

Case Report

A case of bovine ketoacidosis in a lactating woman

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Abstract

A 36 year-old 5 weeks postpartum lactating woman presented to the emergency room with severe nausea and vomiting for 48 hours. The patient was found to be in non-diabetic ketoacidosis with a serum pH 6.9 and a HCO_3^- of $<5\text{mEq/L}$. This condition rapidly improved with the administration of intravenous dextrose and bicarbonate and with the cessation of breast feeding. The course and pathophysiology of the rarely described phenomenon of bovine ketosis in a human is discussed here.

Keywords: acidosis; bovine ketoacidosis; lactation acidosis

Case report

A 36-year-old 5-week postpartum woman presented to the emergency room with complaints of nausea and vomiting for 48 h. She also reported a 2-day history of general malaise and worsening dyspnoea. She denied chest pain, palpitations, diarrhoea or headaches. She reported eating; however, she limited herself to several small high-protein carbohydrate-free meals in an attempt to quickly return to her prepartum weight. Since her delivery, she had achieved an intentional weight loss of 30 lbs. Initial vital signs were: temperature 37°C , blood pressure 140/63 mmHg, heart rate 84, respiratory rate 20 and a weight of 59.8 kg. She appeared unkempt and lethargic. Head and neck examinations were benign. Chest auscultation was clear with no adventitious sounds; heart examination was unremarkable. The abdomen was benign with a well-healed surgical C-section scar; there was no costovertebral angle tenderness. The extremities were without tenderness or oedema. She had no neurologic defects other than her blunted mentation.

Her past medical history was significant for a cesarean section, complicated by bladder damage, now resolved. Her only medication was percocet to manage post-surgical visceral pain. She denied alcohol or illicit substance use.

Initial laboratory data include sodium of 144 mEq/L, potassium 4.8 mEq/L, chloride 104 mEq/L and bicarbonate $<5\text{mEq/L}$ that is the lowest available determination possible at our institution. Her BUN was 3 mg/dL, creatinine was 1.1 mg/dL and glucose was 133 mg/dL. Serum lactate

was 2.1 mmol/L, phosphate 1.0 mg/dL and magnesium 1.6 mg/dL. Arterial blood gas drawn on room air showed a pH of 6.9, PCO_2 of 19 mmHg, PaO_2 of 137 mmHg and SaO_2 of 100%. White blood cell count was $17\,600/\text{mm}^3$ with 88.2% segmented neutrophils, Hg 13.7 g/dL, haematocrit 43.1% and platelets of $340\,000/\text{mm}^3$. Urinalysis had a pH of 5.5 with $<80\text{mg/dL}$ ketones. The serum osmolar gap was 7. A comprehensive drug screen was negative including salicylates and acetaminophen. Chest radiograph was normal. Blood and urine cultures were negative.

The patient was volume-depleted with a metabolic acidosis and a serum anion gap of 35. Fluid resuscitation with three ampules of sodium bicarbonate per litre of D5W at 100 cc/h was initiated for 24 h. Improvement in laboratory values occurred within 12 h with serum bicarbonate improving to 12 mEq/L. The serum bicarbonate improved to 20 mEq/L with normalization of the anion gap at 24 h. Symptomatic improvement occurred likewise. The patient was started on an 1800 kcal diet during her hospitalization, supplemented with a regular diet of home food provided by the husband. Breast feeding was stopped on advice from a lactation consultant and the acidosis did not reoccur. The patient's baby was reported to be in the 90th percentile for weight and height.

Discussion

The differential for increased anion gap metabolic acidosis is broad and includes poorly controlled diabetes, ketoacidosis due to alcohol or starvation, lactic acidosis, some medications or ingestions of methanol or ethylene glycol.

Uncontrolled diabetes mellitus is the most commonly seen cause of increased anion gap metabolic acidosis. Here, insulin deficiency increases lipolysis, free fatty acid delivery to the liver, while glucagon promotes conversion of free fatty acids into ketoacids. Starvation ketosis is less common and results from prolonged absolute caloric deprivation. Acute starvation does not typically lead to acidosis because of the presence of insulin. Stress may also worsen starvation ketoacidosis [1].

Acidosis seen with alcohol ingestion can potentiate the severity of other disorders that are associated with the overproduction of lactate. The combination of alcohol ingestion

and poor dietary intake also leads to an acidosis secondary to ketosis. Alcoholics tend to have a decreased oral intake that reduces insulin secretion and increases glucagon production. In addition, alcohol-induced inhibition of gluconeogenesis and stimulation of lipolysis all contribute to acidosis through increased ketoacid production.

Lactic acidosis is a common cause of metabolic acidosis in hospitalized patients. Decreased oxygen delivery leading to increased anaerobic metabolism is responsible. Many cases in hospitalized patients are due to tissue hypoperfusion that occurs with shock, cardiac failure or sepsis.

Medications can also contribute to or exacerbate acidosis. Metformin for oral therapy of diabetes-causing lactic acidosis has been described. It reduces pyruvate dehydrogenase activity and mitochondrial transport of reducing agents, and thus enhances anaerobic metabolism. Inhibition of pyruvate dehydrogenase results in increased metabolism of pyruvate to lactate and increases the net lactic acid production. Several HIV antiretrovirals can also precipitate lactic acidosis by direct mitochondrial toxicity. It is typically described with all nucleoside reverse transcriptase inhibitors, but patients taking stavudine or didanosine seem to be at a greater risk.

Methanol and ethylene glycol consumptions are well-known causes of anion gap metabolic acidosis. They are found in automotive antifreeze, windshield wiper fluid, solvents and household cleaners.

The syndrome of 'bovine ketosis' is well described in the veterinary literature of lactating cattle. In this disorder, the metabolic and glucose demands of milk production and secretion exceed the amount of ingested carbohydrate and glycogen stores [2] in these chronically lactating animals.

Bovine ketosis is rare in humans because it is uncommon for the mother to be unable to meet her own metabolic demands through diet. The energy cost of breast feeding from birth to 6 months is an additional 500 kcal/day; then for 7 months to 1 year, the additional energy required is 400 kcal [3]. These nutritional requirements needed to support lactation exceed the energy requirements during the non-pregnant state and are even beyond those needed during pregnancy. It is therefore important to emphasize to patients

that active weight loss should not be undertaken during breast feeding.

There have been two previous case reports: Heffner and Johnson reported a 35-year-old lactating woman with ketonuria, nondiabetic ketoacidosis and a pH of 7.24 [4]. Altus and Hickman described the case of a 30-year-old woman postpartum who presented with nausea, vomiting and dehydration [5]. She had been following a high-protein carbohydrate-restricted diet. Her arterial pH was 7.07, serum bicarbonate <5 mEq/L with positive urinary and serum ketones. Both patients improved with intravenous fluid, feeding and cessation of breast feeding.

Similar to the prior reports, our patient was under the stress of lactation but the degree of her acidosis, a serum pH of 6.9, has not been previously reported. We feel that in her case the relative starvation precipitated the ketoacidosis [6] and the increased metabolic demands of lactation produced a life-threatening acidemia. This is a rare but clinically significant case of lactation 'bovine' ketoacidosis. Its recognition and appropriate management are important for good outcomes. Further, lactating women should be advised against excessive caloric restriction.

Conflict of interest statement. None declared.

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