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Case Report

A case report of euglycemic ketoacidosis due to dapagliflozin treatment

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Abstract

Diabetic ketoacidosis (DKA) is a leading cause of mortality and morbidity in type 2 diabetic patients. Sodium-glucose co-transporter (SGLT-2) inhibitors are a new antidiabetic treatment class that increases the renal excretion of glucose. The Food and Drug Administration issued a warning in May 2015 notifying that patients using this class of anti-diabetic drugs may develop DKA. Risk factors for DKA development among patients who take SGLT-2 inhibitors include carbohydrate intake/starvation or acute illness. In the current report, we aimed to present a case of euglycemic DKA using dapagliflozin treatment.

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Keywords: *dapagliflozin, sodium-glucose co-transporter 2 inhibitors, euglycemic ketoacidosis*

Introduction

Recently, new drugs have been developed that reduce plasma glucose levels by inhibiting sodium-glucose co-transporter 2 (SGLT-2) that provide glucose reabsorption in the ultrafiltrate. SGLT-2 inhibitors also called "gliflozins", reduce glucose reabsorption by causing SGLT2 inhibition in the proximal tubules in the kidneys and increase urinary glucose excretion.[1] Dapagliflozin was approved by Europe and the United States as the SGLT-2 inhibitor to be used in the treatment of diabetes. Euglycemic ketoacidosis (EKA) is a rare form of diabetic ketoacidosis that occurs without a severely elevated blood sugar level.[2] It has been reported that patients using SGLT-2 inhibitors have developed EKA as a side effect in the follow-up.[3–5] The mechanism by which SGLT-2 inhibitors cause the development of EKA is not fully elucidated. It is thought that the decrease in insulin secretion from the pancreas due to urinary glucose excretion may increase the glucagon/insulin ratio, and this situation predisposes to increased gluconeogenesis, ketogenesis, and ketoacidosis.[3,6] Herein, we aimed to present an EKA case that developed after the use of dapagliflozin treatment.

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Case Report

A 57-year-old male patient, who had diabetes for 20 years, was admitted to our center with complaints of nausea, dizziness, and weakness. There was no pathological finding in the physical examination of the patient. In venous blood gas, pH: 7.3, lactate: 2.6 mmol/L, and HCO3: 10 mmol/L was detected. In the biochemical analysis, fasting plasma glucose was 167 mg/dL, creatinine level 0.83 mg/dL, and HbA1c level was 9.5%. Urinalysis showed ++ ketone and ++++ glucose, despite serum glucose was below 200 mg/dL. It was learned that the patient used metformin 1000 mg twice daily, linagliptin 5 mg once daily, and dapagliflozin 10 mg once daily for the treatment of diabetes. The patient was diagnosed with EKA. Dapagliflozin treatment was stopped, and insulin infusion was started in addition to fluid therapy. The patient's acidosis status improved with urine ketone becoming negative after intravenous fluid therapy and insulin treatment. Diabetes treatment of the patient has arranged as insulin glargine 14 units in addition to vildagliptin 50 mg twice daily and metformin 1000 mg twice daily. The patient was discharged by recommending outpatient follow-up.

Discussion

If there is no contraindication, SGLT-2 inhibitors are recommended as antidiabetic drugs in patients with a history of atherosclerotic cardiovascular disease, a history of heart failure, or a high risk of atherosclerotic cardiovascular disease.7 Before giving SGLT-2 inhibitors in treatment, a detailed history should be taken, and factors that may predispose the patient to ketoacidosis should be evaluated. SGLT-2 inhibitors should be used with caution in patients with low insulin reserve, restricted oral intake, decreased carbohydrate intake, and cases where insulin needs increase due to acute medical illness or surgery.3,6,8 EKA should be kept in mind in the differential diagnosis, especially in patients using SGLT-2 inhibitors and in whom acidosis is found despite not having very elevated blood sugar levels.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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