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CASE REPORT

General Medicine

Severe lactation ketoacidosis presenting as a respiratory complaint

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Abstract

Ketoacidosis, a type of high anion gap metabolic acidosis, results from 1 of 3 etiologies: diabetic ketoacidosis, alcoholic ketoacidosis, or starvation ketoacidosis (SKA). In rare instances, young and otherwise healthy lactating women have been found to develop lactation ketoacidosis, a form of SKA, when the high energy requirements of breastfeeding are not met with adequate carbohydrate intake. We present the case of a 29-year-old woman who presented to our emergency department with respiratory distress and headache and was found to have severe lactation ketoacidosis. The patient was treated with infusions of dextrose and bicarbonate in the emergency department and medical intensive care unit. She was discharged without complication 3 days later, after nutrition and lactation consultation. This case highlights both the importance of maintaining a broad differential diagnosis that includes lactation ketoacidosis and performing a careful interview to identify patient populations at risk for this pathology.

KEYWORDS
ketoacidosis, lactation, starvation

1 | INTRODUCTION

The accumulation of ketone bodies in the bloodstream results in a high anion gap metabolic acidosis that is represented by 3 distinct processes: diabetic ketoacidosis (DKA), alcoholic ketoacidosis (AKA), or starvation ketoacidosis (SKA).[1] Typically, these pathologies are strongly suggested by a clear history of diabetes mellitus, alcohol abuse, or starvation, except in the specific instance of DKA in the newly diagnosed diabetic when high glucose is strongly suggestive of the diagnosis. However, there is a rare and often unconsidered form of ketoacidosis that occurs in the absence of a clear history of one of these risk factors. Lactation ketoacidosis (LKA) is a form of starvation ketoacidosis that can occur in lactating women if the metabolic demands of breastfeeding are not met with adequate carbohydrate consumption, resulting in a relative starvation state in the absence of fasting.[2,4]

LKA was first noted in lactating dairy cows whose energy requirements for milk production and maintenance would increase faster than their energy intake via feeds in the first 6 to 8 weeks of lactation. In the veterinary literature this has been well described as "bovine ketosis."[3] The literature on this condition in humans is limited mainly to case reports, the first of which was published in 1982.[4] A recent systematic review by Al Alawi identified only 18 published cases globally over the last 30 years.[2] We report a case of severe LKA that was identified in a young postpartum patient who presented with common chief complaints.

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2 | NARRATIVE

A 29-year-old Gravid 4, Para 4004 female who was 3 weeks postpartum from a spontaneous vaginal delivery presented to our emergency department (ED) with 1 day of severe headache and shortness of breath. She denied fever, cough, chest pain, nausea or vomiting, and neurologic deficits. Other than being postpartum, she denied any risk factors for or history of venous thromboembolic disease. Past medical history and family history were non-contributory. She took only prenatal vitamins and denied alcohol, illicit substance, or tobacco use. She did report frequent breastfeeding every 2 hours, poor caloric intake secondary to postpartum nausea and early satiety, a low carbohydrate diet, and a weight loss of 40 pounds since delivery.

Her initial vital signs were heart rate of 115 beats/min, blood pressure of 103/61 mmHg, respiratory of rate 29 breaths/min, oxygen saturation of 100% on room air, and temperature of 37.0°C. She weighed 61.4 kg with a body mass index (BMI) of 28.3. She appeared ill and in mild respiratory distress. Cardiopulmonary exam was notable only for aforementioned tachycardia and tachypnea. The rest of her exam, including a detailed neurologic exam, was unremarkable.

Complete metabolic panel was as follows: sodium 138 mmol/L, chloride 104 mmol/L, bicarbonate 4 mmol/L, glucose 68 mg/dL, BUN 9, creatinine 0.67. She had an anion gap of 30. Lactic acid was 1.1 mmol/L. A venous blood gas revealed a pH of 7.0, pCO2 of 19 consistent with metabolic acidosis with appropriate respiratory compensation. In evaluating for other etiologies of an anion-gap metabolic acidosis, acetaminophen, salicylate, ethanol, ethylene glycol, methanol, iron, and carboxyhemoglobin levels were ordered and were all negative. Beta-hydroxybutyrate was notably elevated at 122.5 mg/dL (upper limit of normal 2.8 mg/dL). Chest radiograph did not show any infiltrates. Computed tomography (CT), including CT venogram of the head and CT angiogram of the chest, did not reveal any thrombosis or thromboembolism, though there was a right intraorbital mass that was found to be an incidental orbital cavernous venous malformation on subsequent magnetic resonance imaging.

After initial results were obtained, the patient was administered 50 mL of sodium bicarbonate and 50 mL of 50% dextrose for treatment of severe acidosis and hypoglycemia, respectively. She was volume resuscitated with 1L bolus of normal saline. Once admitted to the medical intensive care unit (ICU), she was maintained on an infusion of 5% dextrose in sodium chloride (0.45 to 0.9% variably) at a rate of 125 to 200 mL/h for 43 hours. Potassium, phosphorus, and thiamine were repleted. Her anion gap closed after 16 hours, and her serum bicarbonate normalized to 21 mmol/L after 50 hours. She was evaluated by nutrition and lactation specialists and provided education. Their recommendations included continue exclusive breastfeeding for the first 6 months postpartum but increase calorie intake by 400 kcals per day, consume 3.8 total liters of fluids per day, and use nutritional supplements at home to supplement calorie intake as needed. Her symptoms resolved, and she was discharged 3 days after admission without adverse events.

3 | DISCUSSION

We have described the case of a young woman in the postpartum period who presented to the ED in respiratory distress found to be secondary to severe lactation ketoacidosis.

The differential diagnosis for high anion gap metabolic acidosis includes ketoacidosis (DKA, AKA, SKA), renal failure (uremia), lactic acidosis, and various ingestions (methanol, ethylene glycol, salicylates, etc). When dealing with ketoacidosis specifically, history is usually sufficient to differentiate between insulin deficiency, alcohol abuse, or starvation as the etiology. In rare instances, LKA as a form of SKA can occur in lactating women if the energy requirements for breastfeeding are not met by adequate carbohydrate intake.

Because the condition is so infrequent, a high index of suspicion must be maintained and a detailed history obtained to make the diagnosis. The presentation of LKA seems to be non-specific with the most commonly associated chief complaints being abdominal pain, nausea and vomiting, headache, weakness, and dyspnea, all of which are equivocal and can represent any number of pathologies.[3] Although the demographics were as expected for young females with infants, it was interesting to note that most patients, including ours, had a normal BMI. Our patient denied following any specific dieting regimen such as the popular ketogenic diet but did admit to a low carbohydrate diet and lower calorie intake in general because of postpartum nausea and early satiety. This had resulted in a significant amount of weight loss over a short period of time.

Treatment of LKA consists of IV dextrose-containing fluids and/or sodium bicarbonate depending on the severity of the acidosis.[3] Although serum bicarbonate levels < 8 mgEq/L can predispose patients to cardiac disturbance and dysrhythmias, this was not seen with our patient nor in the cases detailed in the systematic review.[1] Nonetheless, the possibility should be considered and patients with severe LKA should be monitored closely on telemetry. Our patient, and the patients described in the review, did well with these interventions. Her acidosis resolved without residual complication. Just as important as the correction of the immediate pathology is the prevention of recurrence, which includes nutritional adjustments and education. Patients are encouraged to continue breastfeeding in order to maintain the health benefits for the infant; however, this should be done with a proportional increase in daily caloric and fluid intake with special consideration to a balanced diet that includes adequate carbohydrates.

CONFLICTS OF INTEREST

There are no conflicts of interest to report for any of the authors.

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REFERENCES

1. Kelen GD, Cline DM. Acid-base disorders. *Tintinallis Emergency Medicine: A Comprehensive Study Guide*. 9th ed.. New York: McGraw Hill Education; 2020:73-78.

2. Al Alawi AM, Al Flaiti A, Falhammar H. Lactation ketoacidosis: a systematic review of case reports. *Medicina (Kaunas)*. 2020;56(6):299. Published 2020 Jun 17.
3. Gross J, van Dorland HA, Bruckmaier RM, Schwarz FJ. Performance and metabolic profile of dairy cows during a lactational and deliberately induced negative energy balance by feed restriction with subsequent realimentation. *J Dairy Sci*. 2011a;94:1820-1830.
4. Chernow B, Finton C, Rainey TG, O'Brian JT. Bovine ketosis" in a nondiabetic postpartum woman. *Diabetes Care*. 1982;5:47-49.

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