

## Case report

## A Rare Case of Renal Tubular Acidosis (type 1 Distal) in Pregnancy

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### Abstract

Distal renal tubular acidosis (type 1 RTA) is associated with systemic non-anion gap metabolic acidosis. It is predominantly due to impaired hydrogen ion secretion and impaired bicarbonate reabsorption in the distal nephrons of the kidney. RTA is rarely encountered during pregnancy and is associated with potential risks for the mother and fetus due to alterations in maternal acid-base status and electrolytes. Anesthetic management aims to ensure the well-being of the mother and fetus. We report, the anesthetic management of a 28-year-old, full-term G4P0A3 (gravida 4, para 0 and abortions 3) female with distal renal tubular acidosis (type 1 RTA) with recurrent history of familial hypokalemic periodic paralysis in the background of ultrasonography detected morphological disorder of both the kidneys and hypothyroidism who was posted for emergency LSCS.

**Keywords:** Renal tubular acidosis, Familial hypokalemic periodic paralysis, Pregnancy

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### Introduction

Distal renal tubular acidosis (type 1 RTA) is associated with systemic non-anion gap metabolic acidosis. It is predominantly due to impaired hydrogen ion secretion and impaired bicarbonate reabsorption in the distal nephrons of the kidney. Distal RTA (type 1 RTA) may be congenital or acquired (1). Renal tubular acidosis is rarely encountered during pregnancy and has been commonly reported with substance abuse like toluene and less commonly associated with systemic lupus erythematosus, hypothyroidism, Sjogren's syndrome, and diabetes mellitus. RTA may also have unclear etiology and may have a course of complete resolution after delivery (2, 3). In the case, which we present, the serum bicarbonate and serum potassium were low,

with metabolic acidosis and the symptoms started at the age of fifteen years. Moreover, this patient gave a history of three episodes of familial hypokalemic periodic paralysis (FHPP), a disorder of calcium channels. During each episode of paralysis, the patient's serum potassium and bicarbonate were very low. Urine was alkaline and the arterial blood gas (ABG) profile was acidic with a normal anion gap.

### Case Report

A 28-year-old, 38 weeks pregnant woman with height 160 centimeters and a weight of 54 kilograms, a diagnosed case of distal renal tubular acidosis (type 1 RTA) with associated hypokalemic periodic paralysis

and hypothyroidism presented with fetal distress and was posted for emergency lower segment Caesarean section (LSCS). She was a booked case and was on regular antenatal follow-up. She did not give any history of substance abuse or diabetes or any other comorbidities. Her serum TSH in the 1st trimester was 64 mU/l for which she was on tablet Eltroxin 150 mcg once daily throughout the antenatal period.

Urgent ABG was done immediately after admission, which revealed bicarbonate 14.5 mmol/l, and pH 7.35, serum potassium 2.8 mmol/l. Blood pressure was 110/80 mm Hg, SPO<sub>2</sub> 99% in room air, pulse rate 98/minute, and regular. Chest examination revealed bilateral vesicular breath sound and other systemic examinations were within normal limits. Her blood investigations done in the third trimester were normal except, serum bicarbonate level of 14.5 mmol/l and she was on syrup potassium citrate at a dose of 15 ml per oral diluted with 1 glass of water three times daily and tablet sodium bicarbonate at a dose of 500 mg three times a day. She had a history of recurrent abortions and kidney stones. The blood and urine investigation parameters in the third trimester were total lymphocyte count 8,500/cumm, Hb% 13.2 gm/dl, RBC 4.41 million/mm<sup>3</sup>, MCH 25.4 pg, MCV 77.3 fl, Platelets 2,00,000/cumm, Urea 24 mg/dl, Creatinine 0.56 mg/dl, Na<sup>+</sup> 139 mmol/l, K<sup>+</sup> 3.6 mmol/l. ABG after admission revealed HCO<sub>3</sub><sup>-</sup> 14.5 mmol/l, Cl<sup>-</sup> 105 mmol/l, pH 7.33, pCO<sub>2</sub> 25 mmHg, PO<sub>2</sub> 95 mm Hg. Urine pH was 5.8. Urine protein and glucose were negative.

Continuous electrocardiography, non-invasive blood pressure monitoring, and pulse oximetry probe were attached. Electronic fetal heart rate monitoring was done which revealed signs of fetal distress. Intravenous access was done in both hands. On one hand, the preloading was started with Ringer's solution and On the other hand, 50 ml sodium bicarbonate was administered over 10 minutes followed by 2 ampoules KCl in 50ml Normal Saline (NS) administered through a syringe pump at a rate of 12.5 ml/hour. Spinal anesthesia was administered with a 27 gauge Whittaker needle with 10 mg hyperbaric bupivacaine 0.5% plus 20mcg Fentanyl after proper aseptic precautions. Spinal anesthesia was given slowly over 20 seconds. After dressing and draping classical incision was given for LSCS. A live male infant weighing 2.2 kilograms

with an Apgar score of 8/9 was delivered. ABG was repeated after 12 hours. Serum bicarbonate was 16.5 mmol/l and serum potassium was 3.5 mmol/l.

Postoperatively, the patient received a bicarbonate intravenous drip measuring 150 mEq in 500 ml normal saline followed by syrup potassium citrate 15 mEq orally three times daily. The patient was discharged with syrup potassium citrate 10 mEq twice daily, Tablet sodium bicarbonate 500 mg thrice daily, and follow for RTA with the nephrology department.

## Discussion

Kidneys are one of the important buffer systems in the body that maintain normal systemic acid-base balance by controlling bicarbonate reabsorption and acid secretion. Loss of tubular function can prevent the kidneys from maintaining this balance (4). The group of diseases termed renal tubular acidosis is characterized by a non-anion gap hyperchloremic metabolic acidosis in the presence of preserved glomerular function. This is caused by the inability of the renal tubule to reabsorb bicarbonate or to secrete H<sup>+</sup> ions (5). Primary RTA is divided into hypokalemic (Type 2 proximal and Type 1 distal) and hyperkalemic (Type 4, associated with hyperaldosteronism) (6).

Distal RTA is the commonest form of RTA which occurs due to the inability of the renal tubules to decrease urine pH and increased urinary ammonium excretion with sustained hyperchloremic acidosis and hypokalemia (5). This is caused by a failure to reabsorb bicarbonate in the intercalated cells in the collecting duct. The disease can be inherited or can be a complication of other systemic diseases (7). Distal RTA can also lead to increased loss of urine calcium resulting in osteopenia, osteomalacia, nephrocalcinosis, and secondary hyperparathyroidism (2). Hypokalemic periodic paralysis which can occur due to calcium channel disorder is a rare disorder characterized by acute episodes of painless muscle weakness and is rarely associated with RTA Type 1. It may be precipitated by heavy exercise, fasting, and high carbohydrate meals. It may be associated with Sjogren's syndrome, RTA Type 1 or medullary sponge kidney or primary hyperaldosteronism, Barter's

syndrome, and Gitelman's syndrome (8). Usually, these patients respond to intravenous potassium and intravenous bicarbonate.

Our patient had the rarest of the rare combination of RTA (type 1 Distal) due to severe hypothyroidism with recurrent hypokalemic periodic paralysis. She also had some morphological disorder of the kidneys proven by ultrasonography. There are few case reports of RTA presenting for the first time in pregnant patients but our case was a known case presenting first at the age of fifteen years (2, 3, 9).

A medical literature review of RTA in pregnancy highlights that the same may be due to maternal systemic diseases like diabetes, Thyrotoxicosis, or substance abuse. Acute metabolic acidosis in pregnancy, which is a common finding in RTA has been suggested to cause decreased fetal circulation leading to possible fetal distress or demise (10). In our case also, there was a history of recurrent abortion, the etiology of which can be associated with metabolic acidosis. Seoud et al have also reported a case of preterm labor. Their case also resulted in the resolution of electrolyte abnormalities after delivery (3). Our case was a diagnosed case of RTA (type 1 Distal) with recurrent history of FHPP with severe hypothyroidism in the 1st Trimester. In our patient, there was low serum bicarbonate, hypokalemia, metabolic acidosis, and urine study was consistent with poor compensation by the tubular system (urine pH 5.8). These findings are consistent with the diagnosis of RTA (type 1 distal).

The treatment goals in such cases should be to keep serum bicarbonate and potassium within normal limits and to correct the acidosis as metabolic acidosis and hypokalemia can cause significant hypotension and can lead to cardiac arrhythmia even cardiac arrest in the perioperative period and independently may alter the fatal outcome. Moreover, it can precipitate FHPP, which can cause a diagnostic dilemma after spinal anesthesia. Fortunately, our patient was prepared adequately in the antenatal period with Tablet Eltroxin, Syrup Potassium citrate, and Tablet Sodium bicarbonate as mentioned above. The patient was prepared optimally before LSCS with intravenous KCl infusion and bicarbonate supplementation.

## Conclusion

To conclude, we presented a very rare case of Renal Tubular Acidosis (type 1 Distal) with a previous history of recurrent episodes of familial hypokalemic periodic paralysis, hypothyroidism, and multiple histories of abortions in the past. She was prepared well in the antenatal period but still had a decreased serum potassium and bicarbonate level, which were corrected quickly before the emergency LSCS. The recovery of the mother and baby was uneventful and treatment for Renal Tubular Acidosis was continued subsequently.

## Acknowledgment

None.

## Conflicts of Interest

The authors declare that there are no conflicts of interest.

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