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Euglycemic Ketoacidosis: Etiologies, Pathogenesis, and Management

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ABSTRACT

Euglycemic ketoacidosis (EKA) is a life-threatening emergency seen in diabetic and non-diabetic populations, such as individuals with chronic alcohol ingestion, starvation, pregnancy, and lactating women. More recently, an increasing incidence has been noted with the rising popularity of sodium-glucose cotransporter-2 (SGLT2) inhibitors for treating type 2 diabetes, heart failure, and kidney disease. This condition is characterized by euglycemia to milder degrees of hyperglycemia, metabolic acidosis, and ketonemia. Near-normal glucose levels can often mislead clinicians, resulting in a delayed diagnosis and treatment of this potentially devastating metabolic condition with consequences ranging from hemodynamic instability and electrolyte disturbances to seizures and cerebral edema. The objective of our review is to describe and educate, in detail, all the common etiologies for euglycemic ketoacidosis in diabetic as well as non-diabetic patients.

INTRODUCTION AND BACKGROUND

Euglycemic ketoacidosis (EKA) is a metabolic emergency seen in both diabetic and non-diabetic patients, marked by the presence of euglycemia (serum glucose < 250 mg/dl), metabolic acidosis (pH <7.3, serum bicarbonate <18 mEq/L), and ketoacidosis.[1,2]

In 1973, Munro et al. were the first to describe this clinical state in young insulin-dependent diabetic patients, with one of them having previously gone undiagnosed with diabetes.[3] Jenkin et al. then published a more thorough case series in 1993.[4] It has been reported in pancreatitis, starvation, pregnancy, cocaine abuse, sepsis, heavy alcohol use, chronic liver disease including liver cirrhosis, surgery, and more recently, with the use of sodium-glucose cotransporter-2 (SGLT2) inhibitors for the management of type 2 diabetes mellitus, heart failure, and kidney disease (Table 1).[1,3–14]

Diabetic ketoacidosis (DKA) is seen in patients with type 1 and type 2 diabetes mellitus. It is associated with hyperglycemia and, subsequently, ketogenesis because of the combined effects of insulin deficiency and often insulin resistance, excess glucagon, and increased counterregulatory hormones (catecholamines, cortisol, and growth hormone) opposing the actions of insulin.[15]

Table 1 reviews the difference between EKA and DKA. The absence of hyperglycemia can often be misleading, resulting in delayed diagnosis and management.

Laboratory Measurement	Reference Values	EKA	DKA
Blood glucose	80-130 mg/dl	<250 mg/dl	>250 mg/dl
Arterial pH	7.35-7.45	<7.3	<7.3
Serum bicarbonate	22-26 mEq/L	<18 mEq/L	<18 mEq/L
Urine ketones	Absent	Present	Present
Serum ketones	Absent	Present	Present
Anion gap	8-12 mEq/L	>12 mEq/L	>12 mEq/L

EPIDEMIOLOGY

Euglycemic diabetic ketoacidosis is rare, occurring in about 1.1-3.2% of patients with diabetic ketoacidosis.[16] SGLT-2 induced euglycemic ketoacidosis was noted in 71% of patients, with an estimated 7-fold increased risk of developing acidosis in type 2 diabetic patients on these medications.[17]

REVIEW

This review aims to assist clinicians by providing an evidence-based synopsis of euglycemic ketoacidosis, including pathogenesis, etiologies, and management.

Table 2 summarizes the causes of euglycemic ketoacidosis.

Causes of Euglycemic Ketoacidosis
• Anorexia or a state of prolonged starvation
• Chronic alcoholism
• Cocaine use
• Infections
• Low carbohydrate/keto diet
• Pregnancy and lactation
• Glycogen storage diseases
• Gastroparesis
• Insulin use
• Liver disease
• SGLT2 inhibitor use
• Surgery

Pathogenesis and Etiologies

The primary cause of ketoacidosis is a variable degree of hormone imbalance between insulin and glucagon (Figure 1). In response to fasting, low insulin levels due to hypoglycemia and high glucagon levels activate hormone-sensitive lipase, releasing long-chain fatty acids and breaking down triglycerides into glycerol in the peripheral fat stores.[18] These fatty acids are then delivered to the hepatocyte mitochondria, where they undergo beta-oxidation, resulting in acetyl-CoA, which in large quantities leads to the generation of ketone bodies (β-hydroxybutyrate and acetoacetate).

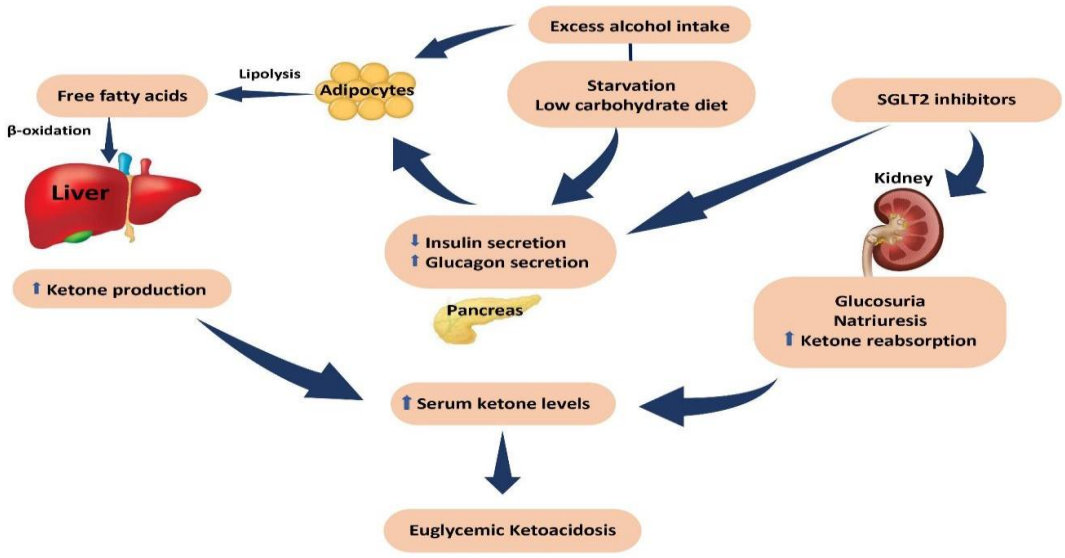

Figure 1: Pathogenesis of euglycemic ketoacidosis

Below are some of the most common etiologies and their pathogenesis.

Starvation and low caloric intake

Fasting ketosis is a milder form of metabolic acidosis often seen in a fasting state of over 12 hours with no reported adverse outcomes. However, with prolonged fasting of over 20–30 hours, serum ketone concentration reaches a peak of 8–10 mmol/L, thereby matching the amount of hepatic ketogenesis to the amount of ketone usage in the brain, muscle, kidney, and peripheral tissues, along with the loss of ketones in the urine. This results in a drop in serum bicarbonate concentration of 7-8 mEq/L and a subsequent rise in the anion gap. Fasting ketoacidosis is observed in prolonged starvation, pregnancy, postpartum states, lactating women, and while on low-carbohydrate diets.[19-22]

Pregnancy and lactation

Pregnancy leads to increased insulin resistance and accelerated lipolysis. Placentally produced hormones like glucagon, cortisol, and human placental lactogen stimulate ketogenesis as free fatty acid concentrations rise.[21] During the third trimester, “accelerated starvation” may emerge to give the fetus enough glucose and amino acids

for its growth and development. Higher levels of free fatty acids and ketone bodies have been noted in pregnant women fasting for as little as 12 hours compared to non-pregnant women. Impaired neural development, fetal distress, and, in some cases, fetal loss have been associated with metabolic acidosis during gestation, making it a severe concern needing immediate workup and treatment.[23-25]

In lactating mothers, a low-carbohydrate diet or prolonged fasting has been associated with EKA.[26] Adhering to ketogenic diets during lactation makes women more susceptible to this condition.[27]

Alcohol intake

Alcoholic ketoacidosis is seen in patients with chronic alcoholism who are often malnourished and have a tendency to binge drink. Hepatic oxidation of ethanol leads to acetaldehyde and acetic acid.[28] This acetic acid is then converted to acetyl-CoA, leading to ketogenesis, lipogenesis, or oxidization in the Krebs cycle. It must be noted that acetic acid and acetyl-CoA inhibit peripheral lipolysis, limiting the delivery of fatty acids to the liver and thus causing only a mild degree of ketoacidosis. Severe ketoacidosis is seen once the alcohol ingestion is halted, leading to a low insulin/glucagon ratio and significant hepatic fatty acid oxidation. Diabetic ketoacidosis (DKA) is seen in patients with type 1 and type 2 diabetes mellitus. Hyperglycemia and, subsequently, ketogenesis is seen because of the combined effects of insulin deficiency and often insulin resistance, excess glucagon, and increased counterregulatory hormones (catecholamines, cortisol, and growth hormone) opposing the actions of insulin.[15]

SGLT2 inhibitors

Euglycemic ketoacidosis (EKA) is frequently encountered in clinical practice due to the widespread adoption of SGLT2 inhibitors for diabetic and non-diabetic indications. These drugs, introduced in 2013, reduce plasma glucose by blocking its proximal tubular reabsorption, thereby increasing urinary glucose excretion.[29] Reduced plasma glucose concentration leads to low circulating insulin levels and an increase in glucagon levels, disrupting the insulin/glucagon ratio and resulting in mild, clinically insignificant ketonemia in patients on this drug. However, the additional generation of ketones during acute illnesses, low caloric intake, or alcohol use through the mechanisms described earlier can lead to overt ketoacidosis. This, along with increased urinary excretion of glucose, can result in relatively normal glucose levels despite ketoacidosis.[30-32]

Continuous Renal Replacement Therapy (CRRT)

In critically ill diabetic patients undergoing CRRT, glucose-free CRRT solutions have been associated with the development of EKA. The clue to the diagnosis is often the persistence of high anion-gap metabolic acidosis despite correcting lactic acidosis, one of the most typical findings in most critically ill patients. The detection of serum β-hydroxybutyrate can confirm the diagnosis. Reduced caloric intake and increased metabolic demands from the stress of the illness can reduce endogenous insulin and increase gluconeogenesis, leading to increased ketogenesis. This can be mitigated by the use of glucose-containing CRRT solutions and providing adequate calorie intake to the patients, all while maintaining adequate glucose control with insulin therapy.[33–36]

Other etiologies

Ketogenic Diet

The keto diet comprises high fats, moderate proteins, and low carbohydrates. It is popular among patients with obesity, drug-resistant epilepsy, and Alzheimer's. Minimal carbohydrate intake can lead to metabolic derangements, including severe metabolic acidosis, electrolyte abnormalities predominantly from poor nutrition, and elevated serum ketone levels, as seen in patients with starvation ketosis.[37,38]

Bariatric Surgery/Gastroparesis

In patients undergoing bariatric surgery, poor oral intake in the perioperative period and postoperative stage can lead to starvation ketoacidosis, resulting in EKA. The use of SGLT-2 inhibitors in these patients, who often have diabetes as a significant comorbidity, has been well documented as an etiology for developing EKA.[39]

Drugs

Cocaine has been implicated in the development of EKA in patients with insulin-dependent diabetes mellitus. The stimulant effects of cocaine on the adrenal gland and its suppressant effects on the brain's feeding centers, which result in starvation, are the potential mechanisms.[6]

Enasidenib, which is used to treat leukemia, is a selectively mutated isocitrate dehydrogenase (IDH) inhibitor that has been linked to EKA.[36] IDH is a significant enzyme in Kreb's cycle, converting isocitrate to alpha-ketoglutarate. Inhibition of IDH leads to citrate accumulation in the mitochondrial cells, excess of which reaches the ketone pathway, thus leading to excess serum acetone and β-hydroxybutyrate levels.

Clinical Manifestations

Similarly, to patients with DKA, the most common symptoms experienced by patients include nausea, vomiting, abdominal pain, fatigue, and malaise. Kussmaul breathing (labored deep breathing) is common; patients' breaths often have a fruity odor due to an excess of circulating acetone. Signs of volume depletion, such as tachycardia, hypotension, and delayed capillary refill, are commonly noted.[40]

Evaluation and Management

There are four basic therapeutic strategies for the management of EKA. Lowering blood glucose, addressing the fluid deficit, closely monitoring potassium levels, and correcting acidosis.

Rapidly diagnosing the presence of EKA is paramount to guiding further management. The mainstay of treatment remains immediate resuscitation with crystalloid fluids, followed by the correction of electrolytes and ketoacidosis. Balanced fluids such as Ringer's lactate and Plasmalyte result in rapid resolution of the ketoacidosis compared to normal saline[41] and reduce the concern for developing hyperchloremic non-anion gap metabolic acidosis.[40,42,43] In all patients with EKA, sepsis must be ruled out as a possible underlying etiology of the metabolic findings and, if identified, must be appropriately treated. To control ketosis in patients with EKA,

insulin infusion must be initiated as soon as possible at 0.05–0.1 units/kg/h, even if the patients are not on insulin at home. In addition, to prevent hypoglycemia, an IV infusion of dextrose must be started concurrently.[40,44,45]

In nondiabetic patients, such as those presenting with EKA due to severe starvation or alcohol abuse, apart from fluid resuscitation, dextrose infusion is pivotal to inhibiting ketogenesis and increasing insulin secretion. Dextrose will further replenish the depleted glycogen stores often seen in these patients, reducing counterregulatory hormones that promote ketogenesis.[46] Thiamine supplementation is essential to prevent the development of Wernicke's encephalopathy in patients with alcohol abuse.[47] Benzodiazepines may be used to curb the risk of alcohol withdrawal leading to seizures.

Electrolyte management, particularly potassium, should be performed cautiously. Repleting potassium before initiating insulin infusion is essential for patients with hypokalemia to prevent a further iatrogenic decline in serum potassium.[40,48,49] Frequent evaluations, every 4-6 hours, of serum electrolytes and glucose are needed to guide treatment for optimal recovery, which is seen when the anion gap, serum bicarbonate levels, and pH are all back to normal and the patient can eat normally.[37]

In patients receiving SGLT2 inhibitors, the drug must be discontinued during the management of EKA. However, unless the patient is found to have underlying risk factors for EKA, such as liver disease or chronic alcoholism, SGLT2 inhibitors may be resumed once the patient's clinical condition improves when accompanied by adequate oral intake.[8] This is especially crucial for patients with chronic heart failure or kidney disease, which are compelling indications for this drug class.

CONCLUSIONS

Euglycemic ketoacidosis is a recognized complication in both diabetic and non-diabetic patients. It is defined by the presence of anion gap metabolic acidosis (pH <7.3, sodium bicarbonate <18 mEq/L), glucose levels <250 mg/dL, and ketosis. Aside from diabetics, it is also seen in patients with chronic liver disease, chronic alcohol use, and pregnancy. SGLT2 inhibitors have recently been associated with this condition. Early recognition and management are essential to avoiding potential metabolic and hemodynamic complications. The mainstay of treatment includes fluids, insulin, dextrose, and management of the underlying triggers promoting ketogenesis.

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Clinical Trial Registration: This is not an interventional study. We only reported the patient's findings from our database as a case report.

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