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A Case Report of Acute Depressive Symptoms Caused by Hyperthyroidism

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Case Report

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Abstract

Background

Thyroid dysfunction is a well-established cause of secondary psychiatric disturbances. While hypothyroidism is classically associated with depressive symptoms and hyperthyroidism with anxiety and irritability, atypical presentations can occur. Here we report a case of hyperthyroidism that presented with prominent acute depressive symptoms—a manifestation that remains strikingly underrecognized and poses significant diagnostic challenges in psychiatric emergency settings.

Case presentation

A 31-year-old woman presented with a three-day history of mutism, intense guilt, frequent crying, insomnia, and loss of appetite. Examination revealed fever (37.5°C), tachycardia (121 bpm), and a tearful, largely mute affect. Emergency investigations including cranial CT and routine blood tests were unremarkable, and a preliminary diagnosis of acute psychotic disorder was made. Admission laboratory screening unexpectedly revealed marked hyperthyroidism: total thyroxine 319.14 nmol/L (reference: 69.71–163.95) and thyroid-stimulating hormone < 0.01 mIU/L (reference: 0.38–5.33). Thyroid ultrasonography and elevated thyroid-stimulating immunoglobulin confirmed Graves' disease with impending thyroid storm. The patient was treated with propranolol, propylthiouracil, and intensive care monitoring. Residual depressive symptoms responded to low-dose lithium carbonate (0.3 g daily) and risperidone (2 mg daily), with antidepressants deliberately avoided. Full remission was achieved by day 16. Retrospective history later revealed a one-year history of unreported hyperthyroid symptoms.

Conclusion

This case challenges the conventional dichotomy of thyroid-related psychiatric presentations, demonstrating that hyperthyroidism can present with predominantly depressive symptoms. Unreliable history and absent classic physical signs delayed diagnosis, underscoring the importance of routine laboratory screening in psychiatric emergencies. The combination of lithium and risperidone, with avoidance of antidepressants, offers a practical management strategy.

Background

Thyroid dysfunction is highly prevalent, affecting an estimated 10% to 15% of the general population [1, 2]. The classical psychiatric presentations are well established: hypothyroidism is commonly associated with depressive symptoms such as low mood, anhedonia, and psychomotor retardation [3], whereas hyperthyroidism typically manifests with anxiety, irritability, and emotional lability [4]. However, the present case challenges this conventional dichotomy. We encountered a patient whose hyperthyroidism presented predominantly with acute depressive symptoms—a manifestation that remains strikingly

underrecognized in both psychiatric and endocrine literature. This atypical presentation created substantial diagnostic uncertainty in the emergency setting, delaying targeted treatment. Following identification and management of the underlying thyroid pathology, the patient's depressive symptoms improved rapidly, underscoring the critical importance of maintaining a high index of suspicion for organic etiologies in acute psychiatric presentations.

Case presentation

On August 28, 2024, a 31-year-old woman was brought to the emergency department by her mother. The patient exhibited fever, complete refusal to speak or eat, and episodes of uncontrollable crying. She actively resisted evaluation and cried loudly when gently guided into the consultation room, repetitively uttering fragmented phrases such as "I am wrong" and "go home." On initial physical examination, her body temperature was 37.5°C, blood pressure 138/84 mmHg, pulse 121 beats per minute, and respiratory rate 21 breaths per minute. Her affect was sad and tearful, and she remained largely mute or provided only minimal responses to direct questioning.

According to her mother, approximately three days prior to presentation, the patient had developed progressive behavioral changes including marked reduction in speech, frequent episodes of crying, and obsessive rumination over minor past events. She expressed intense and disproportionate feelings of guilt, accompanied by severe insomnia and near-complete loss of appetite. Medical attention was ultimately sought after the patient developed a high fever in the early morning hours of the day of admission. Critically, the mother categorically denied any prior medical or psychiatric history, which proved to be a pivotal piece of misleading information that initially obscured the underlying diagnosis.

Emergency evaluation, including cranial computed tomography, complete blood count, and serum biochemistry, revealed no acute abnormalities. The patient received an intramuscular injection of 10 mg haloperidol for acute agitation, and physical restraints were applied as a temporary safety measure. Psychiatric consultation was urgently requested, and a preliminary diagnosis of an acute psychotic disorder was made, with a recommendation for inpatient admission to facilitate comprehensive etiological investigation and multidisciplinary treatment.

Upon admission to the psychiatric ward, the patient's clinical condition remained essentially unchanged. On August 29, routine admission laboratory investigations yielded an unexpected and pivotal finding: thyroid function tests revealed marked hyperthyroidism. Total thyroxine (TT4) was 319.14 nmol/L (reference range: 69.71–163.95), and thyroid-stimulating hormone (TSH) was below the detectable limit of 0.01 mIU/L (reference range: 0.38–5.33). Thyroid ultrasonography demonstrated diffuse glandular enlargement with heterogeneous echotexture. Thyroid-stimulating immunoglobulin (TSI) was elevated at 33.59 IU/L, confirming the diagnosis of Graves' disease. Concurrently, the patient continued to exhibit persistent tachycardia and hyperthermia, raising concern for an impending thyroid storm.

Endocrinology was urgently consulted. Given the imminent risk of progression to a full thyroid storm, the patient was promptly started on propranolol 20 mg three times daily and propylthiouracil 100 mg three

times daily, together with a strict low-iodine diet and aggressive intravenous fluid resuscitation. She was transferred to the intensive care unit for close hemodynamic monitoring and supportive care. After five days in the ICU, both thyroid function indices and inflammatory markers showed sufficient clinical improvement to permit transfer back to the general psychiatric ward. However, despite normalization of her endocrine parameters, the patient's psychiatric symptoms—including persistent guilt delusions and spontaneous, uncontrollable weeping—remained prominent and clinically concerning.

Following a thorough multidisciplinary assessment, low-dose lithium carbonate (0.3 g daily) was initiated for its dual mood-stabilizing and thyroid-suppressing properties, and risperidone was gradually titrated from 0.5 mg to 2 mg daily to target residual affective and psychotic symptoms. Within four days of initiating this combined psychopharmacological regimen, the patient's psychiatric symptoms were substantially alleviated, and she regained the capacity for coherent, goal-directed communication. She was discharged on September 13, 2024, in a markedly improved condition, with arrangements for integrated psychiatric and endocrinology outpatient follow-up. Detailed history obtained during the recovery phase subsequently revealed that the patient had been experiencing classic hyperthyroid symptoms—including palpitations, excessive sweating, unintentional weight loss, and irritability—for over one year, yet had never sought medical evaluation. She explicitly denied any identifiable psychosocial precipitants for the current acute episode.

Discussion

This case offers several instructive clinical lessons regarding the intersection of endocrine and psychiatric emergencies.

Diagnostic challenges and the pitfall of an absent history. The most immediate lesson concerns the diagnostic complexity posed by acute psychiatric presentations of medical illness. Several factors converged to obscure the underlying etiology in this case. First, the family member providing the initial history categorically denied any prior medical conditions, a report that was later proven to be entirely inaccurate. This underscores a fundamental principle: in psychiatric emergencies, collateral history—while invaluable—must never substitute for objective laboratory investigation. Second, the patient exhibited none of the classic physical stigmata of hyperthyroidism, such as exophthalmos or a visibly enlarged thyroid gland, making the endocrine etiology clinically silent on routine physical examination. Third, the autonomic signs present—tachycardia and fever—were non-specific and could plausibly be attributed to acute psychomotor agitation, prolonged refusal of oral intake, and the physiological stress of physical restraint, all of which are common in psychiatric emergency settings. The convergence of these misleading factors created a genuine diagnostic dilemma, and the correct diagnosis was only established through routine admission laboratory screening. This case thus serves as a compelling argument for the inclusion of thyroid function tests—and indeed a broader metabolic panel—in the standard workup of all patients presenting with acute psychiatric disturbances of unclear etiology.

An atypical symptom profile that expands the recognized clinical spectrum. The existing literature on hyperthyroidism-associated psychiatric conditions is heavily weighted toward presentations dominated by psychosis, with hallucinations and delusions as the cardinal features [5, 6]. Descriptions of hyperthyroidism manifesting primarily with depressive symptoms remain exceedingly rare. This case therefore contributes to a more nuanced understanding of the psychiatric phenotype of thyrotoxicosis. The patient's clinical picture—characterized by mutism, intense guilt, psychomotor retardation, tearfulness, insomnia, and anorexia—was phenomenologically indistinguishable from a severe major depressive episode with psychotic features or an acute psychotic disorder. That this presentation was driven by hyperthyroidism, and resolved in parallel with its treatment, challenges the conventional clinical heuristic that neatly assigns depression to hypothyroidism and anxiety to hyperthyroidism. Clinicians should be aware that the psychiatric manifestations of thyroid disease exist on a broader and more overlapping spectrum than is traditionally taught.

A practical psychopharmacological strategy and the rationale for avoiding antidepressants. The management of this patient's residual psychiatric symptoms required careful pharmacological deliberation. While antipsychotics have been reported to be beneficial in hyperthyroidism-related psychosis [5], evidence guiding the management of hyperthyroidism-associated depressive symptoms is sparse. We selected risperidone based on its well-documented efficacy for affective symptoms in psychotic and mood disorders, and the patient demonstrated a favorable response at a relatively low dose. Lithium carbonate was introduced for its mood-stabilizing properties, with the additional theoretical benefit of its known inhibitory effect on thyroid hormone release [8]—a pharmacological property that may confer a unique advantage in the acute management of hyperthyroidism-associated psychiatric disturbances. Equally important is the pharmacological decision we deliberately avoided: we refrained from prescribing antidepressant medications. This cautious approach was informed by the legitimate concern that antidepressants, particularly selective serotonin reuptake inhibitors, could exacerbate the patient's tachycardia, hyperthermia, and diaphoresis—symptoms already prominent in her clinical picture. This concern is not merely theoretical; a previous case report has documented fluoxetine as a potential trigger for hyperthyroidism [7], providing empirical support for our treatment decision. We believe this combined approach—low-dose lithium plus an atypical antipsychotic, with avoidance of antidepressants—represents a safe and practical pharmacological strategy that other clinicians may consider when facing similar cases.

Limitations. Several limitations warrant acknowledgment. As a single case report, the generalizability of our findings is inherently constrained. The temporal overlap between the patient's endocrine improvement and the initiation of psychotropic medications makes it impossible to isolate the relative contribution of each intervention to her psychiatric recovery. Long-term follow-up data beyond the immediate post-discharge period are not available, limiting our understanding of the durability of her improvement and the trajectory of her thyroid and psychiatric conditions.

Conclusion

This case illustrates that hyperthyroidism can present with predominantly depressive symptoms—a manifestation that directly contradicts conventional clinical teaching and poses a significant diagnostic challenge in psychiatric emergency settings. The absence of a reliable history and of classic physical signs of thyroid disease made the diagnosis particularly elusive, reinforcing the indispensable role of routine laboratory screening in acute psychiatric presentations. The favorable outcome achieved through combined antithyroid treatment and carefully selected psychotropic medications—lithium and risperidone, with deliberate avoidance of antidepressants—offers a practical management template for similar cases. Above all, this case underscores a vital clinical principle: every acute psychiatric presentation, no matter how classically "psychiatric" it appears, demands a thorough search for an underlying medical etiology.

Declarations

Ethics and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki.

Approval was granted by the Zhejiang Tongde Hospital Medical Ethics Committee(No205, 2024). Informed consent was obtained from all individual participants included in the study.

Consent to publish

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The author declares no competing interests.

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Author Contribution

Zhangyin He was solely responsible for the clinical management of the patient, conceptualization of the case report, literature review, manuscript drafting and revision, and final approval of the version to be published.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article. No additional datasets were generated.

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