



Thyroid Storm Associated with Diabetic Ketoacidosis: A Case Report

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ABSTRACT

Various autoimmune diseases may accompany type 1 diabetes. Autoimmune thyroid diseases are common in these patients. Herein, we presented a type 1 diabetic female patient with diabetic ketoacidosis accompanied by a thyroid storm.

Turk J Int Med 2022;4(Supplement 1):S166-S168

DOI: [10.46310/tjim.1072719](https://doi.org/10.46310/tjim.1072719)

Keywords: Autoimmune diseases, type 1 diabetes mellitus, thyroid diseases, hyperthyroidism, diabetic ketoacidosis, thyroid storm.

Introduction

Type 1 diabetes is an autoimmune disease in which genetic and immunological factors play a role. 5-10% of diabetes in adult patients is type 1 diabetes.¹ It is caused by insulin deficiency due to immune-mediated destruction of pancreatic beta cells. It constitutes approximately 80% of newly diagnosed diabetes cases in patients under 19.² While non-acidosis hyperglycemia is typically the initial symptom in children, they are diagnosed

with diabetic ketoacidosis with the second frequency.³ Type 1 diabetes may be accompanied by autoimmune diseases such as Graves disease, celiac disease, and primary adrenal insufficiency.⁴ Autoimmune thyroid diseases are the most common immunological disease in these patients. Hyperthyroidism is observed in approximately 1-2% of patients. We aimed to present a case with graves who had type 1 diabetes and was diagnosed with thyroid storm.



Case Report

A 22-year-old female patient presented to the emergency department with complaints of nausea, vomiting, palpitations, and shortness of breath. She had a known diagnosis of type 1 diabetes mellitus (DM) since nine, uses an insulin pump, and has no comorbidities. At presentation, the patient was conscious, oriented, and cooperative. The respiratory sounds were bilaterally equal and natural, the abdomen was relaxed (no defense and rebound), no pretibial edema, peripheral pulses were palpable and equal. In her neurological examination, muscle strength was 5/5, cerebellar and cranial nerve examinations were regular, and she did not find neck rigidity. She had a history of diabetic ketoacidosis four years ago. Her mother had a thyroid nodule and thyrotoxicosis in her four aunts.

Sinus tachycardia was present in the patient's electrocardiography (ECG) (beats 156/min). In her laboratory, capillary blood glucose was 308 mg/dL, no electrolyte imbalance, and standard kidney function tests. There were elevated liver function tests in ALT dominance (ALT: 86 U/L, AST: 53 U/L). We detected bilirubin elevation in indirect bilirubin dominance. (total bilirubin: 2.4 mg/dL, direct bilirubin: 0.33 mg/dL). There was neutrophilic leukocytosis in the hemogram (leukocyte: 14,100/mm³, neutrophil: 9,470/mm³), and CRP was negative. There was decompensated metabolic acidosis in the arterial blood gas of the patient. (pH: 7.19, pCO₂: 34, pO₂: 78, SpO₂: 91%, HCO₃: 13.5 mmol/L). In the complete urinalysis, the pH was 5, protein negative, glucose 4+, ketone 2+, erythrocyte/leukocyte/bacteria negative. We detected pulmonary edema in the thorax computed tomography.

The patient, whose complaints increased and acidosis worsened during the emergency follow-up, was followed up in the intensive care unit (ICU). The patient, who developed respiratory distress during her follow-up in the ICU, was followed up with elective intubation. As the acute phase reactants increased, we started broad-spectrum antibiotic treatment (piperacillin/tazobactam, teicoplanin, oseltamivir) and methylprednisolone. We performed drainage was due to pleural fluid. We detected in the thyroid function tests (TFT)

sent during the patient's hospitalization, TSH: 0 mIU/mL, free T4: 5 ng/dL, free T3: 20 ng/L, and sent control TFT one week later. We observed TSH: 0.01 mIU/mL, free T4: 2.22 ng/dL, free T3: 10.78 ng/L. In the neck ultrasonography, the thyroid parenchyma had a heterogeneous hypoechoic appearance. The gland vascularization increased, and it was evaluated as subacute thyroiditis consistent with the elevation of thyroid hormone. Considering thyroid storm, we started propylthiouracil (PTU) and hydrocortisone 4x50 mg and stopped methylprednisolone. Cardiology performed transthoracic echocardiography because of bilateral pleural effusion; ejection fraction was expected, and pulmonary artery pressure was found to be high. On the other hand, in the lower extremity doppler ultrasonography was taken due to the increase in diameter in the bilateral lower extremities of the patient, a thrombus in the deep venous system, which may be compatible with the acute or chronic period, was observed.

During follow-up, ALT increased 10-fold, so we discontinued PTU and steroid therapy. We planned thyroid apheresis, and a dialysis catheter was inserted through the right femoral vein. Fresh frozen plasma was prepared, and the patient started apheresis treatment. We added propranolol because of tachycardia and applied apheresis for a total of eight doses. We planned a total thyroidectomy for the patient, who was evaluated in the endocrinology, and ear, nose, and throat diseases committee because she could not take anti-thyroid medication and be diagnosed with Graves' disease. The committee deemed it appropriate to make the patient euthyroid by applying high-dose iodine treatment and re-plasmapheresis before the operation. ENT on the patient performed total thyroidectomy.

We started calcium D3 and levothyroxine due to transient hypoparathyroidism in the post-operative period (TSH: 0.01 mIU/mL, free T4: 1.37 ng/dL, free T3: 2.28 ng/L, parathormone: 10 ng/L). Thyroidectomy pathology, diffuse hyperplasia, benign lymph node, and parathyroid tissue were reported. The council deemed it appropriate to make the patient euthyroid by applying high-dose iodine treatment and re-plasmapheresis before the operation. We discharged the patient without complications after the operation. Calcium was

discontinued during outpatient follow-ups, and the levothyroxine dose was increased to 125 mcg. We turned off the insulin pump, we started long, and short-acting insulin therapy.

Discussion

It is common for individuals diagnosed with an autoimmune disease to be accompanied by another autoimmune disease. In the follow-up of diagnosed patients, it may be necessary to closely follow up and examine other possible conditions in line with their complaints. Type 1 DM is the most common autoimmune disease highly related to thyroid diseases.⁵ For this reason, the controls of type 1 DM patients or seeing TFT at the first application are among the situations that should be careful. Hypothyroidism is the most common disease with type 1 DM, and the diagnosis of thyroid disease is usually made before diabetes.⁶

In our case, in a patient with a known diagnosis of type 1 DM, diabetic ketoacidosis and thyroid storm were observed simultaneously. We wanted to present it because the patient underwent surgery later in the clinical process.

Acknowledgment

This study has been presented in 18th Uludag Internal Medicine National Winter Congress, 7th Bursa Family Medicine Association National Congress, 12th Uludag Internal Medicine Nursing Congress, 3-6 March 2022, Bursa, Turkey.

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contribution

Study Conception: NGA; Study Design: CA; Supervision: EH; Materials: EA; Data Collection and/or Processing: YC, BO, FO; Statistical Analysis and/or Data Interpretation: CE; Literature Review: OOG; Manuscript Preparation: SC.

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