RESEARCH PAPER

Hypokalemic Quadriplegia with Respiratory Failure due to Distal Renal Tubular Acidosis Secondary to Primary Sjögren's Syndrome in Early Pregnancy: A Case Report

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ABSTRACT

Background

Hypokalemic paralysis is a rare but potentially life-threatening medical emergency. Distal renal tubular acidosis (DRTA) is an uncommon cause of severe hypokalemia and may occur secondary to autoimmune disorders such as primary Sjögren's syndrome. Pregnancy can further aggravate electrolyte and acid-base disturbances.

Case Presentation

A 29-year-old pregnant female (P1L1) presented with progressive weakness of all four limbs for seven days, bleeding per vaginum for four days, and respiratory distress for one day. Clinical examination revealed quadriplegia with muscle power of 1/5 in all limbs. Laboratory investigations demonstrated severe hypokalemia (serum potassium 1.16 mEq/L), hyperchloremic normal anion gap metabolic acidosis, and elevated urine pH. Electrocardiography showed QTc prolongation, ST depression, T-wave inversion, and U waves. Ultrasonography revealed retained products of conception and bilateral nephrolithiasis. The patient subsequently developed respiratory failure requiring mechanical ventilation. Further evaluation revealed sicca symptoms, positive Schirmer's test, and positive anti-SSA antibodies, establishing the diagnosis of primary Sjögren's syndrome with distal renal tubular acidosis.

Management and Outcome

The patient received aggressive potassium replacement, sodium bicarbonate therapy, ventilatory support, and dilation and curettage for retained products of conception. Clinical and biochemical improvement occurred with correction of hypokalemia and metabolic acidosis. She was successfully extubated and discharged in stable condition.

Conclusion

Distal renal tubular acidosis secondary to primary Sjögren's syndrome should be considered in patients presenting with hypokalemic paralysis, particularly when associated with normal anion gap metabolic acidosis. Pregnancy may exacerbate underlying renal tubular dysfunction, increasing the risk of severe complications including respiratory failure.

Keywords: Hypokalemic paralysis; Distal renal tubular acidosis; Sjögren's syndrome; Pregnancy; Respiratory failure; Hypokalemia.

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Introduction:

Hypokalemic paralysis is characterized by acute onset muscle weakness resulting from severe potassium depletion. Acquired causes include thyrotoxicosis, gastrointestinal potassium losses, and renal tubular disorders. Distal renal tubular acidosis is among the important renal causes of

hypokalemia and may occur secondary to autoimmune diseases such as primary Sjögren's syndrome. Although distal renal tubular acidosis is reported in a significant proportion of patients with Sjögren's syndrome, clinically evident hypokalemic paralysis remains uncommon.

Pregnancy is associated with physiological changes in acid-base balance and renal handling of electrolytes, which may worsen pre-existing renal tubular dysfunction. We report a rare case of severe hypokalemic quadriplegia with respiratory failure due to distal renal tubular acidosis secondary to primary Sjögren's syndrome during early pregnancy.

Case Presentation:

A 29-year-old female, gravida 2 para 1 living 1, presented to the emergency department with amenorrhea of eight weeks, progressive weakness of all four limbs for seven days, Vaginal bleeding for four days, and difficulty in breathing for one day.

The weakness initially involved both lower limbs and gradually progressed to involve both upper limbs. There was no history suggestive of diarrhea, vomiting, or drug-induced potassium loss.

On examination, blood pressure was 110/70 mmHg, pulse rate was 104 beats/minute, respiratory rate was 22/minute, and temperature was 100°F. Pallor was present. Neurological examination revealed muscle power of 1/5 in all four limbs with preserved deep tendon reflexes. Abdominal examination revealed hypogastric tenderness.

Table 1. Baseline Clinical Features observed in the patient

S.No	Parameters	Findings
1	Age	29 years
2	Obstetric Status	P1L1
3	Gestation	8 weeks amenorrhea
4	Presenting Complaint	Progressive weakness of all four limbs
5	Duration of Weakness	7 days
6	Vaginal Bleeding	Vaginal Bleeding Present (4 days)
7	Respiratory Distress	Respiratory Distress Present (1 day)
8	BP	110/70 mmHg
9	Pulse Rate	104/min
10	Temperature	100°F
11	Muscle Power	1/5 in all four limbs

Investigations demonstrated severe hypokalemia (1.16 mEq/L), hyperchloremic normal anion gap metabolic acidosis, urine pH of 6.8, and normal thyroid function tests. Electrocardiography revealed QTc prolongation, ST-segment depression, T-wave inversion, and prominent U waves. Ultrasonography showed retained products of conception and bilateral nephrolithiasis as shown in table 2.

Table 2: Various Clinical Diagnostic Investigation

Investigation	Result
Potassium	1.16 mEq/L
pH	7.18
Bicarbonate	10.1 mEq/L
Anion Gap	13 mEq/L
Sodium	155 mEq/L
Chloride	131 mEq/L
Urine pH	6.8
TSH -.	2.2
Anti-dsDNA	Negative
Anti-Smith	Negative
Anti-SSA/Ro60	Positive
Anti-SSA/Ro52	Positive
Scl-70	Negative
USG Findings	Retained products of conception, bilateral nephrolithiasis
ECG Findings	QT prolongation, ST depression, T-wave inversion, U waves

Despite initiation of potassium replacement, the patient developed worsening respiratory distress and altered sensorium. Arterial blood gas analysis revealed severe respiratory compromise, necessitating endotracheal intubation and mechanical ventilation.

Further history revealed dryness of eyes and mouth. Schirmer's test was positive. Autoimmune evaluation demonstrated positivity for anti-SSA (Ro-52 and Ro-60) antibodies, while anti-dsDNA, anti-Smith, and Scl-70 antibodies were negative.

Based on clinical, biochemical, and immunological findings, a diagnosis of distal renal tubular acidosis secondary to primary Sjögren's syndrome complicated by pregnancy was established.

Treatment Approach:

Aggressive intravenous potassium replacement was initiated. Dilation and curettage was performed for retained products of conception. Sodium

bicarbonate therapy was added following confirmation of distal renal tubular acidosis. Supportive intensive care management and ventilatory support were continued until clinical improvement was achieved.

Progressive normalization of serum potassium levels and acid-base status was observed following treatment, with corresponding clinical improvement. The trend in key biochemical parameters is summarized in Table 3.

Table 3: Key parameters recorded on follow-up

Parameter	**Day1**	Day 2	**Day 3**	Day 8
Potassium (mEq/L)	1.16	1.66	3.16	3.84
pH	7.18	7.24	7.31	7.42
HCO_3^- (mEq/L)	10.1	12.6	14.9	19.4
Creatinine (mg/dL)	1.09	0.64	1.23	0.65

Result:

Serial investigations demonstrated progressive improvement in serum potassium levels from 1.16 mEq/L to 3.84 mEq/L and normalization of acid-base status. Muscle strength improved gradually, respiratory distress resolved, and the patient was successfully extubated. She was subsequently discharged in stable condition with advice regarding future pregnancy monitoring and continued follow-up.

Discussion

Distal renal tubular acidosis is characterized by impaired hydrogen ion secretion in the distal nephron, resulting in hyperchloremic normal anion gap metabolic acidosis. Hypokalemia develops due to increased urinary potassium losses and may occasionally be severe enough to cause paralysis and respiratory muscle involvement.

Primary Sjögren's syndrome is an autoimmune disorder predominantly affecting exocrine glands. Renal involvement is uncommon but may manifest as tubulointerstitial nephritis and distal renal tubular acidosis. Although distal renal tubular acidosis has been reported in up to 25–40% of patients with Sjögren's syndrome, severe hypokalemic paralysis is a rare initial presentation.

Pregnancy can worsen distal renal tubular acidosis through physiological respiratory alkalosis, increased glomerular filtration rate, and enhanced urinary losses of potassium and bicarbonate. These changes may precipitate life-threatening electrolyte disturbances in susceptible individuals.

The present case highlights the importance of considering hypokalemia and renal tubular acidosis in patients presenting with acute flaccid paralysis. Early recognition and prompt correction of electrolyte abnormalities are crucial to prevent complications such as respiratory failure and cardiac arrhythmias.

CONCLUSION

Severe hypokalemic paralysis with respiratory failure may be the initial manifestation of primary Sjögren's syndrome-associated distal renal tubular acidosis. Pregnancy can exacerbate the underlying metabolic disturbance and contribute to life-threatening complications. Prompt diagnosis and aggressive correction of hypokalemia are essential for favorable outcomes.