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Concurrent Thyroid Storm and Diabetic Ketoacidosis Associated With Evolving Autoimmune Diabetes in a Patient With Untreated Hyperthyroidism

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Abstract

Thyroid storm and diabetic ketoacidosis (DKA) are life-threatening endocrine emergencies that rarely occur concurrently. Their coexistence presents significant diagnostic and management challenges, particularly when associated with new-onset autoimmune diabetes.

We report the case of a 26-year-old female with untreated hyperthyroidism who presented with altered mental status, severe tachycardia, dehydration, and generalized weakness. Laboratory evaluation demonstrated thyroid storm with undetectable thyroid-stimulating hormone (TSH) and markedly elevated thyroid hormone levels. Concurrent findings were consistent with severe DKA, including hyperglycemia (828 mg/dL), high anion gap metabolic acidosis, ketonemia, and ketonuria. Hemoglobin A1c was elevated to 9.5% from 5.4% less than three months earlier, suggesting rapid progression to insulin deficiency. Low C-peptide levels of 0.4 further support evolving autoimmune diabetes. The patient additionally demonstrated severe hyperosmolar hypernatremia, acute kidney injury, supraventricular tachycardia, and elevated cardiac biomarkers likely secondary to metabolic stress. She was treated in the intensive care unit (ICU) with aggressive intravenous fluid resuscitation, insulin infusion, electrolyte correction, beta blockade, corticosteroids, and antithyroid therapy, resulting in rapid clinical and biochemical improvement.

This case highlights the rare presentation of concurrent thyroid storm and DKA associated with evolving autoimmune diabetes in a patient without previously established diabetes mellitus. Early recognition and prompt multidisciplinary management are essential to reduce morbidity and mortality.

Categories: Emergency Medicine, Endocrinology/Diabetes/Metabolism
Keywords: autoimmune diabetes, diabetic ketoacidosis (dka), new-onset diabetes mellitus, thyroid-storm, thyrotoxicosis, uncontrolled hyperthyroidism

Introduction

Thyroid storm is a rare but life-threatening endocrine emergency characterized by severe thyrotoxicosis resulting in multiorgan dysfunction and high mortality if not rapidly recognized and treated. Clinical manifestations are variable and may include altered mental status, severe tachyarrhythmias, cardiovascular instability, and metabolic abnormalities [1, 2].

Although hyperthyroidism is known to increase hepatic gluconeogenesis, insulin resistance, and impaired glucose tolerance, concurrent thyroid storm and diabetic ketoacidosis (DKA) remains uncommon [3]. This combination is particularly rare when occurring alongside newly diagnosed type 1 diabetes mellitus, as both conditions may present with overlapping features including dehydration, tachycardia, metabolic derangements, and hemodynamic instability. Excess thyroid hormone may contribute to ketosis through increased catecholamine sensitivity, enhanced hepatic glucose production, and increased metabolic demand, potentially exacerbating underlying insulin deficiency [4].

The coexistence of Graves disease and type 1 diabetes mellitus represents autoimmune polyglandular syndrome type 3 and, although described, simultaneous presentation with thyroid storm and DKA is rare. [5]. This constellation of disease processes may result in severe cardiovascular instability, metabolic collapse, and multiorgan dysfunction, requiring rapid recognition and coordinated multidisciplinary management.

Case Presentation

A 26-year-old female with recently diagnosed hyperthyroidism presented to the emergency department with altered mental status, generalized weakness, and poor oral intake beginning on the day of admission. Three months earlier, she had been hospitalized for thyrotoxicosis with persistent sinus tachycardia and

discharged on methimazole and propranolol with a hemoglobin A1c of 5.4%. However, she was unable to obtain medications or establish outpatient endocrinology follow-up because of lack of insurance coverage. Family history was notable for type 1 diabetes mellitus in her father and thyroid disease in a maternal aunt. Prior to the presentation, she reported progressive fatigue and decreased appetite. She denied fever, chills, cough, chest pain, abdominal pain, nausea, vomiting, dysuria, alcohol use, or illicit drug use.

On presentation, the patient appeared acutely ill with altered mental status and severe dehydration. Initial vital signs demonstrated temperature 36.1°C, blood pressure 123/79 mmHg, heart rate 221 beats/minute, respiratory rate 43 breaths/minute, and oxygen saturation 96% on room air. Physical examination demonstrated a diffuse goiter without palpable nodules. ECG demonstrated supraventricular tachycardia with a ventricular rate of 215 beats/minute, consistent with severe thyrotoxicosis-induced tachyarrhythmia (Figure *1*).

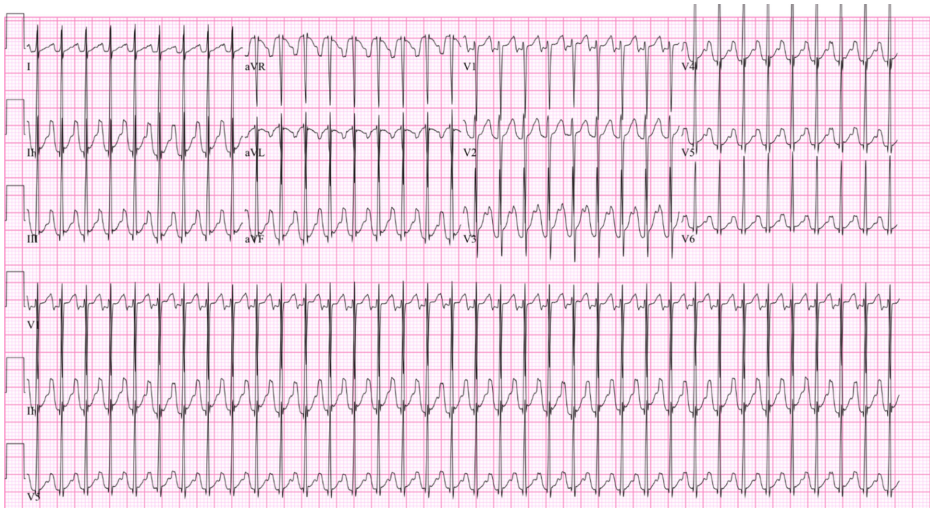


FIGURE 1: ECG demonstrating supraventricular tachycardia with a regular narrow-complex tachycardia. The ventricular rate was approximately 215 beats/minute in the setting of thyroid storm.

Initial laboratory evaluation demonstrated severe thyrotoxicosis, DKA, and profound metabolic derangements (Table *1*). Further endocrine evaluation demonstrated a low C-peptide level of 0.4 ng/mL with markedly positive glutamic acid decarboxylase 65 (GAD-65) autoantibodies at 21,175.0 IU/mL, confirming autoimmune-mediated insulin deficiency consistent with new-onset type 1 diabetes mellitus. Thyroid-stimulating immunoglobulin (TSI) was also elevated at 2.98 IU/L, confirming Graves disease as the underlying etiology of her thyrotoxicosis.

Parameter	Value	Reference Range
Thyroid-stimulating hormone	0.000 µIU/mL	0.45–4.50 µIU/mL
Free thyroxine	6.52 ng/dL	0.82–1.77 ng/dL
Free triiodothyronine	>30 pg/mL	2.0–4.4 pg/mL
Glucose	828 mg/dL	70–110 mg/dL
Hemoglobin A1c	9.5%	<5.7%
Bicarbonate	13.2 mmol/L	22–29 mmol/L
Anion gap	31.8 mmol/L	8–16 mmol/L
Serum acetone	5.2	Negative
Arterial pH	7.16	7.35–7.45
Corrected sodium	168 mmol/L	135–145 mmol/L
Serum osmolality	339 mOsm/kg	275–295 mOsm/kg
Potassium	3.1 mmol/L	3.5–5.0 mmol/L
Creatinine	1.48 mg/dL	0.6–1.2 mg/dL
Troponin	694.7 ng/L	<14 ng/L
Pro-BNP	1,836 pg/mL	<125 pg/mL
Leukocyte count	17.4 ×10³/µL	4.0–11.0 ×10³/µL
Alkaline phosphatase	553 U/L	44–147 U/L
C-peptide	0.4 ng/mL	1.1–4.4 ng/mL
GAD-65 autoantibody	21,175.0 IU/mL	0.0–5.0 IU/mL
Thyroid-stimulating immunoglobulin	2.98 IU/L	0.00–0.55 IU/L

TABLE 1: Pertinent laboratory findings during hospitalization

The patient was diagnosed with thyroid storm complicated by DKA, severe hyperosmolar hypernatremia, and supraventricular tachycardia and was admitted to the intensive care unit (ICU) for close hemodynamic and metabolic monitoring. Initial management included aggressive intravenous fluid resuscitation, electrolyte replacement, insulin infusion per diabetic ketoacidosis protocol, intravenous diltiazem, propranolol, esmolol infusion, propylthiouracil (PTU), and hydrocortisone.

Thyroid ultrasound obtained during her prior hospitalization demonstrated diffuse thyroid enlargement of the thyroid gland with heterogeneous echotexture (Figure *2*) and increased vascularity consistent with acute thyroiditis (Figure *3*). In conjunction with elevated TSI levels, these findings were consistent with Graves disease.

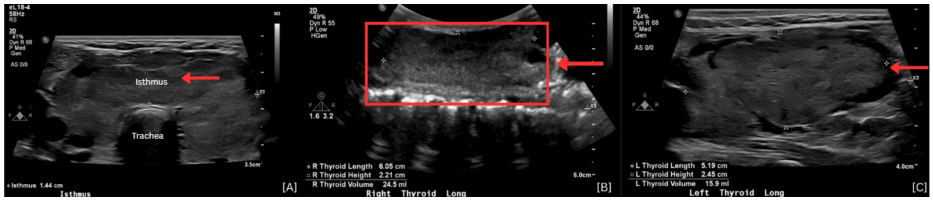


FIGURE 2: Thyroid ultrasound demonstrating diffuse thyroid enlargement and heterogeneous echotexture

(A) Transverse ultrasound image demonstrating diffuse enlargement of the thyroid isthmus measuring 1.44 cm without focal nodularity. The thyroid isthmus is indicated by the red arrow, and the trachea is labeled for anatomical orientation. (B) Longitudinal ultrasound image of the right thyroid lobe measuring 6.0 × 2.2 × 3.5 cm, demonstrating diffuse enlargement and heterogeneous echotexture without a discrete thyroid nodule. The right thyroid lobe is indicated by the red arrow and square. (C) Longitudinal ultrasound image of the left thyroid lobe measuring 5.2 × 2.4 × 2.4 cm, demonstrating diffuse enlargement and heterogeneous echotexture without a discrete thyroid nodule. The left thyroid lobe is indicated by the red arrow.

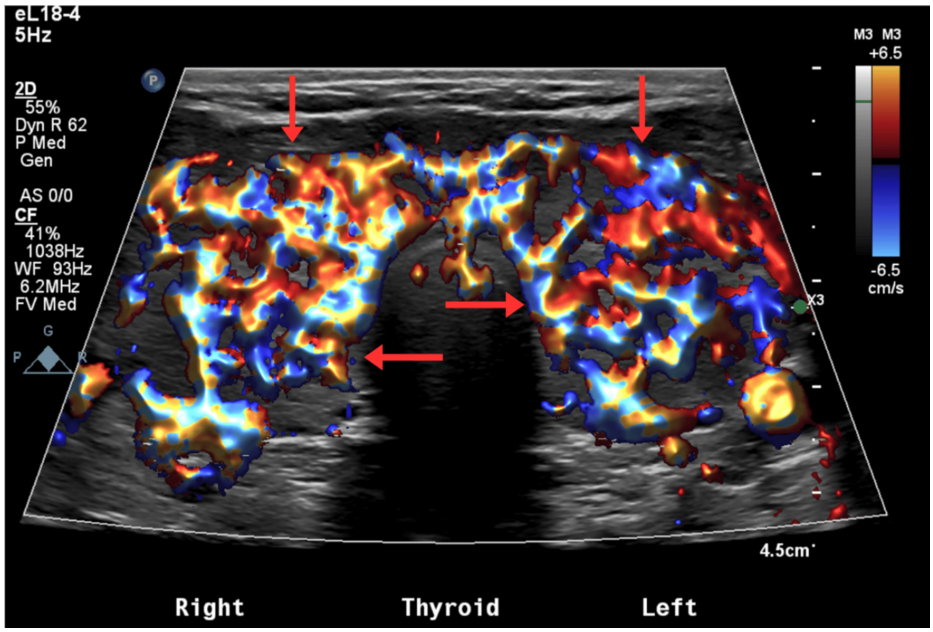


FIGURE 3: Color Doppler ultrasound of the thyroid demonstrating diffuse hypervascularity throughout the thyroid gland, with red arrows indicating representative areas of increased vascularity.

During her ICU course, the patient remained persistently tachycardic with heart rates ranging from 130-170 beats/minute during the first hospital day despite therapy, requiring continued esmolol infusion and escalating propranolol dosing. Over the following 48 hours, her mental status progressively improved with resolution of tachycardia and metabolic acidosis. Serial laboratory studies demonstrated closure of the anion gap from 31.8 to 8.7 mmol/L, with normalization of bicarbonate levels. Corrected serum sodium gradually improved from 168 mmol/L to 146 mmol/L following hypotonic intravenous fluid administration and close electrolyte monitoring.

Following resolution of DKA, the patient was transitioned from insulin infusion to basal-bolus insulin therapy. Repeat thyroid function tests demonstrated improving thyrotoxicosis, with a thyroid-stimulating hormone (TSH) of 0.006 µIU/mL, free thyroxine (T4) of 6.11 ng/dL, and free triiodothyronine (T3) of 7.72 pg/mL. Hydrocortisone and esmolol infusions were subsequently discontinued, PTU was tapered, and the patient was transitioned to methimazole 20 mg three times daily.

By hospital day four, the patient was hemodynamically stable with resolution of supraventricular tachycardia, DKA, and altered mental status, along with significant improvement in hypernatremia. She was transferred from the ICU to the telemetry unit for continued endocrine management and diabetes education. She was discharged on methimazole, propranolol, and basal-bolus insulin therapy with outpatient endocrinology follow-up.

Discussion

Concurrent thyroid storm and DKA represent a rare but potentially fatal combination of endocrine emergencies associated with significant morbidity and mortality. Both conditions may present with overlapping manifestations including tachycardia, altered mental status, dehydration, and metabolic abnormalities, making early diagnosis clinically challenging. Previous literature suggests that concurrent thyroid storm and DKA remain uncommon and are most frequently reported in patients with established diabetes mellitus or medication noncompliance [3].

This case is notable for the rapid progression from previously normal glycemic control to profound insulin deficiency within a three-month period in the setting of severe untreated thyrotoxicosis. During her prior hospitalization, the patient demonstrated hemoglobin A1c of 5.4% without evidence of overt diabetes mellitus. On subsequent presentation, she developed severe DKA with hemoglobin A1c of 9.5% and low C-peptide levels with markedly positive GAD-65 autoantibodies confirming autoimmune-mediated insulin deficiency consistent with new-onset type 1 diabetes mellitus. These findings further supported concurrent autoimmune endocrine involvement affecting both the thyroid and pancreatic β-cells [5]. Family history of type 1 diabetes mellitus and thyroid disease further suggests underlying autoimmune susceptibility.

Excess thyroid hormone contributes significantly to glucose dysregulation through multiple mechanisms, including increased hepatic gluconeogenesis, glycogenolysis, intestinal glucose absorption, accelerated insulin clearance, and peripheral insulin resistance [4]. Enhanced catecholamine sensitivity and hypermetabolic stress may further precipitate ketosis in susceptible individuals [3,4]. Several previously reported cases have described concurrent thyroid storm and DKA in patients with established diabetes mellitus, medication noncompliance, or recurrent DKA episodes [6,7]. Another case report described a similar presentation in a patient without prior history of hyperthyroidism or diabetes mellitus [8]. In contrast, the present case involved a patient with recently diagnosed but untreated hyperthyroidism who subsequently developed severe insulin deficiency and DKA despite absence of overt diabetes mellitus less than three months earlier, suggesting that severe thyrotoxicosis may have contributed to accelerated metabolic decompensation in a genetically predisposed patient.

Autoimmune thyroid disease and autoimmune diabetes mellitus may coexist as part of autoimmune polyglandular syndrome type III, reflecting shared autoimmune susceptibility and underlying immune dysregulation [9]. In this patient, elevated TSI confirmed Graves disease, while markedly positive GAD-65 autoantibodies and low C-peptide levels supported the diagnosis of autoimmune type 1 diabetes mellitus. The simultaneous presentation of Graves disease, thyroid storm, and DKA highlights the importance of recognizing autoimmune endocrine overlap and maintaining a high index of suspicion for concurrent autoimmune disorders in patients presenting with severe endocrine emergencies.

Conclusions

Concurrent thyroid storm and DKA are uncommon but potentially fatal endocrine emergencies requiring rapid recognition and multidisciplinary management. This case highlights the potential for severe untreated Graves disease to precipitate profound metabolic derangements and reveal previously unrecognized autoimmune type 1 diabetes mellitus in genetically predisposed individuals. Clinicians should maintain a high index of suspicion for overlapping autoimmune endocrine disorders in patients presenting with severe metabolic abnormalities and thyrotoxicosis, even in the absence of previously established diabetes mellitus. Early diagnosis, prompt treatment, and coordinated inpatient and outpatient management are essential to improving outcomes and preventing recurrence.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Reem Ekhraiwesh

Acquisition, analysis, or interpretation of data: Reem Ekhraiwesh, Janelle Lopez, Taras Memko, Vijay Dahal, Bryan Dawkins

Drafting of the manuscript: Reem Ekhraiwesh

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Supervision: Janelle Lopez, Bryan Dawkins

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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