

SGLT2 Inhibitor Induced Hyponatremia and Euglycemic Diabetic Ketoacidosis (EDKA)

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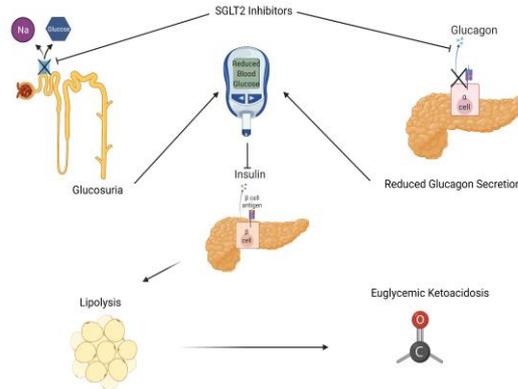
Introduction

Diabetic ketoacidosis is generally associated with uncontrolled Diabetes mellitus resulting in a blood glucose >300 and a metabolic gap acidosis causing nausea and vomiting. EDKA occurs with a blood sugar <250 meeting the other criteria. This case report demonstrates how SGLT2 inhibitors caused both EDKA and hyponatremia concurrently in our patient.

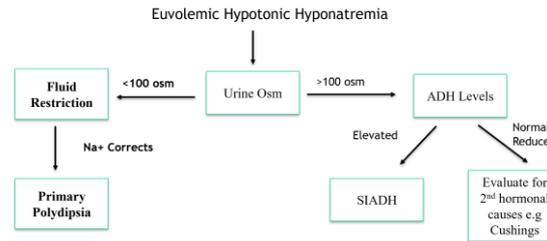
Case Report:

A 54-year-old woman with a past medical history of type II diabetes mellitus treated with metformin and 6-months of empagliflozin presented to the emergency department with a 24-hour history of nausea and vomiting. Labs were significant for an elevated beta hydroxybutyrate of 18 mmol/L, an elevated anion gap of 18, a glucose of 188 mg/dL, and a sodium of 123 mmol. After receiving the normal saline bolus in the ED, her diabetes medications were held, and the anion gap closed without insulin over the course of the next 8 hours. Further history revealed she had consumed 3 L of water per day, along with 4 to 5 cups of hot tea. She had increased her liquid intake after noticing she had been going to the bathroom more often since starting the empagliflozin. She was placed on fluid restriction and her hyponatremia resolved within two days of monitoring. Her SGLT2 inhibitor was discontinued, and she was discharged with close primary care follow up.

Proposed Mechanism of EDKA:



Diagnosis of Euvolemic Hypotonic Hyponatremia:



Discussion:

SGLT2 inhibitors uniquely reduce blood glucose via an insulin independent mechanism

Long-term SGLT2 inhibitors can reduce endogenous insulin production and potentially induce ketoacidosis in insulin insensitive patients

Euvolemic hypotonic hyponatremia that corrects with fluid restriction suggests psychogenic polydipsia

Hyponatremia can further precipitate metabolic dysregulation causing EDKA

Conclusions:

EDKA is a rare side effect of SGLT2 inhibitors that requires urgent management .

SGLT2 inhibitor diuresis can induce secondary polydipsia and subsequent hyponatremia.

Patient counseling on fluid intake and signs of EDKA could prevent complications, especially as SGLT2 inhibitors are more commonly prescribed.