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Graves' Disease Revealed by an Acute Psychotic Episode in A Young Patient: A Rare Clinical Presentation at the Intersection of Psychiatry and Endocrinology

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Abstract | **Case Report**

Graves' disease is a common autoimmune thyroid disorder; however, its initial psychiatric presentations, especially psychotic episodes, are rare and challenging. We report the case of a 25-year-old patient, initially treated for atypical depression, who developed an acute psychotic episode marked by auditory hallucinations, irritability, insomnia, and cognitive decline. Treatment with olanzapine led to improvement in psychiatric symptoms. Biological tests showed severe hyperthyroidism (suppressed TSH, significantly elevated free T4 and free T3), positive anti-thyroid antibodies, and high TSH receptor antibodies (TRAb), confirming Graves' disease. Starting treatment with carbimazole and beta-blockers resulted in steady clinical improvement. This case highlights the importance of thoroughly investigating physical causes in any sudden, unusual psychiatric disorder, especially in young patients. Psychosis can sometimes be the only initial sign of autoimmune thyrotoxicosis, and early targeted treatment is vital for prognosis.

Keywords: psychosis, hyperthyroidism, Graves' disease, autoimmune thyroiditis, secondary psychiatric disorder.

INTRODUCTION

Psychotic disorders are marked by a significant disconnection from reality, presenting symptoms such as delusions, hallucinations, and thought disturbances. While their cause is most often idiopathic, as seen in schizophrenia or schizoaffective disorders, a large proportion may stem from underlying medical conditions, especially endocrine disorders [1,2].

Among the organic causes of psychosis, thyroid dysfunctions, especially hyperthyroidism, deserve special attention. Although hyperthyroidism usually presents with physical symptoms and mood changes, isolated psychiatric initial symptoms, including psychotic episodes, although rare, have been documented [3,4]. These unusual cases highlight the importance of comprehensive physical assessment in any first psychotic episode, particularly in young people [5].

Graves' disease, the primary autoimmune cause of hyperthyroidism, remains an underestimated etiological factor in secondary psychosis. Recent case reports describe inaugural psychotic episodes revealing Graves' disease in the absence of psychiatric history or typical somatic signs [6,7,8]. The underlying mechanisms remain incompletely understood but are likely to involve the direct neurotoxicity of thyroid hormones on the central nervous system, disruptions in dopaminergic transmission, as well as immunoinflammatory processes affecting the brain [9,10].

In young adults, any first psychotic episode requires a comprehensive diagnostic assessment to identify potentially reversible organic causes. The case reported herein illustrates this challenge by highlighting an acute psychotic manifestation as the first presentation of Graves' disease in a patient without prior psychiatric or endocrine history. This case raises questions about the psychiatrist's role in detecting atypical somatic signs and emphasizes the importance of precise etiological diagnosis to tailor appropriate therapeutic management.

CLINICAL CASE

The patient is a 25-year-old male with no significant medical or psychiatric history, followed for

approximately one year in psychiatric outpatient care for atypical depression. His initial clinical presentation included progressive social withdrawal, psychomotor retardation, and substantial impairment in social and occupational functioning. Treatment with fluoxetine at 20 mg/day was initiated, leading to partial improvement in mood symptoms.

In the weeks before hospitalization, his clinical course was characterized by the emergence of active psychotic symptoms, such as auditory hallucinations, notable irritability, subjective cognitive decline, and severe insomnia. Due to this acute psychotic episode, a diagnostic and therapeutic hospitalization was arranged. Olanzapine was started at 10 mg/day and quickly increased to 20 mg/day, leading to significant improvement in psychotic symptoms and normalization of sleep.

A comprehensive biological workup revealed significant thyroid dysfunction, with suppressed TSH levels (< 0.01 mIU/L), elevated free T4 levels (> 5 ng/dL), and increased free T3 levels (> 20 pg/mL). Immunological testing showed elevated anti-thyroid antibodies, including anti-thyroperoxidase (831,22 IU/mL), anti-thyroglobulin (38.61 U/mL), and TSH receptor antibodies (TRAb) at 3.9 IU/L. Cervical ultrasound revealed an enlarged, heterogeneous thyroid gland with inflammatory features. Thyroid scintigraphy indicated diffuse increased uptake, consistent with autoimmune hyperthyroidism such as Graves' disease.

Specific treatment was initiated in collaboration with endocrinology: propranolol (Avlocardyl®) at 60 mg/day combined with carbimazole (Dimazol®) at 40 mg/day. Gradual improvement in both physical and psychiatric health was observed over the following days, supporting a secondary cause of the psychiatric symptoms due to hyperthyroidism.

DISCUSSION

The presented case falls within the still underexplored area of secondary psychoses linked to thyroid dysfunction, especially in the context of Graves' disease. It shows a complex clinical situation where an initially interpreted atypical depressive syndrome develops into an acute psychotic episode, ultimately revealing severe autoimmune hyperthyroidism. This gradual progression, with late onset of hyperthyroid signs, differs from the usually abrupt presentation described in reported cases of psychosis caused by thyroid imbalance.

In the literature, psychiatric disorders linked to thyroid dysfunction, although acknowledged since the 19th century, still often go undiagnosed, especially when they first appear as a psychotic episode. As Aarab *et al.,* [11,12] emphasize, acute psychotic episodes have been documented in patients without any prior psychiatric history amid unmanaged thyroid dysfunction, showing symptoms like agitation, incoherent speech, persecutory delusions, and severe insomnia. Unlike our patient, these cases usually display sudden and isolated psychiatric symptoms without obvious previous mood issues.

Our observation is notable for the initially chronic course marked by social withdrawal and psychomotor retardation, which may suggest a primary affective disorder. The subsequent emergence of psychotic features and activation syndrome (irritability, insomnia, auditory hallucinations) prompted the introduction of an atypical antipsychotic (olanzapine), with favorable response, consistent with case reports describing treatment with olanzapine or amisulpride [11].

The somatic workup revealed a typical biological profile of Graves' disease, characterized by suppressed TSH, significantly elevated free T3 and T4 levels, positive antithyroid antibodies, and scintigraphic hyperuptake. This diagnosis, identified secondarily, highlights the importance of thorough somatic assessment in any acute psychotic episode, especially in young patients without a documented psychiatric history, as emphasized by several authors [11,13,14].

Therapeutically, the combination of antipsychotic treatment and endocrine correction (carbimazole and propranolol) led to rapid clinical improvement, reinforcing the hypothesis of a causal relationship between thyrotoxicosis and psychotic symptoms. This therapeutic approach aligns with implicit recommendations derived from published observations, where psychiatric manifestations regressed with normalization of thyroid function [11,12,15].

Finally, the pathophysiological hypotheses discussed in the literature—namely, the neurotoxic effects of thyroid hormones, dopaminergic transmission disruption, and central immuno-inflammatory mechanisms [9,16,17] may explain the heterogeneous psychiatric expression of Graves' disease. The favorable outcome observed in our patient following endocrine correction and antipsychotic treatment supports these hypotheses and strengthens the concept of a pathogenic continuum between thyroid imbalance and neuropsychiatric vulnerability.

CONCLUSION

Psychiatric presentations of thyroid dysfunctions, although relatively rare in psychotic form, represent an important diagnostic challenge, particularly when they constitute the initial clinical manifestation. Graves' disease, in its purely psychiatric form, may mislead clinicians if somatic signs are subtle or absent at presentation. This case highlights the importance of systematically including thyroid function testing in the

evaluation of a first psychotic episode, especially in young adults without prior psychiatric history.

The positive response to combined treatment an atypical antipsychotic and correction of hyperthyroidism strengthen the hypothesis of a causal link between hormonal imbalance and acute psychiatric disorders. This integrated diagnostic and therapeutic approach often leads to rapid improvement or even complete remission, while reducing the risk of chronicity and inappropriate long-term treatment. This clinical scenario highlights the importance of close collaboration between psychiatrists and endocrinologists to achieve optimal management of these reversible conditions.

REFERENCES

1. Zonana J, *et al., Medical causes of psychosis*. Psychiatr Clin North Am. 2021;44(2):245-260.
2. Lima N, *et al., Endocrine disorders and mental health: clinical insights*. Lancet Psychiatry. 2022;9(1):20-34.
3. Ozdemir I, *et al., A rare case of hyperthyroidism presenting with acute psychosis*. Gen Hosp Psychiatry. 2015;37(2):192.e5-192.e6.
4. Sridharan K, *et al., Hyperthyroidism-induced psychosis: A diagnostic challenge*. Indian J Psychol Med. 2020;42(1):90-92.
5. Freudenreich O, *et al., Evaluation of new-onset psychosis in adults*. N Engl J Med. 2020;382(9):879–889.
6. De Vries F, *et al., Psychiatric manifestations of Graves' disease: a systematic review*. Thyroid. 2022;32(7):721–732.
7. Khemka R, *et al., Psychosis and Graves' disease: a rare but important association*. BMJ Case Rep. 2018.
8. Yoshimura R, *et al., Hyperthyroidism-induced psychosis: report of two cases and review of the literature*. Psychiatry Clin Neurosci. 2014;68(7):508–510.
9. Bauer M, Goetz T, Glenn T, Whybrow PC. The thyroid-brain interaction in thyroid disorders and mood disorders. J Neuroendocrinol. 2008;20(10):1101-1114.
10. Iacovides A, *et al.,* Neuropsychiatric aspects of Graves' disease. Psychoneuroendocrinology. 2023;152:106112.
11. Danion C, Domenech P, Demily C. Symptômes psychotiques dans les affections médicales générales de l'adulte. EMC Psychiatrie. 2007;37-297-A-10.
12. Aarab C, Hammani Z, Aalouane R, Yassari M, Rammouz I. Psychose aiguë secondaire à une dysthyroïdie : à propos de 2 cas. Pan Afr Med J. 2016;25:216. doi:10.11604/pamj.2016.25.216.10247.
13. Bunevicius R, Prange AJ Jr. Psychiatric Manifestations of Graves' Hyperthyroidism. CNS Drugs. 2006;20(11):897–909.
14. Benros ME, *et al.,* Autoimmune diseases and severe infections as risk factors for schizophrenia: a 30-year population-based register study. Am J Psychiatry. 2011;168(12):1303–1310.
15. Alson RL, *et al.,* Psychosis associated with hypothyroidism: a case report and review. J Emerg Med. 1990;8(4):455–461.
16. Chatti S, *et al.,* Une psychose délirante relevant une maladie de Basedow. Ann Endocrinol. 2013;74(4):376–377.
17. Bergink V, Gibney SM, Drexhage HA. Autoimmunity, Inflammation, and Psychosis: a search for peripheral markers. Biol Psychiatry. 2014;75(4):324–331.