Case Report

# Recurrenthypokalemic Weakness Secondary to Sjogren's Syndrome: A dRTA Effect

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Renal involvement is observed in approximately 5% of patients with Sjögren's syndrome (SS), with tubulointerstitial nephritis and membranoproliferative glomerulonephritis being the predominant lesions[1]. We describe the case of a 50-year-old female who presented with lower limb weakness. The comprehensive evaluation revealed hypokalemic paralysis and subsequent investigations confirmed a diagnosis of type 1 distal renal tubular acidosis (dRTA) secondary to SS. The patient was managed with symptomatic treatment, including intravenous potassium chloride and sodium bicarbonate to correct hypokalemia and acidosis, alongside corticosteroids and hydroxychloroquine for immunomodulation of SS. Clinical improvement was observed with resolution of acidosis and stabilization of serum potassium levels. This case underscores the importance of considering dRTA in SS patients presenting with neuromuscular weakness and highlights the effective role of combined electrolyte correction and immunosuppressive therapy in such cases. The patient magically improved with the correction of acidosis and hypokalemia.

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## Introduction

Sjögren's syndrome is a chronic autoimmune disorder that predominantly targets the exocrine glands—most notably the lacrimal and salivary glands—resulting in the classic manifestations of keratoconjunctivitis sicca (dry eyes) and xerostomia (dry mouth). In addition to these hallmark symptoms, the syndrome often involves extra glandular organ systems such as the lungs, kidneys, and nervous system, contributing to a wide spectrum of clinical complications. The observation of high rates of autoimmune disorders in families with a history of Sjögren's syndrome suggests a strong genetic predisposition. Recent studies investigating polymorphisms in the human leukocyte antigen (HLA)-DR and HLA-DQ gene regions have demonstrated that variations in these loci are associated with differential susceptibility to the syndrome, likely due to their influence on the nature and extent of autoantibody production.[3] This complex interplay between genetic factors and immune dysregulation underscores the multifaceted pathogenesis of Sjögren's syndrome and highlights the potential for developing targeted therapeutic interventions.[2]

### *Case Report*

A 32-year-old female presented to an outside hospital with complaints of difficulty in passing stools and progressive weakness in the lower limbs over several days. Notably, her symptoms were not accompanied by vomiting, diarrhea, or fever. On physical examination, she exhibited hypotonia and areflexia, with no focal neurological deficits. Additionally, she reported fatigue, along with dryness of the eyes and mouth. On admission, her vital signs were stable (blood pressure 120/80 mm Hg, pulse 68 beats/min, temperature 36.6°C). Abdominal examination revealed a tense abdomen with high-pitched bowel sounds. Arterial blood gas analysis demonstrated severe metabolic acidosis and the electrocardiogram (ECG) was remarkable for the presence of prominent U waves, consistent with hypokalemia. In view of the severe hypokalemia combined with a normal anion gap metabolic acidosis, a diagnosis of distal renal tubular acidosis (dRTA) was established, and

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intravenous potassium supplementation was initiated. The patient's past medical history was significant for recurrent hospitalizations over the preceding two years due to episodes of paraparesis secondary to hypokalemic crises, which had resolved with potassium correction; however, no further investigations had been undertaken to determine the underlying etiology of her hypokalemia. She denied any family history of kidney disease. Her history was also notable for a poorly functioning left kidney complicated by pyonephrosis and multiple renal calculi. This clinical presentation, along with the recurrent nature of her hypokalemic episodes and the background of structural renal abnormalities, prompted further evaluation for an underlying systemic process contributing to dRTA. Subsequent workup for autoimmune conditions was initiated based on her sicca symptoms, eventually leading to a diagnosis of Sjögren's syndrome.

We had retained the diagnosis of primary SS because she had a score according to the 2016 American College of Rheumatology/European League Against Rheumatism Classification Criteria for Primary SS at 4 (The Schirmer test positive [score = 1], and anti-SSA/Ro positive [score = 3]), and she had no exclusion criteria. Primary SS was identified as the cause of type 1 renal tubular acidosis.

The patient received symptomatic treatment with injectable potassium chloride, sodium bicarbonate, hydration, and a low-protein diet. In terms of etiological treatment, she was given corticosteroids and hydroxychloroquine. The outcome was favorable, with the correction of acidosis and hypokalemia. She improved with these measures, and the power in her lower limbs recovered. She was discharged with oral potassium and bicarbonate supplements and on discharge her serum potassium levels were 4.2 mmol/L.

### *Future Follow-Up Plan*

For long-term management, the patient was advised to undergo regular monitoring with periodic serum electrolyte assessments, renal function tests (including serum creatinine and eGFR), and imaging studies to detect any progression of nephrocalcinosis or structural renal changes. Given the underlying renal tubular dysfunction associated with Sjögren's syndrome, the use of NSAIDs was strictly restricted to prevent further renal impairment.

## Discussion

The case of our 32-year-old female, who presented with recurrent episodes of hypokalemic paralysis, underscores the critical need to evaluate for underlying renal tubular dysfunction in patients with Sjögren's syndrome (SS). The pathogenesis of dRTA in SS is believed to result from autoimmune lymphocytic infiltration of the distal nephron, particularly targeting the $\alpha$-intercalated cells. This infiltration impairs the secretion of hydrogen ions, thereby compromising urinary acidification and leading

**Table 1:** Usg abdomen pelvis

| |
|---|
| Multiple altered echotexture areas in the cortex of the left kidney with a prominent left renal pelvicalyceal system with internal moving echo - Pyelonephrltis with Pyonephrosis.<br>Left renal calculi. |

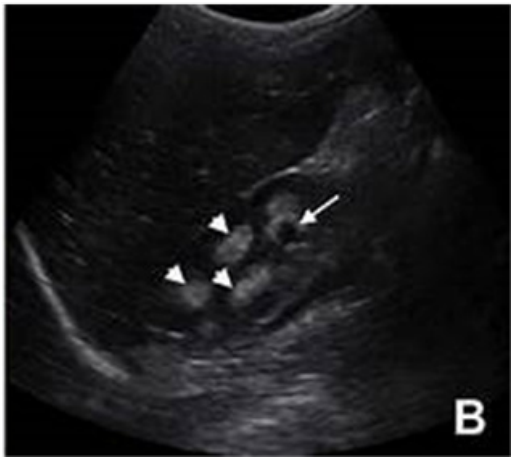


**Table 2:** ECG

| |
|---|
| Normal axis<br>Regular rhythm<br>60/min rate<br>U waves present. |

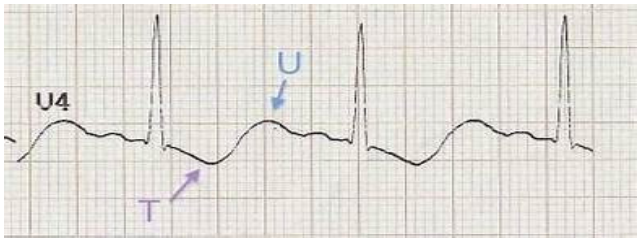


**Figure 1:** (ECG on admission)

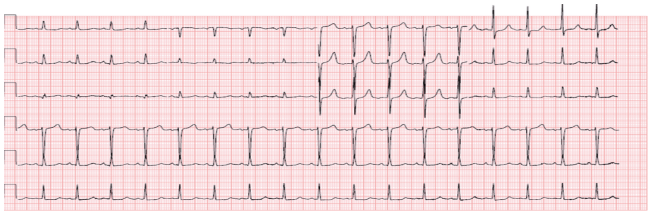

**Figure 2:** (Post-treatment ECG)

**Table 3:** Biochemical markers

| | |
|---|---|
| CBC | 13.2/14,200/1.70k |
| ESR | 14 mm/hr |
| CRP | 7.94 mg/L |
| URINE Routine Micro | +1 Albuminuria |
| Potassium levels (on Admission) - Potassium levels (Post Treatment) - | 1.9 mmol/L 4.2 mmol/L |
| Sodium levels | 149 mmol/L |
| Chloride levels | 112 mmol/L |
| Urea levels | 23 |
| Sr. Creatinine | 1.2 mg/dl (eGFR-52.1 mL/min/1.73 $m^2$) |
| Uric acid | 3.3 mg/dl |
| RA factor | Negative |
| HCO3 | 21 |
| Sr. magnesium levels | 2.41 |
| TSH | 2.20 |

**Table 4:** Autoimmune work-up

| | |
|---|---|
| ANA TITER- IFA | 1:100 (normal <1:40) |
| Pattern | A granular speckled pattern seen |
| R0-60 | Positive (50 intensity) |
| Ro 52 recombinant (52) | 76 |
| Schirmer test | Positive (3 mm in right eye and 2 mm in left eye per 5 minutes) |

to normal anion-gap metabolic acidosis. The resulting electrolyte disturbance, particularly the pronounced hypokalemia, is a frequent precipitant of neuromuscular symptoms such as weakness and paralysis. In our patient, despite a preserved overall renal function (serum creatinine 1.2 mg/dL), the history of a poorly functioning left kidney and prior episodes of pyonephrosis with multiple calculi likely compounded the clinical severity.[4]

Management of dRTA in the context of SS involves both symptomatic and disease-specific strategies. Prompt correction of hypokalemia and acidosis through electrolyte replacement and alkali therapy is critical to prevent life-threatening complications. Additionally, immunomodulatory therapy with corticosteroids and agents like hydroxychloroquine can attenuate the autoimmune process, potentially reducing further renal injury.[5] Our patient's marked clinical improvement following these interventions highlights the importance of early recognition and comprehensive management of this rare presentation.

## Conclusion

In patients presenting with paraparesis secondary to hypokalemia with metabolic acidosis, evaluation of underlying causes such as renal tubular acidosis associated with Sjogren's syndrome should be considered (dRTA1 due to SS).

Hypokalemic paralysis can also be due to renal tubular acidosis secondary to an autoimmune disorder just like in our case one should always consider autoimmune disorders as one of the causes. Delay in the diagnosis can be avoided by meticulous search for the underlying cause of hypokalemia and evaluating the symptoms such as dry eyes, dry mouth, and fatigue.

## Ethics

Written informed consent was obtained from the patient for publication.

## Conflict of Interest

The authors have no conflicts of interest to declare.

## References

1. Thervet E. Traité de néphrologie. Paris: Lavoisier; 2017.
2. Francois H, Mariette X. Renal involvement in primary Sjogren syndrome. Nat Rev Nephrol. 2015
3. Kim YK, Song HC, Kim WY, Yoon HE, Choi YJ, Ki CS, et al. Acquired Gitelman syndrome in a patient with primary Sjögren syndrome. Am J Kidney Dis. 2008;52:1163–7.
4. Vitali C, Bombardieri S, Jonsson R, et al. Classification criteria for Sjögren's syndrome: a revised version of the European criteria proposed by the American-European Consensus Group. Ann Rheum Dis. 2002;61(6):554–558.
5. Takemoto F, Katori H, Sawa N, Hoshino J, Suwabe T, Sogawa Y, et al. Induction of anti-carbonic-anhydrase-II antibody causes renal tubular acidosis in a mouse model of Sjogren's syndrome. Nephron Physiol. 2007;106: p63–8