardia, palmar tremors, and thyromegaly. Neuropsychiatric manifestations such as irritability, anxiety, insomnia, and emotional instability are frequently reported; however, the incidence of frank psychosis in untreated hyperthyroidism is rare, estimated at <1% of presentations [4].

Psychotic features can also include paranoia, delusional ideation, auditory hallucinations, and disorganized behavior, frequently akin to primary psychiatric disorders such as schizophrenia or acute mania. This often results in delayed endocrine assessment and mismanagement with antipsychotics alone [5]. Proposed mechanisms include altered monoaminergic neurotransmission, heightened β-adrenergic receptor sensitivity, and increased cerebral glucose metabolism brought about by supraphysiologic thyroid hormone levels [6,7]. This report presents a diagnostically challenging case of a middle-aged woman whose acute psychosis was the sentinel manifestation of Graves' hyperthyroidism. The case highlights the importance of incorporating targeted endocrine screening into the workup of atypical psychosis, especially in patients with subtle systemic signs or those lacking previous psychiatric history.

RESULTS

A 47-year-old woman without a known medical or psychiatric history was brought to the emergency department by family members due to a three-day history of acute behavioral disturbance. She exhibited persecutory delusions ("neighbors plotting against her"), second-person auditory hallucinations, severe agitation, and incoherent speech. There were no recent psychosocial stressors, substance use, head trauma, or medication changes. Collateral history revealed an unintentional weight loss of approximately 6 kg (10.2% of baseline body weight) over two months, increased diaphoresis, and heightened irritability. These prodromal symptoms were initially attributed to emotional stress. No previous history of thyroid dysfunction or psychiatric illness was documented in the patient or her first-degree relatives.

Physical Examination

On examination, the patient appeared disoriented and paranoid, with marked psychomotor agitation. Key clinical findings included:

- **Vital Signs**:
    - Heart rate: 128 bpm, irregularly irregular (AF)

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- Blood pressure: 142/78 mmHg
- Respiratory rate: 22 breaths per minute
- Temperature: 37.8 °C (low-grade fever)

- **Neurological and General Exam**:
  - Fine postural tremor in both hands
  - Moist, warm skin
  - Subtle bilateral exophthalmos
  - No focal neurological deficits
- **Neck Exam**:
  - Thyroid gland: diffusely enlarged, non-nodular, firm, with a palpable thrill and audible bruit, clinically suggestive of a hypervascular goiter

These findings raised clinical suspicion for thyrotoxicosis, prompting targeted endocrine workup.

Laboratory Investigations

Table 1: Initial biochemical profile showed

Test	Result	Reference Range
TSH	<0.01 mIU/L	0.4–4.0 mIU/L
Free T4	56 pmol/L	9–20 pmol/L
Free T3	19 pmol/L	3–6 pmol/L
Thyroid-Stimulating Immunoglobulin (TSI)	Positive	Negative
CBC, LFT, RFT, Electrolytes	Within normal limits	
Serum Calcium	2.35 mmol/L	2.1–2.6 mmol/L
Urine Toxicology	Negative	—

Neuroimaging

Brain MRI was performed to exclude intracranial pathology given the acute onset of psychotic symptoms. Imaging included T1, T2, FLAIR, DWI, and post-contrast sequences. Findings were as follows:

- No acute infarction, hemorrhage, mass lesions, or white matter demyelination
- No meningeal enhancement or cortical/subcortical atrophy
- No radiological evidence of autoimmune encephalitis or vasculitis

The absence of structural or inflammatory CNS pathology strengthened the case for a metabolic or endocrine etiology.

Endocrine and Imaging Workup

- **Thyroid Ultrasound**:
  - Diffusely enlarged gland with heterogeneous echotexture

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- **Increased vascularity** ("thyroid inferno" on Doppler)
- No nodules or cysts

- **Autoimmune Panel**:
  - Anti-thyroid peroxidase (anti-TPO) antibodies: Mildly positive
  - Anti-thyroglobulin antibodies: Mildly positive

These findings were consistent with **Graves' disease** as the underlying etiology.

Management and Clinical Course

The patient was admitted to a monitored high-dependency unit. Initial treatment included:

- Carbimazole 20 mg once daily (to inhibit thyroid hormone synthesis)
- Propranolol 40 mg TID (for symptomatic relief and inhibition of peripheral T4 to T3 conversion)
- Haloperidol 2.5 mg BID (for acute psychosis, used cautiously due to QTc prolongation risk)

Cardiac telemetry and serial complete blood counts were monitored to detect arrhythmia and agranulocytosis, respectively.

Clinical Response:

- By Day 5: Heart rate reduced to <90 bpm; psychotic agitation diminished
- By Day 14: Complete resolution of hallucinations and delusions; regained insight
- No adverse effects from antithyroid or antipsychotic medications were observed

The patient was discharged on carbimazole and referred for outpatient endocrinology and psychiatry follow-up.

At the 3-month review:

- Thyroid profile normalized (euthyroid state)
- Propranolol had been tapered off
- Psychiatric medications discontinued
- No recurrence of symptoms noted

Radioiodine ablation was discussed as a definitive option, though the patient opted to continue pharmacotherapy.

DISCUSSION

Acute psychosis as the primary manifestation of hyperthyroidism is exceptionally uncommon, documented in fewer than 1% of thyrotoxicosis cases. Most patients with hyperthyroidism present with classic symptoms such as weight loss, tremors, palpitations, and heat intolerance. However, when neuropsychiatric features, particularly hallucinations and delusions dominate the clinical presentation, diagnosis may be delayed or misdirected toward primary psychiatric illness, especially in the absence of a prior psychiatric history. In this case, the patient's

presentation with persecutory delusions and auditory hallucinations, coupled with psychomotor agitation, initially suggested a functional psychosis. Subtle clinical clues such as fine tremors, mild exophthalmos, and an enlarged thyroid prompted further investigation for an organic etiology. The neuropsychiatric sequelae of thyrotoxicosis are believed to result from excessive thyroid hormones increasing central β-adrenergic receptor sensitivity and altering neurotransmission involving serotonin and dopamine. Additionally, supraphysiologic T3 levels may increase cerebral glucose metabolism and

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oxygen consumption, particularly in limbic and prefrontal cortical regions, which are critical to mood and behavior regulation.

Neuroimaging was crucial in ruling out alternate diagnoses such as autoimmune encephalitis, space-occupying lesions, or vascular malformations. A normal MRI further supported a metabolic rather than structural cause for the patient's symptoms. Management included antithyroid therapy with carbimazole 20 mg daily and propranolol 40 mg TID, which provided both symptomatic relief and peripheral blockade of T4-to-T3 conversion. Due to the severity of agitation and potential risk to self and others, a low dose of haloperidol (2.5 mg BID) was administered with ECG monitoring to mitigate the risk of QTc prolongation.

The patient exhibited marked clinical improvement within the first week, with complete resolution of psychotic features by the end of the second week. Her recovery following normalization of thyroid hormone levels provides strong evidence for a causal relationship rather than comorbidity. Studies have shown that in such presentations, long-term psychiatric sequelae are rare when endocrine dysfunction is corrected early. This case highlights the need for physicians to maintain a high index of suspicion for endocrine disorders, especially in middle-aged or older individuals with atypical psychosis. Early identification and multidisciplinary intervention are essential for reversing symptoms, preventing unnecessary antipsychotic exposure, and achieving sustained remission.

CONCLUSION

This case highlights that hyperthyroidism, particularly due to Graves' disease, can present as acute psychosis even in the absence of prior psychiatric history. Early recognition of subtle systemic signs and timely initiation of antithyroid therapy can lead to complete psychiatric remission, underscoring the importance of including thyroid function testing in the diagnostic workup of new-onset psychosis.

LIMITATION

This is a single-patient case report, limiting generalizability to broader populations.

RECOMMENDATION

Routine thyroid screening should be considered in patients presenting with first-episode psychosis, especially in middle-aged individuals.

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CONFLICT OF INTEREST

The author declares no conflict of interest related to this study.

LIST OF ABBREVIATION

TSH – Thyroid-Stimulating Hormone

T3 – Triiodothyronine

T4 – Thyroxine

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TSI – Thyroid-Stimulating Immunoglobulin

MRI – Magnetic Resonance Imaging

AF – Atrial Fibrillation

ECG – Electrocardiogram

CBC – Complete Blood Count

LFT – Liver Function Test

RFT – Renal Function Test

QTc – Corrected QT Interval

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