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Severe Euglycemic Ketoacidosis Induced by Ketogenic Diet

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Abstract Ketogenic diets are diets based on restricted carbohydrate intake in favor of foods high in protein, fats, and fiber. As a result, blood sugar levels tend to drop. This process ultimately leads to the production of ketones which are then used by the body for fuel resulting in ketonemia. This case report focuses on a non-obese male with no past medical history who presented with several days of nausea and vomiting after recently starting himself on a ketogenic diet. Upon admission, the patient was found to have an increased anion gap metabolic acidosis with a gap of 37. Secondary causes of elevated anion gap metabolic acidosis were ruled out. The patient was appropriately fluid resuscitated with a dextrose containing fluid with improvement in symptoms and closure of the gap. Underlying hepatic steatosis may contribute to the development of euglycemic ketoacidosis in those on a low carbohydrate diet. A minimum amount of carbohydrates (approximately 100 grams) may prevent ketosis in these patients. It is important for providers to understand and recognize that severe euglycemic ketoacidosis can be a complication of a ketogenic diet.

Keywords: *Euglycemic ketoacidosis, ketogenic diet, increased anion gap metabolic acidosis. Ketonemia*

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1. Introduction

Ketogenic diets are based on restricted carbohydrate intake in favor of foods high in protein, fats, and fiber. As a result, blood sugar levels tend to drop stimulating the pancreas to release glucagon leading to the conversion of glycogen to glucose. After glycogen stores run out, the liver begins to break down fats and proteins into glucose and ketones via gluconeogenesis and ketosis. Ultimately, this is the mechanism through which we see starvation ketonemia. There are a few case reports in literature about ketogenic diet causing life threatening ketoacidosis. [1,2,3,4] However, the patient population in these case reports were either females who were lactating or patients who were hyperglycemic at the time of presentation. Currently one similar case exists in a female, of similar age, with no past medical history who presented with diet induced ketoacidosis, although with hyperglycemia. [5] Here we report a case of a 50-year-old male with no past medical history who presented with an euglycemic elevated anion gap metabolic acidosis in the setting of a low carbohydrate diet.

2. Case Presentation

A 50-year-old male with no past medical history-on no medications, a remote past history of alcohol abuse (no recent use in 13 years) and normal BMI (24) presented to the emergency department with 4-day history of nausea and vomiting. On presentation, he was afebrile and tachycardic to 110, with a blood pressure of 105/45 and breathing 18 breaths per minute, saturating 100% on room air. On physical exam, the patient had some mild central obesity and tenderness to palpation over right lower abdomen. Shown in Table 1 are his pertinent admission labs. His VBG was notable for a pH of 7.2 and PCo2 of 19. A negative urine toxin screen, non-detectable methyl alcohol and ethylene glycol levels and the osmolar gap helped to rule out other causes of the elevated anion gap.

CT abdomen and pelvis with contrast showed a distended gallbladder believed to be secondary to fasting as well as hepatic steatosis. On subsequent history, it was revealed that the patient had started a ketogenic diet one week prior to onset of symptoms. His diet consisted of a standard ketogenic diet which consisted of approximately 80% protein, and 20% fats, with no carbohydrate intake. The patient was monitored closely in an ICU stepdown unit. He was appropriately fluid resuscitated with improvement in his symptoms and resolution of lab abnormalities.

3. Discussion

This case was unique as this patient was a presumably healthy male with no significant medical history.

Additionally, this patient presented with euglycemic ketoacidosis whereas in Slade and Ashurst's case the patient was hyperglycemic to 163 on presentation to the emergency department. One possible hypothesis is that the patient was more easily predisposed to developing a severe ketoacidosis given his underlying hepatic steatosis which may have resulted in decreased glycogen stores, however, no clear link has been established to date. It may be pathophysiologically similar to pregnancy or lactation where the body has lower glycogen stores, increased insulin resistance, and increased lipolysis leading to a state of ketosis. [6]

Table 1. Laboratory data

Variable	Reference range	On presentation to the emergency department
Sodium (mEq/L)	137-145	131
Potassium (mEq/L)	3.5-5.1	5.4
Chloride (mEq/L)	100-108	86
Carbon dioxide (mEq/L)	22-30	8
Anion Gap	1-12	37
Glucose (mg/dL)	74-106	86
Urea (mg/dL)	6-20	47
Creatinine (mg/dL)	0.52-1.04	1.86
Calcium (mg/dL)	8.4-10.2	8.8
Phosphorous (mg/dL)	2.5-4.5	6
Magnesium (mg/dL)	1.6-2.3	2.4
Serum osmolality	275-295	312
Serum Beta Hydorxy-butyrate	0.4	12
Albumin (g/dL)	3.5-5.0	4.8
Alcohol (mg/dL)	0-10	<10
Acetaminophen (mcg/mL)	10-30	<5
Salicylate (mg/dL)	0-2	<0.3
Lactic acid (mmol/L)	0.7-2.0	0.6
Ethylene glycol	Not detected	Not detected
Methyl alcohol	Not detected	Not detected

4. Conclusion

Severe ketoacidosis is a life-threatening condition which can cause cardiac arrhythmias and even death. The etiology of ketoacidosis can vary and proper recognition of underlying cause is important as treatment modalities are different. It is important to educate patients on ketogenic diets and the complications associated with it. As little as 100 grams of carbohydrates might prevent ketosis and may be clinically indicated in some medical conditions such as epilepsy, [7] and obesity. It is important to recognize in a patient's history that this may be their only contributing factor to the development of anion gap metabolic acidosis.

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