**Background**

Diabetic ketoacidosis (DKA) is defined by metabolic acidosis, ketosis and hyperglycemia. It is considered to be a consequence of significant insulin deficiency and/or insulin resistance and is usually precipitated by the presence of hyperglucagonemia or other counterregulatory hormones. In patients on oral sodium-glucose cotransporter 2 (SGLT2) inhibitors, decreased carbohydrate availability through renal glucose excretion can cause serum glucose levels to be lower than what is normally seen (< 200 mg/dL) in DKA cases, masking the diagnosis. This phenomenon is termed euglycemic DKA (EuDKA). Existing evidence suggests that EuDKA in the setting of SGLT2 inhibitor use is rare but can occur in patients with either type 1 diabetes mellitus (T1D) or type 2 diabetes mellitus (T2D). Most published reports of EuDKA in patients with T2D describe patients on SGLT2 inhibitors with clear inciting events such as decreased insulin doses, surgery, or severe acute illness. To our knowledge, none have reported EuDKA precipitated by ertugliflozin. This is also the first report of EuDKA of a patient in the United States with T2D initiating SGLT-2 inhibitor use while on a low carbohydrate diet.

**Clinical Case**

A 53-year-old female with a history of poorly controlled T2D was admitted to the hospital with EuDKA within seven days of starting ertugliflozin and alogliptin. Patient admitted to strict adherence to a low-carbohydrate diet for one week prior to admission. On admission, the patient was afebrile. Initial labs showed blood glucose 104 mg/dL, serum bicarbonate 8 mmol/L, anion gap 22, pH 7.100, beta-hydroxybutyrate 66.94 ng/mL (0.20-2.81), and a hemoglobin A1c of 11.2%. Urinalysis revealed glucosuria ≥500 mg/dL, ketonuria 80 mg/dL, hyaline cast 20/lpf, no nitrites or leukocyte esterase, WBC 1/hpf. Flu PCR negative. WBC count was 17.4 x10e3/uL initially, though all CBC cell lines decreased with fluid administration. CXR was negative for acute pulmonary disease. All oral T2D agents were held and our patient was initiated on a DKA protocol based on ADA guidelines (below). Her EuDKA subsequently resolved with successful transition to a weight-based basal-bolus insulin regimen.

**Conclusions**

There are no published case reports identifying patients with T2D developing euglycemic DKA precipitated only by a low carbohydrate diet and ertugliflozin initiation. We hypothesize that our patient’s ketogenic diet lowered the threshold for a euglycemic ketoacid crisis resulting directly from the new addition of the SGLT2 inhibitor in the setting of pre-existing glucose toxicity. In patients considering, starting and being maintained on ertugliflozin or other SGLT2 inhibitors, the importance of effective, early and frequent dietary counseling with close follow-up cannot be overstated. Further, this report of EuDKA in a patient starting ertugliflozin supports that EuDKA is an SGLT2 inhibitor class risk.

**References**