EUGLYCEMIC DIABETIC KETOACIDOSIS CAUSED BY EMPAGLIFLOZIN

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INTRODUCTION: Empagliflozin is an antidiabetic medication that works by inhibiting the sodium-glucose cotransporter-2. It has become increasingly popular after the EMPA-REG OUTCOME trial demonstrated Empagliflozin may have cardioprotective properties in patients with type 2 diabetes mellitus. However, in a subgroup of patients, the harm of its adverse effects may outweigh cardiovascular benefit. Euglycemic diabetic ketoacidosis (DKA) is a known, possible adverse, and life threatening side effect of Empagliflozin. We describe a case of severe euglycemic DKA caused by Empagliflozin.

CASE PRESENTATION: A 60 year old male presented to an outside hospital with progressive dyspnea and lethargy that began the night prior to presentation. Medical history included type 2 insulin dependent diabetes. Surgical history and social history were non-contributory. Vital signs on presentation demonstrated tachycardia and hypotension. Pertinent exam findings were noncontributory. Labs revealed arterial blood pH of 6.8, white blood cell count of 15 (x 10^3 ml), bicarbonate less than 8 mmol/L, anion gap of 30, blood glucose of 215 mg/dL, beta-hydroxybutyrate of 12.00 mmol/L. Due to his critical state and concern for DKA, he was intubated at the outside hospital, started on an insulin drip, and transferred to our intensive care unit for higher level of care. Upon arrival, a central hemodialysis line was placed in the setting of acute renal failure. He presented meeting SIRS criteria and required norepinephrine for blood pressure support. Broad spectrum antibiotics, insulin drip, and bicarbonate drip were initiated. Infectious workup was negative and antibiotics were stopped. Discussion with family revealed that patient was started on Empagliflozin one month prior to presentation. Additional workup including toxic ingestion was unremarkable. The diagnosis of Empagliflozin associated euglycemic diabetic ketoacidosis was subsequently made. The patient was treated per standard DKA treatment protocols and rapidly recovered. He was ultimately discharged home with strict instruction to avoid Empagliflozin.

DISCUSSION: As new drugs are marketed and indications for existing drugs grow, providers must be aware of both the benefits and harms that these drugs possess. Empagliflozin has recently been shown to demonstrate cardioprotective properties; however, this comes at the cost of potential adverse advents such as euglycemic DKA. Additionally, euglycemic DKA must be on the differential in a patient with unremarkable blood glucose levels and metabolic acidosis, as this is a simple condition to treat and a lethal diagnosis to miss.

CONCLUSIONS: Although studies suggest Empagliflozin may have cardioprotective properties, providers must also be weary of its ability to cause euglycemic diabetic ketoacidosis. Additionally, it is important to include euglycemic DKA in one's differential when a patient presents with metabolic acidosis.


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