A short note on pathophysiology involved in euglycemic diabetic ketoacidosis

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Introduction

Euglycaemic Diabetic Ketoacidosis (EDKA) is a clinical syndrome present in both type 1 and type 2 diabetes mellitus, and in the presence of severe metabolic acidosis and ketonemia characterized by euglycemia. DKA is one of the most serious and life-threatening complications of diabetes mellitus and is seen in a wide variety of conditions. Diagnosis is also often delayed, posing diagnostic challenges to physicians due to various etiologies and normal blood glucose levels. EDKA in a diabetic has many known causes. The overall mechanism is based on general starvation leading to ketosis while maintaining euglycemia. Thus, conditions such as anorexia, gastroparesis, fasting, use of ketogenic diets, and alcohol use disorders can lead to carbohydrate deprivation and subsequent ketosis. Other triggers for EDKA include pregnancy, pancreatitis, glycogen storage disorders, surgery, infections, cocaine addiction, cirrhosis, and insulin pump use.

Description

A new category of oral antidiabetic agents of SGLT2 inhibitors, including canagliflozin, dapagliflozin, empagliflozin, or ertugliflozin, may also lead directly to EDKA. The underlying mechanism of EDKA follows carbohydrate starvation, which generally results in decreased serum insulin and excess counter-regulatory hormones such as glucagon, epinephrine and cortisol. Increased glucagon/insulin ratio leads to increased lipolysis, increased free fatty acids, and ketoacidosis. Ketone body formation with EDKA is similar to DKA with acetoacetate and acetone. The resulting anion-gap metabolic acidosis causes a sensation of respiratory compensation and dyspnea, as well as nausea, anorexia, and vomiting. Decreased oral intake, vomiting, and volume depletion due to osmotic diuresis from diabetes aggravate elevated glucagon, cortisol, and epinephrine, impairing lipolysis and ketogenesis. In addition, decreased hepatic gluconeogenesis occurs during fasting if liver glycogen is already depleted or if increased renal diabetes contributes to this EDKA.

Patients using insulin often do not recognize symptoms as DKA because serum glucose does not rise and insulin doses can be maintained or reduced. Furthermore, SGLT2 inhibitors have been found to directly stimulate glucagon release from the pancreas, further exacerbating the glucagon/insulin imbalance and inhibiting renal elimination of hydroxybutyrate and acetoacetate is a risk factor for EDKA due to increased hypoinsulinemia physiologic status and starvation. Elevated levels of cortisol and placental lactogens can lead to insulin resistance and episodes of vomiting and fasting can lead to excessive starvation ketosis make it worse. Alcoholic ketoacidosis can present with symptoms similar to EDKA, with anorexia, vomiting, dyspnea, and significant anion gap metabolic acidosis and ketonemia.[1-4]

Conclusion

Signs and symptoms vary from case to case, but may be similar to hyperglycemic DKA but without polyuria, polydipsia, or severe mental status changes. EDKA patients may present with nausea, vomiting, shortness of breath, general malaise, lethargy, loss of appetite, fatigue, or abdominal pain. Patients may not exhibit polydipsia or polyuria because serum glucose is normal. Patients may exhibit deep, rapid breathing known as kussmaul respiration, which is the respiratory compensation for severe metabolic acidosis. Due to the loss of acetone, you may have a fruity bad breath. Tachycardia, hypotension, altered mental status, increased skin tone, and delayed capillary refill are signs of global fluid loss. In severe cases, severe dehydration and metabolic disturbances can lead to hypovolemic shock, lethargy, respiratory failure, coma, and death.

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Conflict of Interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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