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CASE REPORT

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A case of severe metabolic acidosis during pregnancy

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Key Clinical Message

Renal tubular acidosis (RTA) is a disorder that impairs renal acid-base regulation leading to normal anion gap metabolic acidosis. It is rare to encounter this entity during pregnancy. Pregnancy can worsen renal tubular acidosis (RTA) due to the physiological changes that happen during pregnancy. Teamwork including nephrologist and obstetrician is very important to identify and properly manage pregnant women with RTA to ensure smooth pregnancy and prevent complications.

KEYWORDS

hypokalemia, pregnancy, renal tubular acidosis, severe metabolic acidosis

1 | INTRODUCTION

We report a case of a 32-week pregnant woman presenting with profound hypokalemia and metabolic acidosis due to previously undiagnosed type 1 distal renal tubular acidosis.

2 | CASE REPORT

A twenty-seven-year-old woman presented at 32 weeks of pregnancy (gravida 3, para 1) with bilateral lower extremity weakness, abdominal pain, and nausea. Past medical history was notable for gestational diabetes and previous postpartum DVT. She had no previous surgical history. On presentation, she was afebrile with heart rate of 71 bpm and hypertensive at 151/97 mm Hg. Electrocardiogram (EKG) showed normal sinus rhythm without abnormalities. Significant upper and lower extremity weakness was noted on physical examination. MRI/MRA of the spine was unrevealing. Laboratories showed hypokalemia of 2.0 mmol/L, serum creatinine of 1.04 mg/dL, and mixed gap and nongap metabolic acidosis with serum bicarbonate of 8 mmol/L. Her lactic acid was within normal limits at 0.7 mmol/L. No ketones were identified on her first urinalysis but her serum ketones were positive. Review of laboratories from one year prior to presentation demonstrated hyperchloremic nongap metabolic acidosis with bicarb of 16. The patient also reported a previous history of hypokalemia requiring potassium supplementation which she had discontinued after running out of the prescription. Urine anion gap was positive at 17 and urine pH was 6.5, consistent with type 1 distal RTA (Table 1).

3 | CLINICAL COURSE

The patient was admitted to the intensive care unit for close fetal and cardiac monitoring. She was managed medically with intravenous (IV) fluid and electrolyte replacements. Anion gap was corrected, but nongap acidosis and hypokalemia persisted. The patient required high doses of intravenous and oral bicarbonate along with potassium supplementation to correct the metabolic abnormalities. Despite having received a total of 330 mEq of potassium in the first 24 hours, potassium remained low at 2.4. With continuous potassium replacement, her weakness and nausea resolved with normalization of potassium in range of 3.7-4.4 mmol/L. She was discharged with oral sodium bicarbonate tablets, 1950 mg four times daily. Initial laboratories after discharge showed recurrent metabolic acidosis due to medication nonadherence. The patient subsequently restarted her oral sodium

TABLE 1 Laboratory data

Serum studies	
Sodium	137 mmol/L (136-145)
Potassium	2.0 mmol/L (3.5-5.0)
Chloride	113 mmol/L (98-107)
Bicarbonate	8 mmol/L (22-29)
BUN	19 mg/dL (6-23)
Creatinine	1.04 mg/dL (0.6-1.0)
Glucose	171 mg/dL (74-109)
Calcium	8.7 mg/dL (8.4-10.2)
Magnesium	2.4 mg/dL (1.6-2.6)
Lactic acid	0.7 mmol/L (0.5-2.2)
Albumin	3.1 g/dL (3.5-5.2)
Anion gap	16 (5-14)
Arterial blood gas	
pH	7.09 (7.35-7.45)
pCO2	21 mm Hg (35-48)
pO2	118 mm Hg (83-108)
HCO3	6.4
Urine studies	
Specific gravity	1.01
Hemoglobin	Moderate (Negative)
pH	6.5 (4.5-8.0)
Urobilinogen	0.2 (Negative)
Nitrites	Negative (Negative)
Leukocyte esterase	Moderate (Negative)
White blood cells	10-19 (0-4)
Red blood cells	0-4 (0-4)
Bacteria	None
Creatinine	31.8 mg/dL
Sodium	32 mmol/L
Potassium	10.7 mmol/L
Chloride	25 mmol/L
Protein	60 mg/dL

bicarbonate tablets with initial improvement in her acidosis. However, despite ongoing oral supplementation she became more acidotic with bicarb of 14 and required IV repletion. The fetus remained healthy with no significant abnormalities identified through frequent fetal testing.

4 | DISCUSSION

Distal (type 1) renal tubular acidosis is an uncommon form of metabolic acidosis, which is either inherited or acquired. It is a tubular dysfunction defined by normal anion gap metabolic acidosis and inability to acidify the urine which is caused by reduced ammonium excretion.[1] The most common causes of distal RTA in adults are autoimmune diseases (eg, Sjögren's syndrome and rheumatoid arthritis) and hypercalciuria. Hereditary distal RTA is most common in children.[2] Drugs such as ifosfamide and amphotericin can cause distal RTA in both adults and children. Distal RTA can also cause hypokalemia that sometimes is profound to cause severe muscle weakness.[3] Distal RTA can also lead to increased loss of urinary calcium resulting in osteopenia, osteomalacia, nephrocalcinosis and even secondary hyperparathyroidism. Patients with distal RTA typically present with signs and symptoms related to severe hypokalemia such as proximal muscle weakness, polydipsia, and polyuria.

In our case, patient had features of distal RTA as her blood workup which was done before she was pregnant showed normal anion gap metabolic acidosis and hypokalemia. During her admission, she was found to have mainly normal anion gap metabolic acidosis. She denied any diarrhea. We sent urine analysis with urine electrolytes. Her calculated urine anion gap was positive pointing toward a renal dysfunction in the form of renal tubular acidosis that can explain her severe acidosis. Her urine pH was 6.5 despite her severe acidosis which means that she is unable to acidify her urine as seen in type 1 distal RTA. We could not identify what is the cause of her distal RTA, her autoimmune disease workup was supposed to be sent in our clinic but she did not follow-up on that. She denied any medications usage such as NSAIDS. She has never been exposed to amphotericin. She also never used or inhaled toluene. She used to be healthy before her pregnancy with no systemic illness. She only had gestational diabetes and postpartum DVT during her previous pregnancies. She denied any history of poor growth, constipation, vomiting, and polyuria. She denies any history of joints pain and skin rash. She also does not have eye dryness. She denies any hearing problems. Her renal ultrasound showed nephrocalcinosis which is likely the result of her chronic dRTA. As for treatment, she was not taking any potassium or alkali therapy before admission. At this point, we assumed that her distal RTA is likely idiopathic. She was having mild anion gap metabolic acidosis when she presented which is likely due to her ketoacidosis as her serum ketones were positive.

Little is known about the impact of RTAs in general on pregnancy, including mother and the child. However, chronic metabolic acidosis may affect fetal bone growth and fetal development. Also, chronic metabolic acidosis might compromise the fetal circulation leading to possible fetal distress or demise.[4] Pregnancy has been reported to worsen RTA. It is well understood that during pregnancy, there is mild respiratory alkalosis with some urinary bicarbonate loss as a compensation. The volume of distribution for bicarbonate increases as well during pregnancy.[5-7] These physiological factors and changes during pregnancy may explain worsening renal tubular acidosis. As in our

case, patient presented with really profound hypokalemia and metabolic acidosis with large bicarbonate deficit that required a lot of IV alkali therapy. In our case due to severe profound hypokalemia and acidosis, patient required IV potassium replacement along with IV bicarbonate. Once her hypokalemia improved then potassium supplements were stopped, and the focus was to correct her acidosis due to the fact that patients with distal RTA usually correct their hypokalemia by correcting their acidosis with alkali therapy. So chronically, most of these patients will require alkali therapy alone. Fetal monitoring during her hospitalization did not reveal any signs of fetal distress. Her muscle weakness improved a lot when her potassium normalized. After discharge, she needed two back to back admissions for IV bicarbonate due to her non-compliance with outpatient treatment. Alternatively, baking soda could be used as a treatment option. Each teaspoon of baking soda contains 4.8 g, corresponding to 59 mEq of sodium and 59 mEq of bicarbonate; by comparison, oral sodium bicarbonate tablets (650 mg) contain only 7.7 mEq of sodium and 7.7 mEq of bicarbonate. Baking soda may be an important alternative for noncompliant patients.

We were able to maintain her potassium in range of 3.5-4 mmol/L and serum bicarbonate in range of 14-17 mmol/L. She has delivered a healthy baby boy at 37.1 weeks of gestation, weighing 3460 g with Apgar score of 7 and 9 at 1 and 5 minutes, respectively. Her potassium was 4 mmol/L and serum bicarbonate 14 mmol/L on day of delivery. She has been clinically stable as per 6 weeks postpartum visit with maternal-fetal clinic. Her latest laboratories showed potassium of 3.4 mmol/L, serum bicarbonate 15 mmol/L, and serum creatinine of 0.8 mg/dL.

5 | CONCLUSION

Renal tubular acidosis gets worse during pregnancy. Increased doses of alkali therapy and potassium are needed. Regular follow-up and management by team of nephrologist and obstetrician are generally recommended to control the disease for better pregnancy outcome and prevent hospitalization.

CONFLICT OF INTEREST

Authors do not have any conflicts of interest at the time of publication of this case report.

AUTHOR CONTRIBUTION

MA: contributed to writing of discussion section. JJ: contributed to clinical history and course writing. GK: contributed to creating the initial case report draft, and editing it multiple times.

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