Euglycemic Diabetic Ketoacidosis Induced by Jardiance in a 37-Year-Old Female: A Case Report

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Background: Diabetic ketoacidosis (DKA) is a complication of diabetes, very common throughout the Rio Grande Valley region, can be life threatening and is characterized by hyperglycemia, ketonemia and metabolic acidosis. Nevertheless, euglycemic diabetic ketoacidosis (euglycemic DKA) is an atypical presentation in which patients show the classic signs of DKA but with normal or near-normal blood glucose levels. The use of sodium-glucose cotransporter 2 (SGLT2) inhibitors, such as Jardiance (empagliflozin), has been associated with an increased risk of euglycemic DKA. This case report describes a case of euglycemic DKA in a 37-year-old woman with type 2 diabetes mellitus who had been on treatment with Jardiance for the past 9 months.

Case Presentation: A 37-year-old female patient with a past medical history of type 2 diabetes mellitus presented to the emergency department with complaints of nausea, multiple emetic episodes, associated with abdominal pain and fatigue. Patient denies febrile episodes or recent infections, denies dietary indiscretions. Reported to be adherent to her medical management for diabetes which consists of Jardiance 1000 mg twice a day. On physical examination, the patient was dehydrated and tachycardic. Noticeably, her blood glucose of 129 mg/dL, within normal range.

Laboratory workup on admission revealed a high anion gap metabolic acidosis, with the following results: arterial blood gas (ABG) showed a pH of 7.1, bicarbonate of 8.5 mEq/L and an anion gap of 24. Serum ketones were markedly elevated, confirming the diagnosis of DKA. In spite of his normoglycemia, the diagnosis of euglycemic DKA was established.

Management and Outcome: The patient immediately received intravenous fluids to address volume depletion and electrolyte imbalances. She was transferred to the ICU for close monitoring. An insulin dextrose infusion protocol was started to manage ketosis and acidosis, despite her normal glucose levels. Electrolyte replacement, particularly potassium, was also administered to prevent hypokalemia, a frequent complication during the treatment of ketoacidosis. During her hospital stay, continuous monitoring of vital signs and blood glucose was done. Treatment goals were guided by anion gap closure.

In the following 72 hours, the patient's acidosis began to disappear, the anion gap closed and the patient tolerated the diet adequately. Her symptoms gradually improved and she was switched to subcutaneous insulin therapy. Given the

unusual presentation and possible association with Jardiance, the decision was made to discontinue the SGLT2 inhibitor. The patient was discharged in stable condition with a revised diabetes management plan, including close outpatient follow-up with her endocrinologist and primary care physician.

Conclusion: Euglycemic diabetic ketoacidosis is a challenging clinical diagnosis due to its atypical presentation, which can delay the initiation of treatment and increase the risk of complications. SGLT2 inhibitors like Jardiance are well-known triggers of euglycemic DKA. The mechanism involves an increase in urinary glucose excretion, decreasing plasma glucose levels and thereby promoting ketogenesis, mainly in situations of stress or insulin deficiency. This case report highlights the importance of recognizing euglycemic DKA in patients treated with SGLT2 inhibitors such as Jardiance. Physicians should have a high index of suspicion for this disease in diabetic patients presenting with unexplained metabolic acidosis and normal glucose levels. Early diagnosis and prompt treatment are crucial to prevent serious outcomes.