Severe High Anion Gap Metabolic Acidosis in Pregnancy

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Ketoacidosis can occur most often as a result of uncontrolled diabetes mellitus. However, it can be seen with fasting and alcohol consumption, as well. Ketoacidosis in association with fasting has less severity, and ketoacid levels do not exceed 10 mEq/L. In the literature, there are a few reports about severe high anion gap acidosis that were associated with fasting. We report a case of pregnancy associated with high anion gap acidosis as a result of fasting.

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INTRODUCTION

In approach to the patients who present with high anion gap metabolic acidosis, we evaluate the main causes of it, which consist of lactic acidosis, ketoacidosis, ingested toxins, and kidney failure.[1] In this article, a pregnant patient is presented who had high anion gap acidosis without any history of toxin ingestion, diabetes mellitus, or kidney failure.

CASE REPORT

A 27-year-old pregnant woman presented at week 30 of gestation. Her previous pregnancy was normal. She was admitted to hospital with a history of severe vomiting for 3 days, with more than 30 times vomiting in 1 day. She had no history of stomachache or diarrhea. Her vomiting was not related to eating.

Her past medical history was associated with nausea and vomiting from the beginning of this pregnancy, leading to multiple visits to clinic and serum therapy, which would improve the symptoms. However, the last time, she appeared so ill. Her arterial blood gas was checked that showed severe acidosis, which led to admission to the hospital.

She did not have any psychological problems and no history of suicide or drug use, and her rapid response to serum therapy decreased the possibility of intoxication. On examination, blood pressure was 100/70 mm Hg, pulse rate was 94 beats per minute, and body temperature was 36.5°C. Blood pressure assessed on sitting position ruled out orthostatic hypotension. Respiratory rate was 24 per minutes with a Kussmaul pattern. Lung auscultation revealed no abnormal signs. Urine output was satisfactory. Serum laboratory data on admission are shown in Table 1. Urine dipstick showed acetone (3+), no

Table 1. Laboratory Data of Patient on Admission

Parameter	Value	Reference Range
Leukocyte count, × 10^3/L	10.7	4 to 11
Hematocrit, %	32.2	38 to 50
Platelet count, × 10^3/L	267	150 to 450
Erythrocyte sedimentation rate, mm/h	19	…
Fasting blood glucose, mg/dL	111	70 to 110
Serum creatinine, mg/dL	0.6	≤ 1.5
Serum chloride, mg/dL	102	100 to 108
Serum lactate, mg/dL	15.0	4.5 to 19.8
Serum sodium, mEq/L	135	135 to 145
Serum potassium, mEq/L	5.0	3.8 to 5.0
Serum calcium, mg/dL	9.1	8.5 to 10.5
Aspartate aminotransferase, U/L	21	5 to 40
Alanine aminotransferase, U/L	17	5 to 40
Alkaline phosphatase, U/L	214	150 to 450
Lactate dehydrogenase, U/L	316	340 to 500
Total bilirubin, mg/dL	1	< 1.5
Amylase, U/L	115	35 to 115
Lipase, U/L	25	0 to 160
Albumin, g/dL	3.8	3.5 to 5.2
Thyroid stimulation hormone, U/L	3.2	0.4 to 5.0
Serum magnesium, mg/dL	1.7	1.7 to 2.1

Case Report

Table 2. Arterial Blood Gas Changes During Hospital Stay

Time	pH	HCO_3	pCO_2	pO_2	Base Excess
Admission	7.24	7.9	15.2	72.0	-14.0
1st day	7.25	11.8	27.5	65.1	-11.6
2nd day	7.36	17.0	31.0	62.5	-6.8
3rd day	7.44	25.0	44.0	64.6	-4.0

proteinuria, a pH of 6, and in urine microscopy, there was no evidence of oxalate calcium crystal (ethylene glycol intoxication). Blood gas analysis showed severe metabolic acidosis with a pH of 7.24 (Table 2). Based on the arterial blood gas data, the calculated anion gap was 25.1.

The patient was commenced on intravenous fluid and antiemetic. Due to severe vomiting, gastroenterology, hepatobiliary ultrasonography and upper gastrointestinal endoscopy were done, but no abnormality was found. After 3 days of hydration with dextrose in saline, gradually blood gas improved (Table 2), and her vomiting totally disappeared. At the 40th week of gestation, the patient was admitted for spontaneous labor and delivered a healthy infant.

DISCUSSION

Lactic acidosis occurs as a result of severe hypoperfusion of the liver due to shock or cardiopulmonary arrest. Definite diagnosis of this kind of acidosis is based on blood laboratory data.[2] Serum lactate level in our patient remained low, which made the diagnosis of lactic acidosis unlikely. In addition, advanced renal failure causes high anion gap acidosis due to accumulation of inorganic and organic acids.[2] However, our patient had normal kidney function. In adult patients, acidosis may be produced by the metabolism of toxic agents such as methanol and ethylene glycol.[3] Based on history and laboratory data (absence of oxalate calcium crystals in urine) the possibility of this diagnosis in our patient was considered low.

Ketoacidosis is due to excessive blood concentration of ketone bodies (acetoacetate, beta-hydroxybutyrate, and acetone).[4] The pathogenesis of all kinds of ketoacidosis is insulinopenia.[5] Severe and acute ketoacidosis following starvation is not common and starvation ketosis appears to require prolonged fasting, often 2 to 3 weeks or longer.[5] However, our patient did not have a history of diabetes mellitus or alcohol consumption, and her duration of fasting was too short. Based on the literature, insulin resistance increases in pregnancy, especially in the 3rd trimester, largely due to the production of human placental lactogen, cortisol, and glucagon by the placenta, and this tends to exaggerate any tendency to ketosis.[6]

We conclude in this case that acidosis was due to starvation and increase in insulin resistance, which especially aggravated in the 3rd trimester of pregnancy and led to ketone production. After hydration by glucose replacement, the patient's symptoms improved and discharged.

CONFLICT OF INTEREST

None declared.

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