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Thyroid Crisis and Diabetic Ketoacidosis in Uncontrolled Diabetes

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ABSTRACT

Introduction: Thyroid crisis is a medical emergency that causes damage to various organs of the body so that proper diagnosis and immediate treatment are needed. Excess thyroid hormone also affects blood sugar metabolism, especially in diabetic patients. The occurrence of thyroid crisis in conjunction with diabetic ketoacidosis (DKA) is rare, and the exact pathogenesis is still being investigated. We report a case of thyroid crisis in a DKA patient.
Case Illustrations: A 35-year-old woman was brought by her family to the ER due to a loss of consciousness since 6 hours before admission to the hospital. The patient was said to have complained of fever, shortness of breath, palpitations since the previous day. The patient has a history of uncontrolled Type 2 Diabetes Mellitus (DM) since 5 years ago. Physical examination showed somnolence, blood pressure 120/72 mmHg, pulse rate 126 beats per minute, irregular, respiratory rate 28 breaths per minute, fast and deep breaths. Investigations showed blood sugar 320 mg/dL, uncompensated metabolic acidosis, ketonuria. The patient received DKA therapy, but her consciousness and pulse rate had not improved. TSH levels was low with an increase in fT4 levels, with the Burch-Wartofsky score supporting a thyroid crisis. The patient then received treatment for thyroid crisis and her clinical condition showed significant improvement.
Conclusion: The occurrence of thyroid crisis along with DKA is a life-threatening emergency. Untreated hyperthyroid induce severe hyperglycemia and causes acute condition in both organs.

Keywords: thyroid crisis; diabetic ketoacidosis; diabetes mellitus

INTRODUCTION

Thyroid crisis is a life-threatening complication of acute hyperthyroidism, if ignored, can result in cardiovascular diseases and death. In a thyroid crisis, mortality ranges from 10% to 30%[1]. Thyroid crisis is common among women and can strike at any age. Surgery, sepsis, burns, DKA, acute cardiovascular disease, delivery, status epilepticus, radioactive iodine therapy, and the use of iodized contrast material can all be the cause of the trigger[2].

Diabetic ketoacidosis (DKA) is a life-threatening condition caused by severe insulin deficiency or resistance, which results in anion gap metabolic acidosis, ketosis, hyperglycemia, and electrolyte imbalance[3].

Variations in insulin secretion, insulin clearance, gluconeogenesis, glycogen synthesis, glucose oxidation, non-oxidative glucose metabolism, adipokine signaling, and lipid oxidation, according to Potenza M et al. (2009), will increase blood glucose levels and insulin resistance in patients with excessive thyroid hormone levels. This mechanism will disrupt glucose metabolism, resulting in DKA and a thyroid crisis[4].

Thyroid crisis and DKA are both acute, fatal conditions that rarely occur together and can lead to death. As a result, early detection and aggressive treatment are critical in preventing further complications.

CASE PRESENTATION

A 35-year-old woman was brought to the emergency room (ER) by her family because she had been losing consciousness for 6 hours prior to admission. Since previous day, the patient had complained of a fever, shortness of breath, and weakness that did not improve with rest. The patient has a history of diabetes that was diagnosed 5 years ago and has been receiving insulin therapy since 1 year of being diagnosed with DM, but recently the patient was not under control because she felt healthy. There was no family history of similar complaints, no history of thyroid disease, no history of surgery or drug use, and no history of trauma or injury. Physical examination revealed drowsiness, blood pressure of 120/72 mmHg, irregular pulse rate of 126 beats per minute, respiratory rate of 28 times per minute, fast and deep breaths, and a temperature of 38.70C. The head and neck examinations were normal, and the thorax and abdomen examinations revealed no abnormalities. Extremities are palpably warm, with a capillary refill time of 2 seconds. At the time of admission, blood sugar was 320 mg/dL, blood osmolarity was 290 mOsm/L, hemoglobin was 11.7 g/dL, and leukocytes were 15600 cells/10^3. The blood gas analysis results were pH 7.11, base excess -10.2 mmol/L, and HCO3- 11.3 mmol/L. The urinalysis revealed positive urine ketones, positive bacteria, and a leukocyte count of 20-50 cells per field of view. Electrocardiography (ECG) revealed atrial fibrillation, rapid ventricular response, and 146 beats per minute. The results of the chest X-ray were normal. The patient was given 0.9% saline fluid loading therapy, and insulin was given using the DKA procedure with bolus insulin and continuous iv insulin. In addition, the patient was given empiric antibiotic therapy with Ciprofloxacin 400 mg/12 hours iv, and rate control using digoxin 0,25 mg intravenous (iv). Clinically, the patient has not improved in consciousness and is still delirious after 24 hours of treatment, despite the fact that blood sugar has stabilized in the 150-200 mg/dL range, urine ketones are negative, and blood gas analysis is normal. Glycated hemoglobin (HbA1c) level is 10,3%. An ECG examination revealed that the patient was still in atrial fibrillation with a rapid ventricular response. Thyroid stimulating hormone (TSH) levels were 0.005 uIU/mL, and free-T4 (fT4) levels were greater than 100 pmol/L when thyroid function was tested. The “Burch-Wartofsky” score was 55, supporting the diagnosis of thyroid crisis. Propylthiouracil (PTU) 250 mg/4 hours orally, Propranolol 40 mg/4 hours orally, and Hydrocortisone 100 mg/8 hours iv are used to treat thyroid crisis. On the third day of treatment, the patient demonstrated clinical improvement, fully alert, a pulse rate of 98 beats per minute, and regular and stable blood sugar levels in the 170-210 mg/dL range. The patient was allowed to return home with insulin, PTU, and propranolol home therapy after the sixth day of treatment.

DISCUSSION

Thyroid crisis can be caused by a number of factors, including irregular drug intake or discontinuation of anti-thyroid medications, infection, DKA, surgery, radioiodine therapy, adrenocortical insufficiency, and the administration of iodized contrast media[5]. Thyroid crisis is characterized by disorders of various organs such as the cardiovascular system, thermoregulation, gastrointestinal, liver, and central nervous system precipitated by a drastic increase in thyroid hormone release, sympathetic nerve hyperactivity, relative adrenal insufficiency, and increased peripheral cellular response to thyroid hormone[6]. Mortality rates in this disease can reach 10%, and 30% of deaths are caused by shock, multiple organ failure, or disseminated intravascular coagulation[7].

The presence of concurrent DKA conditions can make it difficult to diagnose thyroid crisis. Thyroid hormone stimulates hepatic glucose production while interfering with blood glucose

metabolism by increasing the number of glucose transporters in the hepatocyte plasma membrane. This rise in hepatic glucose production is exacerbated by an increase in free fatty acids caused by catecholamine-induced lipolysis caused by excess thyroid hormone[8].

Concurrent DKA and thyroid crisis are uncommon, but both are fatal emergencies. Because these two diseases can trigger each other, the mechanism of the disease that occurred first is unknown. A detailed history taking is required to ensure the correct diagnosis and etiologic factors[9].

In this case, the patient had been diagnosed with diabetes since the age of 30 and had no family history of the disease. One year after being diagnosed with diabetes, the patient began taking insulin. This supports the suspicion of adult-onset latent autoimmune diabetes (LADA). Concurrent thyroid crisis suggest that the patient has autoimmune polyglandular syndrome type 3 (APS3), characterized by autoimmune thyroiditis. Anti-GAD antibodies (glutamic acid decarboxylase) and anti-Islet cell antibodies should be tested to confirm the diagnosis of LADA, and anti-TPO antibodies (thyroid peroxidase) should be tested to confirm the diagnosis of autoimmune thyroiditis[10,11].

Poor medication adherence is a common cause of thyroid crisis and DKA. A systematic review of previous case reports revealed that noncompliance with anti-thyroid and anti-diabetic medications was the most common precipitating factor. This assertion is supported by evidence of elevated HbA1C and fT4 levels in these case reports, as well as low TSH levels[12]. Thyroid crisis and DKA have similar predisposing factors, so a common precipitating factor, such as infection or an uncontrolled disease condition, can trigger both at the same time.

CONCLUSION

Thyroid crisis and DKA rarely occur together, but this condition is a life-threatening emergency that requires prompt diagnosis and treatment. To prevent recurrence, the etiological factors must be tracked and the disease must be treated comprehensively, including education on anti-thyroid and anti-diabetic drug adherence.

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