

Thyroid Storm; A Rare Manifestation of Thyrotoxicosis, A Case Report

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ABSTRACT

Thyroid storm is a life-threatening endocrine emergency resulting from hyperthyroidism, characterized by widespread multisystem involvement. We present the case of a middle-aged female who presented with fever, diarrhea, and vomiting. She gave a history of weight loss and palpitations for the past year. On examination, she appeared restless and dehydrated. Notable findings included resting tremors, tachycardia, and a wide pulse pressure. Based on clinical evaluation and confirmed by thyroid function tests (TFTs), she was diagnosed with a thyroid storm precipitated by acute gastroenteritis. She had a rapid improvement after the start of treatment consisting of IV fluids, beta-blockers, antithyroid drugs, and steroids. This case demonstrates that prompt clinical insight can be crucial for saving lives in rare endocrine emergencies such as thyroid storms.

Keywords: Acute Liver Dysfunction, Case Report, Graves' Disease, Portal Hypertension.

Authors' Contribution:

All authors contributed equally to the conception, literature search, manuscript drafting, editing and review

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Article info:

Received: February 04, 2025
Accepted: July 20, 2026

Cite this article. Qurat ul Ain, Khalid A, Rasheed R. Thyroid Storm; A Rare Manifestation of Thyrotoxicosis, A Case Report. J Islamabad Med Dental Coll. 2026; 15(3): 364-367.
DOI: <https://doi.org/10.35787/jimdc.v15i3.1353>

Funding Source: Nil
Conflict of interest: Nil

Introduction

Thyroid storm is a rare but severe manifestation of hyperthyroidism, accounting for approximately 1% to 2% of hospital admissions related to hyperthyroidism. Despite advances in medical therapy and supportive care, it remains associated with a high mortality rate ranging from 8% to 25%.¹ This critical condition results from a sudden and excessive increase in thyroid hormone activity. While it most commonly develops in the context of untreated or poorly controlled Graves' disease, it can also arise from any form of thyrotoxicosis, including toxic multinodular goiter and thyroiditis. The diagnosis is primarily clinical, based on the presence of severe hyperthyroidism accompanied by systemic decompensation manifesting as high

fever, marked tachycardia, atrial fibrillation, gastrointestinal disturbances, and central nervous system abnormalities.^{1,2} The Burch-Wartofsky Point Scale is a widely used clinical tool that aids in the early recognition and risk stratification of suspected thyroid storm cases.³

Case Presentation

A 48-year-old female, a resident of Jhelum presented to the ER with a history of multiple episodes of loose motion and vomiting accompanied by fever for 2 days. The stools were large volume, watery, and did not contain blood or mucus. And there was only mild dull abdominal pain. Fever was moderate grade and continuous. On further inquiry, she reported an unintentional weight loss of 5 kg

over one year, but it was not accompanied by anorexia, vomiting, abdominal discomfort, fever, or joint pain on examination, she was conscious and well-oriented in time place, and person., She was severely dehydrated. She had resting tremors but no goiter or any exophthalmos. Observations were as follows: pulse rate of 138 beats/minute(bpm), blood pressure (BP) of 170/82 mmHg, respiratory rate of 18/minute, oxygen saturation (SPO2) of 96% on room air, and temperature of 37.9 C. CNS - conscious, oriented, with no focal neurological deficit, Resp - chest B/L clear, GIT - soft, nontender abdomen, CVS - S1 + S2 (Tachycardia).

The patient's complete blood count (CBC) revealed values within normal limits: hemoglobin 13.5 g/dL, total leukocyte counts 9,900/mm³, and platelet count 240,000/mm³. Liver Function Test (LFT) were normal: Total Bilirubin 0.8 mg/dL, AST 27 IU/L, ALT 34 IU/L, ALP 121 U/L. Renal function tests (RFT) were also normal, with a serum creatinine of 0.9 mg/dL and blood urea nitrogen (BUN) of 14 mg/dL. Electrolytes were within reference ranges: sodium 138 mmol/L, potassium 4.2 mmol/L, and chloride 102 mmol/L. However, C-reactive protein (CRP) was elevated at 45 mg/L (normal <5 mg/L), indicating an underlying inflammatory or infectious process. The patient was hyperthyroid biochemically with a low thyroid-stimulating hormone of 0.01 (reference: 0.4-4.5 uIU/mL) and high total T4 of 3.25 (reference: 0.8-2.7 ng/dL) total T3 was 1.88 (reference 0.55-1.70 ng/mL). The Burch-Wartofsky point scale score was 50 (temp=10, CNS=0, Gastrointestinal=10, HR=20, CCF=0, Precipitating factor=10(present)) consistent with a diagnosis of thyroid storm.

She was admitted to the ICU with a diagnosis of "thyroid storm precipitated by gastroenteritis". Rehydration was initiated with intravenous fluids. Gastroenteritis was treated with antibiotics and antiemetics. Intake and output were strictly monitored, and IV fluid boluses were administered accordingly. Oral propranolol was administered at an initial dose of 40 mg stat, followed by 40 mg every 6 hours. A loading dose of oral carbimazole 40 mg

was given, followed by 20 mg every 6 hours. Intravenous hydrocortisone 100 mg was administered three times daily. Lugol's iodine was unavailable; therefore, the patient was managed with other standard treatment protocol. The patient showed significant improvement after 24 hours and the same treatment regimen was continued. She was discharged on the fifth-day post-admission. At discharge, she was prescribed a beta-blocker, carbimazole, and a tapering dose of oral steroids. TFT at discharge showed improvement, with TSH at 0.08 mIU/L, T4 at 2.98 ng/dL, and T3 at 1.80 ng/mL. She was referred to an endocrinologist for further evaluation and management of her thyroid disorder.

Discussion

Thyroid storm is a severe hypermetabolic state resulting from uncontrolled thyroid hormone release. It is often associated with underlying Graves' disease but can occasionally occur with other thyrotoxic conditions like destructive thyroiditis, toxic multinodular goiter, or metastatic thyroid cancer.³ Common triggers include acute stress, pregnancy, medication non-compliance, and amiodarone use.^{1,4} The Burch-Wartofsky Point Scale, introduced in 1993, aids in predicting thyroid storm risk by assessing factors such as body temperature, central nervous system dysfunction, tachycardia, atrial fibrillation, heart failure severity, gastrointestinal symptoms, and precipitating events.^{1,3,4} A score above 45 strongly suggests a thyroid storm.

As the metabolic activity in hyperthyroidism is increased, it enhances myocardial contractility which results in increased stroke volume and reduced systemic vascular resistance. This effect leads to a widened pulse pressure (PP).^{5,6} Sinus tachycardia, defined as HR of >100 bpm, is a common sign of hyperthyroidism present in 70–100% of patients.^{5,7} Our patient had both of these clinical findings of hyperthyroidism; tachycardia and hypertension with wide pulse pressure,

Moreover, she had resting tremors. Tachycardia can be explained by dehydration but it was relatively more pronounced, and hence added in diagnosis of thyroid storm. Notably, she had no liver, cardiac, or central nervous system involvement; however, she fulfilled other diagnostic criteria, supporting a strong clinical suspicion.

Thyroid storm is managed with both supportive care and specific treatments to address hyperthyroidism and prevent further damage to the thyroid gland. Patients with suspected thyroid storm should be transferred to the ICU for close monitoring, as systemic decompensation is possible.^{1,8} Immediate stabilization involves administering intravenous fluids with dextrose, oxygen, ventilatory support if needed, correction of electrolyte imbalances, management of cardiac arrhythmias, and aggressive treatment of hyperthermia using acetaminophen or physical cooling methods.¹ For symptomatic management, a beta-blocker such as esmolol and propranolol is administered to control adrenergic symptoms. Glucocorticoids, such as hydrocortisone (100 mg IV every eight hours) or dexamethasone (2 mg IV every six hours), are cornerstone treatments.^{1,4,8} These medications not only reduce the peripheral conversion of T4 to T3 but also prevent adrenal insufficiency associated with severe thyrotoxicosis and improve vasomotor stability.⁴

Thionamides, including propylthiouracil (PTU) or methimazole, should be administered promptly. These agents begin inhibiting thyroid hormone synthesis within 1–2 hours. PTU also reduces the conversion of T4 to T3, providing an additional benefit.¹ Iodine compounds, which inhibit thyroid hormone release, can be effective within hours but should be given at least one hour after thionamide administration to prevent the synthesis of new thyroid hormones. Bile acid sequestrants, such as cholestyramine, can serve as adjunctive therapy by reducing thyroid

hormone levels through interference with enterohepatic circulation and increasing fecal excretion. Additionally, plasmapheresis has shown efficacy in reducing free T3 and free T4 levels in thyroid storms, particularly for cases caused by type II amiodarone-induced thyrotoxicosis.⁴

It is critical to identify and treat precipitating factors contributing to the thyroid storm. Infections should be actively investigated and managed with appropriate antibiotics based on culture results or likely pathogens. Other potential triggers, such as trauma, myocardial infarction, diabetic ketoacidosis, or other underlying conditions, should be addressed following standard protocols.

Conclusion

Thyroid storm can present as the initial presentation of thyrotoxicosis. It has high mortality and if detected in time, can save the patient's life. Primary care physicians should be vigilant in detecting this endocrinological emergency and its precipitating factor. Informed consent was obtained from the patient.

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