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STARVATION KETOACIDOSIS ON THE ACUTE MEDICAL TAKE: AN EASILY MISSED COMPLICATION OF THE KETO DIET

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ABSTRACT

Starvation ketoacidosis represents one of the three forms of metabolic acidosis caused by the accumulation of ketone bodies within the blood stream. It can be easily missed in patients who present acutely and are found to have an unexplained or profound metabolic acidosis. Here, we present a life-threatening case of severe ketoacidosis in a breast-feeding mother without diabetes who was on a strict ketogenic diet. Although a ketogenic diet has been previously considered to be safe in non-pregnant individuals, its safety in breast-feeding mothers in the post-partum period is less known and may be associated with greater harm.

Health professionals and mothers should be aware of the potential risks associated with a strict ketogenic diet when combined with breast-feeding, especially in the earlier stages of the post-partum period. Prompt investigation, diagnosis and immediate management is vital to avoid life-threatening complications.

We report a case admitted on the acute medical take with starvation ketoacidosis associated with ketogenic diet and adequate calorie consumption who was breast-feeding at the time of admission.

KEYWORDS

Ketoacidosis, keto diet, weight-loss diet

LEARNING POINTS

- Always check ketones in patients with an unexplained metabolic acidosis; there can be overlap between starvation, alcohol-related and lactic acidosis.
- Management of starvation ketoacidosis is often empirical, involving close monitoring of fluid status and electrolytes.
- Clinicians should discuss the risk of ketoacidosis associated with the ketogenic diet in women who plan to breast-feed and lose weight following pregnancy.

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INTRODUCTION

Ketoacidosis is common in the acute medical take, typically a consequence of uncontrolled diabetes. However, there are several causes of high ketones, including diabetic ketoacidosis, starvation ketoacidosis and alcohol-related ketoacidosis. Starvation ketoacidosis can be overlooked when patients present with an unexplained metabolic acidosis and a low/normal blood glucose. It is relatively simple to manage, but there are potential pitfalls in diagnosis and treatment. Management consists of intravenous glucose, with cautious monitoring of acid-base balance and electrolytes[1], which differs from diabetic ketoacidosis given the need for insulin therapy. Serum lactate may also be raised which can mislead the clinician and is relevant if the acidosis is out of proportion to the serum lactate, highlighting the need to check ketones. Here, we present a case of severe ketoacidosis secondary to a keto diet in a woman 12-weeks post-partum who presented to the acute medical take.

CASE DESCRIPTION

A woman in her 40s presented to the emergency department 12-weeks post-partum complaining of fatigue, severe nausea, vomiting and epigastric pain for 12 hours. She had no significant pre-existing medical history and denied use of any prescribed or over-the-counter medications. The patient reported adhering to a strict ketogenic diet post-partum to lose weight since the birth of her 12-week-old baby. This had also been an issue during her first pregnancy, and her previous back pain resolved with weight loss. She had uncomplicated prenatal care and normal vaginal delivery at 40 weeks gestation with uneventful puerperium. Dietary review revealed that she ate eggs, meat, vegetables, cheese and fish. Protein intake was chicken, white fish, nuts, peanut butter and salmon. She avoided all carbohydrates, did not consume alcohol and denied calorie restriction. She admitted to focusing on the 'keto diet' to achieve rapid weight loss post-partum.

On presentation to the emergency department her observations were: blood pressure 114/76 mmHg, heart rate 82 beats/min, respiratory rate 14 breaths/min, oxygen saturation 99% and temperature 36.9°C. An examination revealed dry mucus membranes with epigastric tenderness. Laboratory studies demonstrated sodium 142 mmol/l; potassium 3.8 mmol/l; chloride 111 mmol/l; urea 3.3 mmol/l; creatinine 89 µmol/l, bicarbonate 4 mmol/l; glucose 4.3 mmol/l and anion gap 24 mEq/l. Arterial blood gas showed a metabolic acidosis with pH 6.98, partial-pressure carbon dioxide 2.8 kPa; bicarbonate 4 mmol/l; base excess -26.7 mmol/l, lactate 0.7 mmol/l. Capillary ketones were 5.6 mmol/l on admission. An electrocardiogram revealed a sinus rhythm with a rate of 90 beats/min, QTc interval 428 ms and no significant abnormalities. *Figure 1* demonstrates the trend in capillary glucose, ketone, venous bicarbonate and pH over the initial 24 hours.

She was initially treated with intravenous 10% glucose and isotonic intravenous bicarbonate solution. She was reviewed by the intensive care team as her acidosis did not improve following 4l fluid resuscitation, and then commenced on variable rate intravenous insulin infusion (VRIII) with frequent blood gas and capillary glucose, and ketone monitoring. However, associated with VRIII she had frequent hypoglycaemia and no improvement in ketoacidosis despite a 10% glucose infusion. Hence, VRII was discontinued after 4-6 hours, and intravenous glucose was administered. After eight hours of further fluid resuscitation her blood gas improved with pH 7.25, partial-pressure carbon dioxide 4.4 kPa, bicarbonate 15 mmol/l; base excess −12.7 mmol/l. She was able to tolerate soft foods and fluids orally and was restarted on a carbohydrate diet. She was closely monitored for hypokalaemia, hypophosphatemia and hypocalcaemia suggestive of refeeding syndrome.

Following the initiation of oral carbohydrates and 8l intravenous 10% glucose her acid-base balance normalised, and the anion gap closed to normal limits within 48 hours. She was discharged on day three of her admission with follow-up arranged.

DISCUSSION

The ketogenic diet is a high-fat, adequate-protein, low-carbohydrate diet (<50 g/day), a dietary measure often

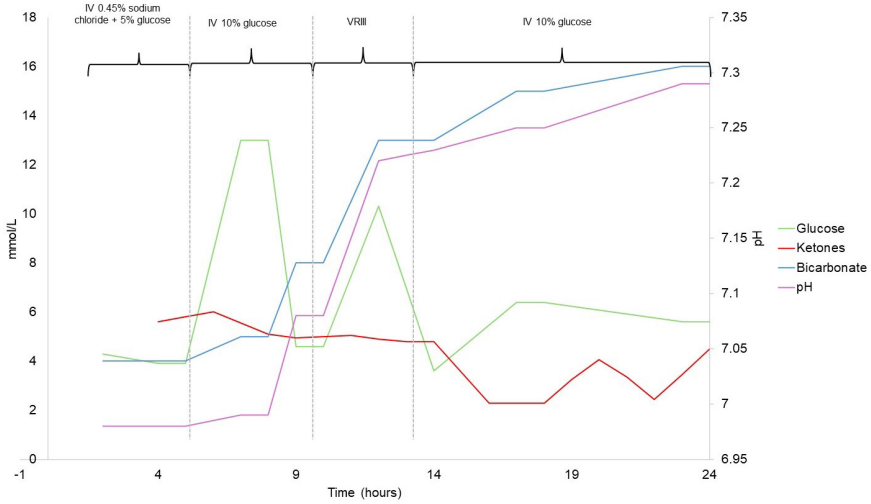

Figure 1. The trend in capillary glucose, capillary ketones, venous bicarbonate and pH over the first 24 hours of admission. Abbreviations: IV, intravenous; VRIII, variable rate intravenous insulin infusion.

associated with greater weight loss compared to others[2,3]. The mechanism culminates in nutritional ketosis induced by low availability of carbohydrate as the body shifts to utilise fats instead of carbohydrate. This process involves conversion of free fatty acids into ketone bodies, resulting in accumulation of ketones in the blood and urine but not typically to a level associated with changes in blood pH[2]. The ketogenic diet has been described since the 1920s and is one of the most studied interventions for weight loss[4].

Previous studies describe healthy breast-feeding women on a low-carbohydrate diet having significantly greater gluconeogenesis with ketosis, and both increased protein and free fatty acid oxidation[5]. The increased energy demands of breast-feeding with increased glucose utilisation may dysregulate the compensatory mechanisms that regulate ketone levels in a ketogenic diet[5]. In this case, the severity of starvation ketoacidosis is likely a product of these mechanisms, exacerbated by breast-feeding. Previous reports of patients admitted with starvation ketoacidosis highlight the importance of physiological stressors such as pancreatitis[6], recreational drug and alcohol use[2], and concurrent infections in pregnancy[7]. While there are reports highlighting the ketogenic diet as a precipitant for starvation ketoacidosis[8,9], these were in the context of type 2 diabetes where by virtue of their insulin resistance, patients are more likely to develop ketones.

Lactation ketoacidosis is also previously described in breast-feeding mothers, though this is typically associated with concurrent infection and a low-carbohydrate diet[10]. Like starvation ketoacidosis, there is an increase in catabolic hormones including catecholamines and glucagon, with lower serum insulin from the lower carbohydrate intake, contributing to increased ketone body production[2,10].

There is a debate around the appropriate use of insulin. Replenishing glucose with intravenous hydration in people without diabetes should facilitate *in vivo* insulin production to resolve ketosis. Exogenous insulin therapy would suppress endogenous ketone production, so theoretically should improve ketoacidosis in this context. However, insulin therapy does not seem to improve outcomes with associated hypoglycaemia in the context of low glycogen stores. In the absence of trial evidence, the consensus is to treat with intravenous glucose solution, and consider intravenous bicarbonate with close electrolyte, glucose and ketone monitoring[10]. In this case, we observed no benefit with intravenous insulin, though it did cause hypoglycaemia.

CONCLUSION

While the ketogenic diet is considered a safe intervention for weight loss, the case highlights the need to stress caution in breast-feeding mothers to avoid such life-threatening complications which could be overlooked on admission.

REFERENCES

1. Gall AJ, Duncan R, Badshah A. Starvation ketoacidosis on the acute medical take. *Clin Med (Lond)* 2020;**20**:298–300.
2. Gupta L, Khandelwal D, Kalra S, Gupta P, Dutta D, Aggarwal S. Ketogenic diet in endocrine disorders: current perspectives. *J Postgrad Med* 2017;**63**:242–251.
3. Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* 2003;**88**:1617–1623.
4. Paoli A, Rubini A, Volek JS, Grimaldi KA. Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets. *Eur J Clin Nutr* 2013;**67**:789–796.
5. Mohammad MA, Sunehag AL, Chacko SK, Pontius AS, Maningat PD, Haymond MW. Mechanisms to conserve glucose in lactating women during a 42-h fast. *Am J Physiol Endocrinol Metab* 2009;**297**:E879–E888.
6. Chan KH, Ramahi A. A rare case of severe starvation-induced ketoacidosis in a patient with recurrent pancreatitis. *Cureus* 2020;**12**:e7368.
7. Nana M, Nelson-Piercy C. Starvation ketosis in a pregnant woman with COVID-19: a case report. *Endocrinol Diabetes Metab Case Rep* 2022;**2022**:22–0222.
8. Blanco JC, Khatri A, Kifayat A, Cho R, Aronow WS. Starvation ketoacidosis due to the ketogenic diet and prolonged fasting – a possibly dangerous diet trend. *Am J Case Rep* 2019;**20**:1728–1731.
9. Malhi MS, Duerson F, Salabei JK, Okonoboh P. Starvation ketoacidosis induced by ketogenic diet and consumption of ketone supplement. *Cureus* 2021;**13**:e15778.
10. Al Alawi AM, Al Flaiti A, Falhammar H. Lactation ketoacidosis: a systematic review of case reports. *Medicina (Kaunas)* 2020;**56**:299.