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DISTAL RENAL TUBULAR ACIDOSIS IN PREGNANCY: A CASE REPORT AND SYSTEMATIC LITERATURE REVIEW

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ABSTRACT

Introduction: Distal renal tubular acidosis (dRTA) is a rare disorder characterized by normal anion gap metabolic acidosis and hypokalaemia. Physiological changes during pregnancy may aggravate acid-base imbalance and influence foetal outcomes.

Case description: A 40-year-old pregnant woman at 24-25 weeks of gestation presented with recurrent hypokalaemia and lower limb weakness. Laboratory findings showed normal anion gap metabolic acidosis, consistent with dRTA. The patient was treated with oral potassium supplementation and intravenous sodium bicarbonate, resulting in clinical improvement. In the third trimester, intrauterine growth restriction was identified. Delivery at term resulted in a live neonate with low birth weight.

A literature review was conducted according to Preferred Reporting Items for Systematic reviews and Meta-Analyses guidelines using PubMed, ScienceDirect, Wiley Online Library, SpringerLink, and ResearchGate for studies published between 2016 and 2026. Ten case reports met the inclusion criteria. Distal RTA was the most frequently reported subtype, accounting for 80% of cases. Most cases (70%) were first diagnosed during pregnancy. The most common treatment strategy was potassium supplementation combined with bicarbonate therapy (90%), administered either orally, intravenously, or as combination therapy. Adverse neonatal outcomes were reported in 50% of cases.

Conclusion: Distal RTA should be considered in pregnant patients presenting with recurrent hypokalaemia and muscle weakness. Early recognition and appropriate correction of electrolyte imbalance and metabolic acidosis are essential to optimize maternal and foetal condition. Further studies are needed to better define clinical characteristics and management strategies of renal tubular acidosis during pregnancy.

KEYWORDS

Distal renal tubular acidosis, pregnancy, hypokalaemia, metabolic acidosis

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LEARNING POINTS

- *Vigilance is essential for rare distal renal tubular acidosis (dRTA)-pregnancy overlap:* As the leading form of RTA, (seen in 80% of reported cases), distal RTA (dRTA) frequently first appears during pregnancy (70% of instances), where it closely resembles routine hypokalaemic conditions. Clinicians should routinely check for anion-gap metabolic acidosis in pregnant women experiencing repeated muscle weakness, allowing timely treatment to avert severe maternal decline.
- *Bicarbonate and potassium therapy boosts maternal-foetal success:* In this case, combined oral and intravenous replacement (effective in 90% of documented scenarios) quickly alleviated symptoms, yet the 50% chance of neonatal issues like intrauterine growth restriction or low birth weight underscores the approach's value for the foetus, providing internists with a reliable, safe strategy amid pregnancy's natural acid-base changes.
- *Diagnostic pathway to reveal hidden dRTA dangers:* This experience arms internists with a straightforward algorithm (start with hypokalaemia evaluation, advance to acidosis testing) that cuts late-pregnancy risks, especially in settings with limited resources.

INTRODUCTION

Renal tubular acidosis (RTA) impairs renal acid-base regulation, yielding hyperchloremic metabolic acidosis with a normal anion gap from defective urinary acidification. Distal RTA (type 1), the focus here, arises from impaired distal nephron hydrogen secretion, reducing ammonium excretion and sustaining acidosis. It manifests primarily or secondarily via autoimmune disorders, genetic defects, drugs, nephrocalcinosis, or tubulointerstitial disease[1–4].

Pregnancy intensifies distal RTA through renal adaptations elevated plasma flow, volume expansion, and an approximately 50% glomerular filtration rate increase amplifying electrolyte losses and bicarbonate/potassium demands. Resultant chronic acidosis and hypokalemia risk profound maternal-foetal complications: impaired foetal growth, circulatory disturbances, preterm labour, and neonatal acidosis. Muscle weakness exacerbates with foetal potassium demands and dilutional hypokalemia[2].

We report distal RTA diagnosed in pregnancy, with a literature review of its treatment and clinical characteristics.

CASE DESCRIPTION

A 40-year-old gravida presented at 24–25 weeks' gestation with bilateral lower-extremity numbness and weakness, nausea, vomiting, and mild dyspnoea, following a prior diagnosis of hypokalaemic periodic paralysis. She had recurrent hypokalaemia since early pregnancy without renal or autoimmune disease. Routine prenatal supplements included calcium, folic acid, and iron. Examination showed stable vital signs, intact orientation, and normal foetal well-being on cardiotocography and ultrasonography.

Laboratory evaluation revealed hypokalaemia (3.29 mmol/l), normal-anion-gap metabolic acidosis (pH 7.256, HCO_3^- 10.2 mmol/l), 24-hour urinary potassium 41 mmol, alkaline urine pH 7.5, normocytic normochromic anaemia, and normal thyroid function and magnesium. No clinical features suggestive of connective tissue disease were identified in this case. these findings supported distal renal tubular acidosis (type 1) unmasked or exacerbated by pregnancy-induced renal and electrolyte shifts.

Oral potassium and intravenous sodium bicarbonate produced gradual clinical improvement, and maternal foetal stability was maintained. At 32–33 weeks, ultrasonography first disclosed intrauterine growth restriction (IUGR). introducing additional risks of placental insufficiency and foetal compromise. At 37–38 weeks, with prior IUGR, active RTA with recurrent hypokalaemia, a caesarean section was performed. A live low birth weight neonate was delivered and managed per neonatal protocols, while maternal recovery remained stable with continued supplementation.

This literature review was conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines. A PRISMA flow diagram is provided in *Figure 1*. A comprehensive search was performed in PubMed, ScienceDirect, Wiley Online Library, SpringerLink, and ResearchGate for studies

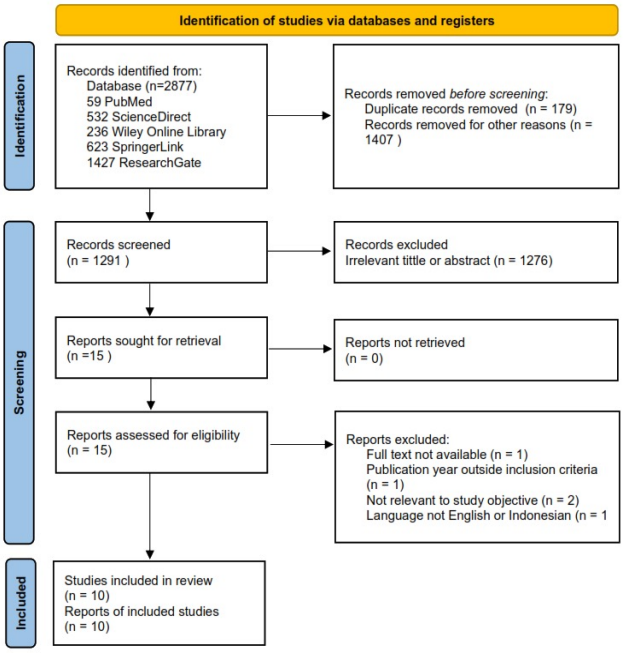


Figure 1. Preferred reporting items for systematic reviews and meta-analyses flow diagram.

published between January 2016 and January 2026. The search strategy used the keywords "renal tubular acidosis", "treatment", and "pregnancy", along with their synonyms. The search was limited to articles published in English or Indonesian.

The inclusion criteria were articles published between 2016 and 2026, written in English or Indonesian, and consisting of case reports or studies describing renal tubular acidosis during pregnancy with discussion of treatment. The exclusion criteria included duplicate articles, publications outside the 2016–2026 period, studies on renal tubular acidosis not occurring during pregnancy, and articles that did not address treatment.

Risk of bias assessment was conducted using the Joanna Briggs Institute critical appraisal tool, and all included studies were categorized as having low risk of bias. Based on these criteria, a total of 10 studies were included in the final analysis.

A total of 10 studies, all designed as case reports, were included in this review. The most frequently reported type of renal tubular acidosis was distal renal tubular acidosis (dRTA), accounting for 8 out of 10 cases (80%). Most dRTA cases were associated with autoimmune disorders, particularly Sjögren's syndrome and systemic lupus erythematosus (SLE). Less frequently reported subtypes included proximal RTA in 1 case (10%) and hyperkalemic RTA in 1 case (10%).

Regarding the timing of onset, the majority of cases were first diagnosed during pregnancy, reported in 7 out of 10 patients (70%). Two cases (20%) had a known diagnosis prior to pregnancy, while 1 case (10%) did not clearly specify the timing of onset.

The predominant treatment regimen entailed potassium supplementation coupled with bicarbonate therapy in 90% of cases (9/10), administered orally, intravenously, or combinatorically based on severity typically intravenous for acute episodes, transitioning to oral maintenance. A single study (10%) prioritized hyperkalemia correction via metabolic management. Collectively, interventions targeted core RTA pathophysiological hallmarks: hypokalemia and metabolic acidosis, underscoring a standardized, efficacious approach.

Adverse neonatal outcomes predominated in this review, affecting 5 of 10 cases (50%), with neonatal death reported in 2 (20%) and favorable outcomes defined as live birth without major complications in 2 (20%); 1 study (10%) lacked clear reporting. Adverse events encompassed prematurity, low birth weight, IUGR, neonatal intensive care unit admission, or metabolic issues, while neonatal death denoted mortality within 28 days postpartum. These findings underscore heightened neonatal vulnerability in such cohorts, warranting cautious clinical management.

DISCUSSION

This a 24-25-week pregnant woman with recurrent hypokalaemia, manifesting as lower extremity numbness and weakness. Laboratory findings revealed normal anion gap metabolic acidosis (low bicarbonate), urine pH 7.5, confirming distal renal tubular acidosis (dRTA) due to impaired distal nephron H^+ secretion, yielding defective urinary acidification and hypokalaemia. Consistent with prior reports, pregnancy-associated RTA typically features similar biochemistry—normal anion gap acidosis with hypokalaemia often presenting as muscle weakness or paralysis[5–7].

Various RTA subtypes distal, proximal, and type 4 have been reported in pregnancy, with distal RTA predominating[5,6]. Pathophysiologically, distal RTA stems from defective distal tubular acid secretion, culminating in metabolic acidosis and hypokalaemia[6,7]. Common manifestations encompass muscle weakness, fatigue, and hypokalaemic paralysis, driven by renal potassium wasting that impairs neuromuscular function[5–7]. Our patient initially exhibited limb weakness with a prior hypokalaemic periodic paralysis diagnosis, underscoring how pregnancy-associated RTA can mimic primary neuromuscular disorders, evading detection without metabolic scrutiny[5,6].

Physiological changes during pregnancy may contribute to the development or worsening of RTA. Pregnancy is associated with increased renal plasma flow and approximately 50% increase in glomerular filtration rate, which increases filtered solute load and urinary electrolyte loss[5]. Increased bicarbonate excretion, increased potassium requirement, and mild respiratory alkalosis with compensatory bicarbonate loss may further aggravate disturbances in acid base balance in patients with RTA[5,6,8]. Several reports indicate that RTA may first become clinically apparent during pregnancy due to these physiological adaptations[7,8]. In this case, the patient had no previous history of renal disease, but recurrent hypokalaemia occurred during pregnancy, suggesting that pregnancy may have unmasked an underlying tubular disorder[7,8].

The literature review suggests that the maternal condition generally improves after correction of electrolyte imbalance and metabolic acidosis[5,6]. However, foetal outcomes appear to be variable, as several studies have reported complications such as IUGR, possibly related to chronic metabolic acidosis and electrolyte imbalance[6]. Chronic metabolic acidosis may interfere with foetal growth and circulation, which may contribute to adverse neonatal outcomes[6]. In this case, foetal growth was initially appropriate for gestational age but later progressed to IUGR in the third trimester, followed by delivery of a low-birth-weight infant. The presence of maternal syphilis infection, which is an independent risk factor for low birth weight and impaired foetal growth, may also have contributed to the neonatal outcome. Therefore, the foetal outcome in this case may be multifactorial[9,10].

Management of RTA in pregnancy prioritizes hypokalaemia correction and metabolic acidosis treatment via alkali therapy. The literature underscores oral or intravenous potassium supplementation and sodium bicarbonate

as cornerstone interventions to restore electrolyte equilibrium and acid-base homeostasis. In hypokalaemic pregnant patients, bicarbonate repletion is critical, as uncorrected acidosis exacerbates maternal symptoms and risks adverse foetal outcomes[6,10].

In this case, combined oral potassium and intravenous sodium bicarbonate yielded progressive clinical amelioration and normalized laboratory indices, aligning seamlessly with established protocols. These observations advocate routine acid base evaluation, including serum bicarbonate, in gravid women with hypokalaemia to expedite RTA diagnosis and bicarbonate initiation.

This report spotlights recurrent hypokalaemia with myopathy in pregnancy as a red flag for occult metabolic derangements like RTA, rather than primary neurology. Prompt recognition and targeted therapy mitigate maternal foetal perils. Limitations include the single-case format and paucity of cohort data evidence remains case report dominant. Larger prospective studies are imperative to delineate RTA's clinical trajectory, perinatal impacts, and refined therapeutics in pregnancy.

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