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Research Article

A 47-Year-Old Woman with Polyglandular Autoimmune Syndrome III Type A: A Case Report

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Abstract
Background: Polyglandular Autoimmune Syndrome (PAS) are extreme conditions characterized by the functional insufficiency of several endocrine organs due to immunologically mediated destructive processes. This report is showed a rare case of grave diseases. The one of grave disease diagnose is characterized by enlargement thyroid gland. Polyglandular Autoimmune Syndrome III Type A usually occur in adulthood (particularly middle-aged women). This condition can be related to autoimmune thyroid disease and diabetes mellitus type 1.
Case Presentation. A 47-year-old female patient presented to the emergency room at Dr. Soetomo General Academic Hospital, with a chief complaint of generalized weakness. Through comprehensive evaluation, including anamnesis, physical examination, and supporting assessments such as hematology, chest X-ray, EKG, and urinalysis, the patient was diagnosed with diabetic ketoacidosis (DKA), partially compensated metabolic acidosis, impending thyroid storm (Burch-Wartofsky score of 25), and hypovolemic hypotonic hyponatremia. The diagnosis of hyperthyroidism or thyrotoxicosis was confirmed by laboratory results, which included positive thyroid-stimulating hormone receptor (TSH-R) antibodies.
Management and Outcomes. Conservative management was adopted, prioritizing hemorrhage control over immediate platelet reduction. Serial monitoring demonstrated a gradual decline in platelet counts from 945,000/µL to 535,000/µL over one week without pharmacological intervention. Blood smear analysis confirmed reactive thrombocytosis, effectively ruling out myeloproliferative disorders. The patient remained stable throughout the observation period without developing thrombotic complications.
Conclusions. DKA is an emergency complication resulting from uncontrolled diabetes mellitus and can develop rapidly, often within 24 hours, without the patient being aware. Hyperthyroidism can act as an initial trigger for DKA, which may subsequently progress to a thyroid storm. Although thyroid storm and DKA are acute complications that rarely occur simultaneously, their concurrent presentation can be life-threatening and potentially fatal.

Keywords: Polyglandular Autoimmune Syndromes, Diabetic Ketoacidosis, Thyroid Storm

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Introduction

Polyglandular Autoimmune Syndrome (PAS) are severe conditions defined by the functional insufficiency of multiple endocrine organs, stemming from immunologically mediated destructive processes. The autoimmune mechanisms underlying these disorders often involve a progressive T cell–mediated response that results in organ damage and the generation of organ-specific autoantibodies, resembling patterns observed in isolated type 1 diabetes. Various types of PAS have been classified based on genetic predisposition and clinical manifestations (Husebye & Anderson, 2010).

Diabetes mellitus encompasses a spectrum of metabolic disorders characterized by hyperglycemia due to defects in insulin secretion, insulin function, or a combination of both. Persistent hyperglycemia in diabetes mellitus is linked to chronic damage, dysfunction, and failure of multiple organs, including the eyes, kidneys, nerves, heart, and blood vessels (Cho et al., 2018). The prevalence of diabetes mellitus is significant, affecting approximately 10.2 million individuals in the United States. In Indonesia, the prevalence among individuals over 15 years is 1.5-2.3%, with notably higher rates in certain regions, such as Manado, where it reaches 6.1% (3). The incidence of type 2 diabetes mellitus is generally higher in women compared to men (American Diabetes Association, 2016, 2019; Evans, 2019; Soelistijo et al., 2021).

Diabetic ketoacidosis (DKA) is a medical emergency resulting from hyperglycemia, where an excess of acids accumulates in the blood. DKA arises due to severe insulin deficiency and disrupts the metabolism of proteins, carbohydrates, and fats (Loscalzo et al., 2022; Marino, 2013; Setiati, 2017). This condition, sometimes called "accelerated fasting," represents the most critical metabolic complication in insulin-dependent diabetes and requires urgent diagnosis and treatment to mitigate life-threatening outcomes (Benoit et al., 2018; K. K. Dhatariya, 2019).

The diagnostic criteria for DKA in adults with diabetes mellitus can be summarized by the acronym "DKA": 'D' for blood glucose >200 mg/dL (11.1 mmol/L) at presentation, 'K' for serum beta-hydroxybutyrate ≥3.0 mmol/L or urine ketones >2+ on a urine ketone stick, and 'A' for pH <7.3 or serum bicarbonate <15 mmol/L. This definition is in accordance with the International Society of Pediatric and Adolescent Diabetes (ISPAD) (Benoit et al., 2018; K. K. Dhatariya, 2019).

Thyrotoxicosis describes clinical symptoms resulting from excessive thyroid hormones, while hyperthyroidism refers specifically to thyrotoxicosis with thyroid gland hyperactivity. Causes of hyperthyroidism include Graves' disease (most common), toxic adenoma, toxic multinodular goiter, and thyroiditis (Wisnu et al., 2018).

Thyroid hormones and insulin are both pivotal in cellular metabolism. Thyroid dysfunction can adversely affect diabetes mellitus control, while poor glycemic control can impair thyroid function. Research suggests links between thyroid dysfunction and carbohydrate and lipid metabolism, although the underlying mechanisms remain unclear, raising questions about potential coincidence (Wisnu et al., 2018).

A thyroid storm, the most severe form of thyrotoxicosis, is an acute and life-threatening emergency that can lead to irreversible cardiovascular collapse and death if untreated, with an estimated mortality rate of 10%-30%. DKA is similarly life-threatening, caused by profound insulin deficiency or resistance leading to metabolic acidosis, ketosis, hyperglycemia, and electrolyte imbalance. Although thyroid storm and DKA are dangerous in isolation, their concurrent occurrence is rare and potentially fatal, necessitating early diagnosis and aggressive treatment to prevent further decompensation (Griffiths et al., 2020).

Case Presentation

A 47-year-old woman arrived at the Emergency Department of Dr. Soetomo General Academic Hospital, on March 5, 2024, with the main complaint of generalized weakness. The patient was referred from Mitra Keluarga Hospital with a diagnosis of hyperglycemic diabetes mellitus. The patient reported feeling weak for 3 days prior to admission. She also experienced nausea and vomiting 4 times a day for 1 week prior to admission. The patient complained of chills and dizziness and reported a decreased appetite. She mentioned that urination and defecation were within normal limits. The patient had previously been admitted to the Emergency Department at Mitra Keluarga Hospital, where her blood glucose level was recorded at 567 mg/dL.

The patient denied any prior history of diabetes mellitus. She only discovered her high blood glucose levels on Saturday, March 2, 2024. There was no history of hypertension, heart disease, kidney disease, or stroke. The patient stated a family history of diabetes mellitus, as both her father and mother had the condition.

Physical examination revealed that the patient appeared weak, with a Glasgow Coma Scale (GCS) score of E4V5M6, indicating Compos Mentis. Vital sign examination showed blood pressure of 130/82 mmHg, pulse rate of 136 bpm, respiratory rate of 23 breaths/min, temperature of 36.5°C, and oxygen saturation of 99% with a nasal cannula at 2 liters per minute.

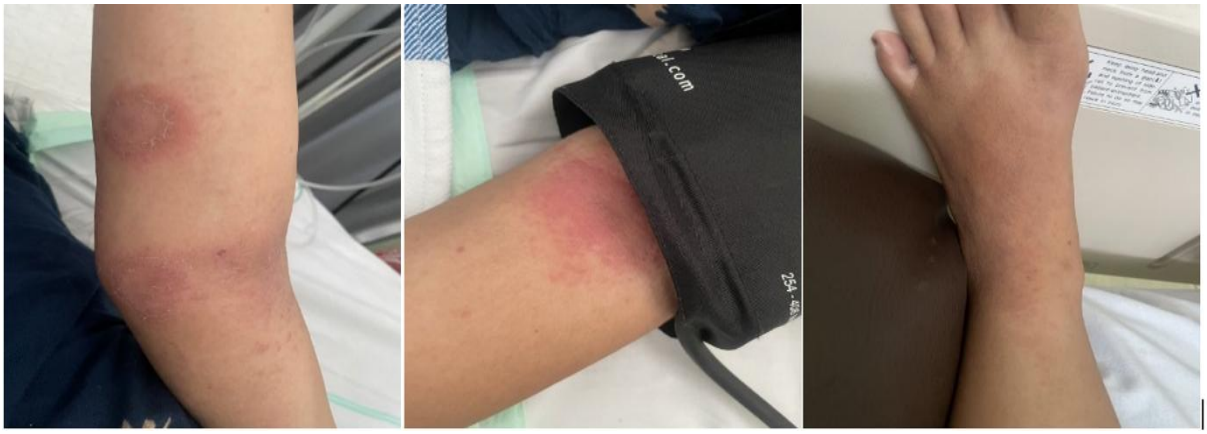

Figure 1 The patient exhibited erythematous and dry skin on the upper and lower extremities.

On general examination, findings were within normal limits. The conjunctivae were not anemic or icteric, and there was no lymph node enlargement. Breath sounds were vesicular without rhonchi or wheezing. The heart sounds (S1) were regular, without murmurs or gallops. The abdomen was supple, with normal bowel sounds and no abdominal tenderness or hepatosplenomegaly. The extremities were warm, with a capillary refill time < 2 seconds, and no edema was observed. Integumentary examination revealed erythematous and dry patches on the upper and lower extremities (Fig. 1).

In the hematology examination, the blood test results were as follows: Hemoglobin (Hb) 12.3 g/dL; Hematocrit 36.8%; Leukocytes 11.76 x 10^3/µL; Platelets 318 x 10^3/µL; Mean Corpuscular Volume 129 mmol/L; Potassium (K) 4.00 mmol/L; Chloride (Cl) 105.0 mmol/L. Arterial blood gas (ABG) analysis results (MCV) 72.6 fL; Mean Corpuscular Hemoglobin (MCH) 24.3 pg; Mean Corpuscular Hemoglobin Concentration (MCHC) 33.4 g/dL; Erythrocytes 5.07 x 10^6/µL; Basophils 0.3%; Eosinophils 0.3%; Neutrophils 70.9%; Lymphocytes 20.3%; Monocytes 8.2%. The clinical chemistry results were: Serum albumin 3.79 g/dL; Random blood glucose 381 mg/dL; HbA1c 11%; Automatic lactate 0.85 mmol/L; APTT 25.5 seconds; PT 10.7 seconds; Procalcitonin 0.02 ng/mL; Blood ketones 0.7 mmol/L; Anti-TSH 11.84 mIU/L. Liver function test results were: SGOT (AST) 18 U/L and SGPT (ALT) 19 U/L. Kidney function test results showed: eGFR 115.1 mL/min/1.73 m²; Creatinine 0.8 mg/dL; BUN 8.0 mg/dL. Electrolyte test results were: Sodium (Na)

were: pH 7.14; PCO2 16 mmHg; PO2 129 mmHg; HCO3 5.4 mmol/L.

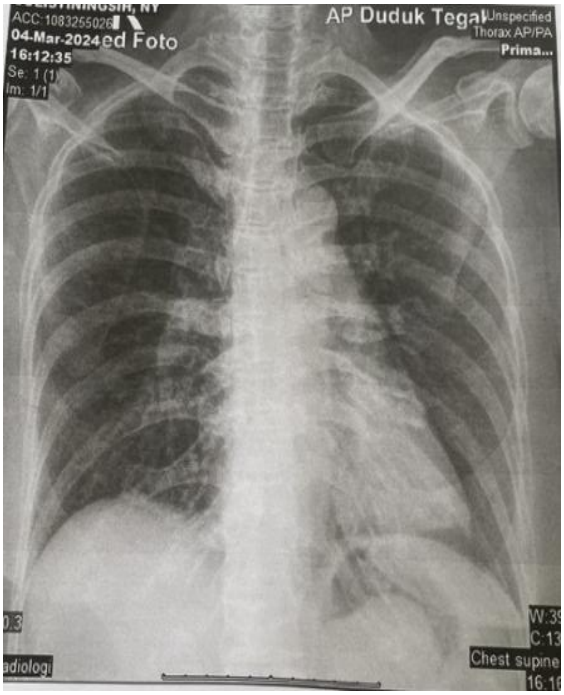

Figure 2 Chest X-ray AP

On the chest X-ray, the AP (anteroposterior) view appeared asymmetrical. Currently, the heart and lungs did not show any abnormalities, and there was an old fracture observed in the left clavicle (Fig. 2). The EKG examination revealed sinus tachycardia (Fig. 3).

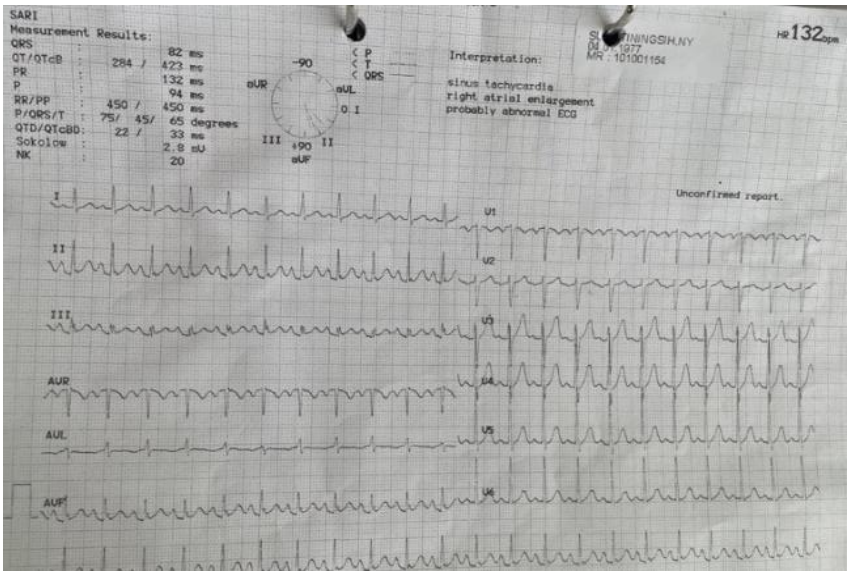

Figure 3 Echocardiogram

Urinalysis revealed yellow, clear urine with a specific gravity of 1.030, pH 5.0, negative for protein, glucose 1+, ketones 3+, negative for bilirubin, erythrocytes, urobilinogen, and nitrites. The working diagnosis for the patient was DKA, partially compensated metabolic acidosis, impending thyroid storm (Burch-Wartofsky score of 25), and hypovolemic hypotonic hyponatremia. The treatment administered included 0.9% normal saline 500 cc over 12 hours, insulin pump at 1 unit/hour, a bolus of Furosemide (1 ampule), followed by maintenance at 3x1, Insulin short-acting 3x8 units, Insulin long-acting 1x16 units, insulin pump at 1 unit/hour, premixed KCL (Potassium Chloride) 50 meq in 500 ml 0.9% normal saline over 12 hours, injection of Methimazole 1 ampule, Ceftriaxone 1 g, and admission to the High Care Unit (HCU).

The patient was discharged on March 10, 2024. On Friday, March 15, 2024, the patient was directed to follow up at Siloam Hospital Surabaya due to having independent health insurance. Laboratory results indicated findings consistent with Graves' disease and non-specific bilateral cervical lymphadenopathy. On Monday, March 18, 2024, the patient was re-admitted to Mitra Keluarga Hospital, Waru, Sidoarjo, with a diagnosis of DKA and impending thyroid crisis.

Discussion

Polyglandular Autoimmune Syndrome (PAS) are entities comprising several distinct conditions, although not all patients present with multiple endocrine abnormalities, and many exhibit non-endocrine autoimmune diseases. Various tissues and organs involved do not necessarily share any specific molecules but can be targeted when the immune system fails to maintain tolerance to different molecules. The main autoimmune polyendocrine syndromes have a strong genetic component, with type 2 syndrome occurring across multiple generations and type 1 syndrome among siblings (Cutolo, 2014).

Patients with PAS-1 and PAS-2 develop multiple diseases over time, and approximately one in seven of their relatives has undiagnosed autoimmune disorders, with hypothyroidism being the most common for type 2 syndrome (Cutolo, 2014). In this case, the patient may potentially have PAS-2, as the patient developed symptoms of diabetes and thyroid disorders.

DKA is an emergency complication resulting from uncontrolled diabetes mellitus (K. K. Dhatariya, 2019). Triggers for DKA include infections, comorbid conditions such as acute coronary syndrome (ACS), issues with insulin pumps, poor adherence, and non-compliance with insulin therapy. Recent studies have highlighted the significant impact of poor treatment adherence on the incidence of DKA. Non-compliance is a primary contributing factor to the development of DKA. Consequently, poor adherence to insulin therapy accounts for more than 50% of DKA hospital admissions in major urban areas (K. Dhatariya, 2016; K. Dhatariya et al., 2016).

The diagnosis of DKA in adults with diabetes mellitus requires three components, remembered by the acronym DKA. 'D' indicates a blood glucose concentration >200 mg/dL or 11.1 mmol/L at the time of initial presentation. For 'K', there must be a serum beta-hydroxybutyrate concentration ≥3.0 mmol/L or urine ketones greater than 2+ on a urine ketone stick. 'A' requires a pH <7.3 or serum bicarbonate <15 mmol/L (K. K. Dhatariya, 2019; Smart et al., 2018). Symptoms of uncontrolled diabetes mellitus may appear over several days, but the specific metabolic changes of DKA typically manifest within a shorter time frame, usually less than 24 hours. In many cases, all symptoms may appear or develop more acutely, and patients can present with DKA without prior signs or symptoms.(American Diabetes Association, 2016; Loscalzo et al., 2022; Setiati, 2017)

The pathophysiology underlying DKA involves reduced circulating insulin activity accompanied by an increase in counter-regulatory stress hormones such as glucagon, epinephrine, norepinephrine, cortisol, and growth hormone (Nyenwe & Kitabchi, 2016). Elevated concentrations of these hormones associated with severe insulin deficiency activate hormone-sensitive lipase in adipose tissue. This enzyme-mediated lipolysis of endogenous triglycerides releases significant amounts of

free fatty acids (FFA) and glycerol into the circulation. These FFAs are oxidized into ketone bodies in the liver mitochondria through a process driven by high glucagon concentrations. Glucagon reduces the hepatic concentration of malonyl-CoA, the initial intermediate in the lipogenic pathway (Loscalzo et al., 2022; Price & Wilson, 2016).

Insulin exerts various effects depending on its circulating concentration. At very low levels, insulin inhibits lipolysis and halts ketone production. At higher concentrations, insulin stimulates glucose uptake into cells, inhibits glycogenolysis, and promotes glycogen synthesis. In the absence of insulin, such as in type 1 diabetes mellitus or when counter-regulatory hormones (cortisol, catecholamines, or glucagon) are elevated due to acute illness, glucose uptake into cells is reduced, necessitating alternative energy substrates. Insulin deficiency increases the activity of hormone-sensitive lipase, resulting in triglyceride breakdown and the release of FFAs (K. K. Dhatariya, 2019).

These FFAs are converted into acetyl coenzyme A (CoA) via beta-oxidation and enter the tricarboxylic acid (TCA) cycle. However, in states of insulin deficiency and high FFA levels, the TCA cycle becomes overwhelmed, leading to the conversion of acetyl CoA into ketones in the liver. This accumulation of ketones results in high anion gap metabolic acidosis, characteristic of DKA (K. K. Dhatariya, 2019). DKA develops rapidly, typically within 24 hours, often without the patient noticing. Symptoms of hyperglycemia include polyuria, polydipsia, polyphagia, and weight loss. Other symptoms include nausea, vomiting, abdominal pain, weakness, and, in severe cases, altered consciousness (Loscalzo et al., 2022).

Initial laboratory evaluation includes plasma glucose measurement, creatinine/blood urea nitrogen, serum ketones, electrolytes (with anion gap calculation), osmolality, urinalysis, urine ketones via dipstick, arterial blood gas analysis, and a complete blood count with differential. Glucose levels can range from 300 to 800 mg/dL, with some patients presenting with lower or higher levels (up to or exceeding 1000 mg/dL), depending on the degree of dehydration. Some patients with severe acidosis may have glucose levels between 100-200 mg/dL, while others might not show DKA even at glucose levels of 400-500 mg/dL (American Diabetes Association, 2016, 2019).

Electrocardiography, chest X-ray, sputum examination, and blood cultures may be performed as indicated to identify DKA triggers.18 Electrolyte measurements, particularly sodium and potassium, should be conducted. The extracellular hyperglycemia effect shifts water to the intravascular space, causing a decrease in serum sodium by approximately 1.6 mEq/L for every 100 mg/dL increase in glucose above 100 mg/dL. As glucose levels decrease, serum sodium levels rise correspondingly. Serum bicarbonate levels are low (0-15 mEq/L), and pH is reduced (6.8-7.3). Low pCO2 levels (10-30 mmHg) reflect respiratory compensation (Kussmaul breathing) for metabolic acidosis. Ketone accumulation is indicated by blood and urine ketone measurements. Arterial blood gas analysis often reveals pH <7.3. Urinalysis confirms the presence of ketone bodies (American Diabetes Association, 2016; Loscalzo et al., 2022).

Initial management of DKA, a complication of type 1 diabetes mellitus, involves assessment of dehydration levels, consciousness (using the Glasgow Coma Scale), and blood and urine sample analysis. Insert an intravenous line concurrently with blood sample collection (Nyenwe & Kitabchi, 2016). Ensure the patient's breathing is adequate; if compromised, initiate resuscitation according to guidelines. Secure the airway in patients with impaired consciousness. Once the airway is secured, insert a nasogastric tube if the patient is comatose or vomiting, and keep the nasogastric tube open for drainage. Attach an EKG to monitor the effects of potassium level changes caused by ketoacidosis and its treatment. Measure urine output to assess fluid balance. In unconscious patients, insert a urinary catheter to facilitate fluid balance measurement (Nyenwe & Kitabchi, 2016).

Rehydration

The patient may require 6-10 L of IV fluid (0.9% normal saline infused at a high rate of 0.5-1 L/hour over 2-3 hours) to replace fluids lost due to polyuria, hyperventilation, diarrhea, and vomiting. Hypotonic saline solution (0.45%) can be used in cases of hypertension or hypernatremia and for those at high risk of heart failure. This solution is preferred (at a rate of 200 to 500 mL/hour for several additional hours) after the initial period, provided blood pressure is stable and sodium levels are not low. The initial choice of fluid is isotonic saline administered at a rate of 15-20 mL/kg body weight per hour or 1-1.5 liters during the first hour. When blood glucose levels reach 300 mg/dL (16.6 mmol/L) or less, the IV solution can be switched to 5% dextrose in water (D5W) to prevent a rapid drop in blood glucose levels. Plasma expanders are unresponsive to IV fluid therapy (Nyenwe & Kitabchi, 2016; Self et al., 2020). The primary goal of fluid correction is the restoration of circulatory volume, clearance of ketones, and correction of electrolyte imbalances.

Restoring Electrolytes

The dilutional effect of hyperglycemia can suppress serum sodium levels in the blood. If sodium levels do not increase while glucose disturbances persist during treatment, or if hyponatremia occurs, this usually indicates excessive volume correction and inadequate electrolyte replacement. Consult a senior physician if sodium levels exceed 160 mmol/L, as this situation places the patient at risk of developing cerebral edema (Loscalzo et al., 2022; Vasilios, 2015).

Close monitoring is essential for DKA patients with elevated potassium levels. Although total body potassium is depleted, mild to moderate hyperkalemia is often seen due to acidosis, which shifts intracellular potassium to the extracellular space, proteolysis, and insulin deficiency. Insulin therapy, acidosis correction, and volume expansion can reduce serum potassium concentration (Wémeau et al., 2018).

To prevent hypokalemia, potassium administration should begin when serum potassium levels fall below 5.3 mEq/L in patients with adequate urine output (50 mL/hour). Typically, 20–30 mEq of potassium in infusion fluids is sufficient to maintain serum potassium concentrations within the normal range of 4–5 mEq/L (Wémeau et al., 2018).

Reversing Acidosis

The acidosis in DKA is reversed using insulin, which inhibits lipolysis. Only regular insulin is used and is infused continuously at a slow rate (e.g., 5 units per hour). IV fluid solutions with higher glucose concentrations, such as normal saline solutions (e.g., D5NS, D5, 45NS), are administered when blood glucose levels reach 250-300 mg/dL (13.8-16.6 mmol/L) to prevent a rapid drop in blood glucose levels. IV insulin should be continued until serum bicarbonate levels rise and the patient is able to eat (K. K. Dhatariya, 2019).

Insulin Therapy

Insulin should be administered until ketones are cleared. Insulin has several effects, including suppression of ketogenesis, reduction of blood glucose, and correction of electrolyte imbalances. Estimation or information about the patient's body weight is necessary for calculating the appropriate dose for therapy. To correct electrolyte imbalances, the patient is given an insulin infusion at a rate of 0.14 units/kg/hour (Loscalzo et al., 2022; Marino, 2013).

A low-dose insulin infusion protocol can reduce blood glucose levels at a rate of 50–75 mg/dL per hour. If blood glucose does not decrease by 10% within the first hour, administer 0.14 U/kg as an intravenous bolus, followed by continuous infusion at the previous rate (Loscalzo et al., 2022; Marino, 2013).

Once ketones have disappeared and blood glucose levels reach 200 mg/dL, the insulin infusion rate is reduced to 0.02–0.05 U/kg/hour. At this stage, dextrose fluids can be administered as needed to maintain blood glucose concentrations between 150 and 200 mg/dL until DKA resolution. Insulin may be given subcutaneously after DKA has resolved and the patient can eat (Loscalzo et al., 2022; Marino, 2013).

Thyroid storm is a severe form of thyrotoxicosis and an acute, life-threatening complication of hyperthyroidism that, if untreated, can lead to irreversible cardiovascular collapse and death. Thyroid storm and DKA are acute, potentially fatal complications arising from pre-existing conditions (American Diabetes Association, 2016). Thyroid function and glucose metabolism are closely related, with normal thyroid function being essential to maintaining glucose metabolism balance. However, excess thyroid hormone levels are thought to be responsible for increased intestinal glucose absorption, enhanced hepatic glucose production from glycogen, reduced pancreatic insulin secretion, increased insulin resistance, and accelerated renal insulin clearance. Previous research has shown that hyperthyroidism worsens glycemic control in diabetic patients (Iino et al., 2022).

Additionally, thyroid storm and DKA share similar tendencies and are often triggered by common factors. Previous reports indicate that initial excessive thyroid hormone levels can trigger DKA, which then precipitates a thyroid storm. The coexistence of diabetes mellitus with hyperthyroidism is a known clinical condition, and hyperthyroidism exacerbates glucose intolerance through various mechanisms. A recent review suggested that efforts should be made to maximize patient adherence to antithyroid and antidiabetic agents when managing concurrent thyroid storm and DKA (Iino et al., 2022).

Graves' disease is suspected in patients presenting with symptoms of tremor, exophthalmos, palpitations, weight loss, and thyroid gland enlargement. The diagnosis is confirmed by laboratory results showing hyperthyroidism or thyrotoxicosis and positive thyroid-stimulating hormone receptor (TSH-R) antibodies. Thyroid ultrasound can reveal a hypoechoic and hypervascular thyroid gland (Kahaly et al., 2018). The Burch-Wartofsky score is a symptom-based scoring system recommended to determine the likelihood of thyroid storm (American Diabetes Association, 2016).

There are three main treatment options for Graves' disease: oral antithyroid drugs (ATDs) such as methimazole, radioiodine therapy, and surgical removal of the thyroid gland (thyroidectomy) (Kahaly et al., 2018).

Conclusions

Thyroid storm and DKA are acute, potentially fatal complications of their respective pre-existing conditions; however, the simultaneous development of both conditions is rare and can ultimately be fatal. Therefore, early diagnosis and aggressive treatment are crucial to prevent further decompensation.

In this case, the patient was diagnosed with DKA, partially compensated metabolic acidosis, impending thyroid storm (Burch-Wartofsky score of 25), and hypovolemic hypotonic hyponatremia. The patient was treated with IV fluids (NaCl 2 liters/24 hours) and an insulin pump at 1 unit/hour, corresponding to a blood glucose level of 381 mg/dL. Subcutaneous insulin (Insulin short-acting 3x8 unit and Insulin long-acting 1x16 unit) was also administered. To prevent hypokalemia, potassium supplementation was initiated when serum potassium levels fell below 5.3 mEq/L, as mild hypokalemia (3.8 mEq/L) was detected. This was corrected with premixed KCL (Potassium Chloride) 50 mEq in 500 mL norma saline over 12 hours and maintained with oral KSR (potassium slow release) 3x1 tablet.

For thyroid management, the patient was given an injection of Methimazole (1 ampule) in accordance with an Anti-TSH level of 11.84 IU/L. As maintenance therapy for thyroid treatment, the patient was prescribed Propranolol 3x20mg and Thiamazole 3x10mg.

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Conflict of interest
The authors declare that there is no conflict of interest.

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