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Case Report

Diet Induced Ketoacidosis in Euglycemic Patient

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ABSTRACT

Anion gap metabolic acidosis is a frequently encountered clinical issue in emergency medicine, often signalling a wide range of underlying pathologies. The diagnostic process requires careful consideration of various potential causes, which may span toxic ingestions, renal insufficiencies, endocrine disturbances, infectious diseases, and cardiac-related conditions. In high-income or developed healthcare settings, one relatively uncommon but clinically significant aetiology of metabolic acidosis is ketosis. While more prevalent in scenarios involving uncontrolled diabetes or prolonged fasting, ketosis is less often seen as a primary presentation in emergency departments in these regions. Its atypical nature in such contexts can delay diagnosis and appropriate management. Recognizing ketosis as a potential contributor to anion gap acidosis remains essential, particularly when more common causes have been excluded. Prompt identification and targeted treatment based on the underlying cause of the ketosis are crucial to improving patient outcomes. In this case report, we discuss an interesting case of diet induced ketoacidosis, the immediate emergency diagnosis and prompt treatment in the emergency department and final outcome.

KEYWORDS

• Ketoacidosis • Ketogenic diet • Euglycemic ketoacidosis • Dieting

INTRODUCTION

Anion gap metabolic acidosis is a frequently encountered and potentially life-threatening condition in the emergency department (ED). This acid-base disturbance arises when there is an accumulation of acids in the body that are not routinely measured, leading to an increased anion gap in laboratory

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investigations.[1] Its presence often signals serious underlying pathology, and rapid identification of the root cause is critical for appropriate clinical management. Given the wide spectrum of potential aetiologies, clinicians frequently use diagnostic mnemonics such as **"CAT MUD PILES"** to systematically recall common causes. These include conditions such as carbon monoxide / cyanide poisoning / congenital heart failure, aminoglycosides / alcoholic ketoacidosis, toluene ingestion / theophylline, methanol or metformin toxicity, uraemia, diabetic ketoacidosis (DKA), paraldehyde, iron or isoniazid toxicity, lactic acidosis, ethylene glycol poisoning, and salicylate overdose. While this framework provides a practical approach for evaluation, it does not encompass all possible origins of anion gap metabolic acidosis.

One such lesser-known but important cause is **starvation ketosis**, a condition resulting from significant carbohydrate deprivation. In normal metabolic states, ketone body production is suppressed by adequate carbohydrate intake.[2] Generally, consuming around 100 grams of carbohydrates per day is sufficient to prevent ketosis. Remarkably, even a minimal daily intake of approximately 7.5 grams of glucose can significantly curb the production of ketone bodies. However, when carbohydrate intake falls below these thresholds especially over prolonged periods the body begins to metabolize fat as its primary energy source, leading to the accumulation of ketones and subsequent metabolic acidosis.[3]

Despite its rarity in developed countries, starvation ketosis is becoming more clinically relevant due to the rising popularity of low-carbohydrate diets such as the ketogenic diet. These dietary patterns promote high fat and protein consumption while minimizing carbohydrate intake, often to levels low enough to induce ketosis for weight loss purposes. While generally safe when monitored appropriately, such diets can occasionally lead to significant metabolic disturbances, particularly in individuals with underlying health conditions or poor nutritional awareness.

We report a case of a 43-year-old female who presented to the ED with unexplained high anion gap metabolic acidosis. Initial history and diagnostic workup did not align with typical causes as outlined in the CAT MUD PILES mnemonic. Further investigation revealed that the patient had been following a strict zero-carbohydrate diet as part of a personal weight loss regimen. This led to the development of ketoacidosis in the absence of diabetes, alcohol use, or other known triggers.

This case underscores the importance of including **diet-induced ketosis** in the differential diagnosis of high anion gap metabolic acidosis, especially in settings where low-carbohydrate dietary practices are gaining traction. Increased awareness of this condition is essential for timely recognition and management, particularly as more individuals adopt extreme dietary modifications for metabolic health or weight loss. Emergency physicians should remain vigilant for non-traditional causes of metabolic acidosis and consider dietary history as an integral part of patient evaluation.

CASE

A 43-year-old female patient was brought to the emergency department (ED) of our hospital in critical state on oxygen support in an ACLS ambulance. On examination in the ED, patient also complained of generalised progressive weakness with lower abdominal pain. The patient was ill-looking with obvious signs of dehydration. Her initial vitals in the ED: pulse rate – 124/min, blood pressure – 90/40 mmHg, Oxygen saturation – 95% on room air, Random Blood Sugar – 105 mg/dL, GCS – E3V4M6 (13/15). She looked tired. There was no other significant history suggestive of fever, chest pain, alcohol use, any medication intake.

Patient was started on intravenous fluid (*approximately 2 litres*) along with other supportive measures. Her arterial blood gas analysis showed – pH: 7.21, pCO2: 14, pO2: 56, HCO3: 8.6, Lac: 1.2, AG: 22, Osmo – 287. All the possible diagnosis (using mnemonic "CAT MUD-PILES" were ruled out on the basis of history and initial investigations. Further detailed history from the patient and her parents were taken which elaborated that the patient was into severe dietary restriction and rigorously followed it. She was on ketogenic diet and consumed very less carbohydrate in her diet in fear of weight gain. On the basis of this, a diagnosis of euglycemic ketoacidosis was established. Patient was admitted and

managed with intravenous fluids and other supportive care. Rest blood parameters were within normal limits.

Patient was discharged after 3 days of in-hospitalisation. Psychiatric consultation was done, dietary consultation was done for the patient prior to discharge.

The follow-ups with the patient were satisfactory.

DISCUSSION

The ketogenic diet, characterized by high fat, moderate protein, and very low carbohydrate intake, has been used therapeutically, particularly in managing refractory epilepsy.[4] However, in modern clinical practice, emergency physicians are more likely to encounter its use as part of weight loss strategies. When carbohydrate consumption is drastically reduced over several days, glycogen stores are depleted, prompting the body to switch to fat metabolism. This results in the breakdown of fatty acids and the production of ketone bodies beta-hydroxybutyrate, acetoacetate, and acetone by the liver through ketogenesis. While these ketones serve as alternative energy sources, their excessive accumulation can result in ketoacidosis.

Certain populations, such as children and pregnant or lactating women, are more vulnerable to diet-induced ketosis. These groups tend to have lower glycogen reserves and may exhibit increased lipolysis or insulin resistance, increasing their risk for metabolic acidosis. Typically, starvation ketosis does not lead to profound acidosis, and serum bicarbonate levels remain above 18 mEq/L. However, in rare situations as seen in our patient a significantly lower bicarbonate level (8 mEq/L) may occur, indicating a more severe metabolic disturbance likely exacerbated by prolonged carbohydrate restriction.[4]

Although ketogenic diets are generally regarded as safe for weight loss, there are reported exceptions. A similar case related to the ***Atkins diet*** in 2004 highlighted comparable findings, and several other case reports have since linked low-carbohydrate, high-protein diets to severe anion gap metabolic acidosis [5]. Interestingly, these cases predominantly involved women and had favourable outcomes following treatment.

Management focuses on reversing ketosis, primarily by reintroducing carbohydrates either orally or intravenously via dextrose containing fluids. Individuals with underlying risk factors such as diabetes, chronic alcoholism, pregnancy, or lactation should be advised against extreme carbohydrate restriction. Ensuring a daily intake of at least 100 grams of carbohydrates is essential to prevent this potentially serious complication.[6]

CONCLUSION

Anion gap metabolic acidosis is a critical condition that necessitates prompt evaluation and intervention in the emergency setting. While common causes are well-documented and often easily recognized through standard mnemonics like "CAT MUD PILES," less typical etiologies, such as diet-induced ketoacidosis, should not be overlooked especially in the context of evolving dietary trends. The increasing popularity of low-carbohydrate and ketogenic diets for weight loss presents a growing clinical concern, as prolonged carbohydrate restriction can, in rare instances, lead to significant metabolic disturbances.

This case highlights the importance of considering dietary history as a vital component of the clinical assessment, particularly when traditional causes of high anion gap metabolic acidosis have been ruled out. Although ketogenic diets are generally considered safe for the general population when properly balanced, they can pose a risk to certain individuals, especially those with limited glycogen reserves, insulin resistance, or increased metabolic demands, such as in pregnancy or lactation.

The presented case involved a patient who developed severe ketoacidosis due to a strict zero-carbohydrate diet, despite lacking typical risk factors. This underscores that even seemingly healthy individuals may be susceptible to adverse effects from extreme dietary regimens. Early recognition and intervention, including the administration of carbohydrates either orally or via intravenous dextrose, are key to reversing the metabolic derangement and preventing further complications.

Ultimately, patient education regarding the potential risks of extreme diets is essential. Healthcare providers must stay informed

about current dietary trends and be vigilant in identifying their potential consequences. Encouraging balanced nutrition and monitoring at-risk individuals who follow restrictive diets can help prevent avoidable emergency presentations and ensure better patient outcomes.

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