RESEARCH PAPER

A 22-Year-Old Postpartum Female with Quadriparesis Secondary to Sjögren's Syndrome: A Case Report

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ABSTRACT

Background: Sjögren's syndrome is a chronic autoimmune exocrinopathy that may involve multiple organ systems. Renal involvement in the form of distal renal tubular acidosis (dRTA) can lead to severe hypokalemia, occasionally manifesting as acute flaccid motor weakness, which can mimic neuromuscular or endocrine emergencies in postpartum females.

Case Presentation: A 22-year-old woman, six months postpartum, presented with acute onset progressive quadriparesis associated with severe lower limb pain and one brief episode of loss of consciousness. Initial clinical suspicion included Sheehan's syndrome due to postpartum status and hypotension. Examination showed symmetric quadriparesis with preserved reflexes. Investigations revealed persistent hypokalemia and normal anion gap metabolic acidosis. Autoimmune evaluation demonstrated ANA, Anti-Ro/SSA and Anti-La/SSB positivity. Schirmer's test confirmed ocular dryness. She was diagnosed with Sjögren's syndrome–associated distal RTA. Potassium and bicarbonate therapy led to rapid improvement. In postpartum women presenting with quadriparesis, autoimmune dRTA should be considered, especially when persistent hypokalemia accompanies metabolic acidosis. Early diagnosis of Sjögren's syndrome enables targeted therapy and prevents long-term complications.

Keywords: Sjögren's syndrome, distal renal tubular acidosis, postpartum quadriparesis, hypokalemia, autoimmune disease.

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Introduction

Sjögren's syndrome is a chronic, systemic autoimmune disorder characterized predominantly by lymphocytic infiltration of the exocrine glands, resulting in the classical manifestations of keratoconjunctivitis sicca and xerostomia (1). It primarily affects women, particularly in the third to fifth decades of life, and may occur as either primary Sjögren's syndrome or secondary to other autoimmune conditions such as rheumatoid arthritis and systemic lupus erythematosus (1). Although glandular involvement is the hallmark, up to one-third of patients develop extraglandular manifestations that can involve the renal, pulmonary, neurological, vascular, and musculoskeletal systems (2). Among these, renal involvement particularly distal renal tubular acidosis (dRTA) is well described but often under-recognized in early stages. dRTA results from the inability of the distal nephron to excrete hydrogen ions, leading to hyperchloremic normal anion gap metabolic acidosis and secondary potassium wasting (3).

Hypokalemia resulting from dRTA may be severe enough to cause flaccid motor weakness, paralysis, or even life-threatening arrhythmias. While hypokalemic paralysis is a known but uncommon presentation of Sjögren's syndrome, it poses a significant diagnostic challenge because it can mimic various neurological, metabolic, or endocrine emergencies (4). The situation becomes more complex in postpartum females, who present a unique diagnostic landscape due to physiological, hormonal, and hemodynamic changes associated with pregnancy and childbirth (5). In the postpartum period, neuromuscular weakness accompanied by hypotension or altered consciousness often prompts clinical suspicion of Sheehan's syndrome, an endocrinopathy caused by postpartum pituitary infarction. This frequently leads physicians to prioritize endocrine evaluations before considering autoimmune or metabolic causes (6).

Moreover, renal tubular involvement in Sjögren's syndrome may precede the development of classical sicca symptoms, leading to diagnostic delays. Patients may initially lack prominent ocular or oral dryness, and autoimmune serology may be the only early indicator of the disease (7). Consequently, presentations dominated by muscular weakness or altered sensorium due to profound hypokalemia may mislead clinicians toward primary neurological disorders such as Guillain–Barré syndrome, periodic paralysis, myelopathies, or acute myopathies. A high index of suspicion is therefore required to recognize dRTA as a potential underlying cause in such atypical scenarios (8).

The postpartum state further complicates the diagnostic process by introducing overlapping symptoms that may be attributed to obstetric or endocrine causes. Hypotension, fatigue, muscle weakness, or episodes of altered consciousness common features in dRTA may also be seen in postpartum hemorrhage–related complications or endocrine dysfunctions (9). Thus, differentiating these conditions requires careful evaluation of biochemical, hormonal, and autoimmune parameters. Identification of hypokalemia with a normal anion gap metabolic acidosis strongly suggests renal tubular pathology and shifts the diagnostic direction toward distal RTA (10).

This case report presents a 22-year-old, six-month postpartum female who developed acute quadriparesis secondary to severe hypokalemia from Sjögren's syndrome–associated dRTA. Her presentation initially raised suspicion of Sheehan's syndrome due to hypotension and a brief loss of consciousness. Detailed metabolic evaluation, however, revealed dRTA as the underlying pathology, leading to the diagnosis of Sjögren's syndrome. This case underscores the importance of considering renal tubular acidosis in postpartum women presenting with acute neuromuscular weakness and highlights the need for early recognition of atypical extraglandular manifestations of autoimmune diseases.

CASE PRESENTATION

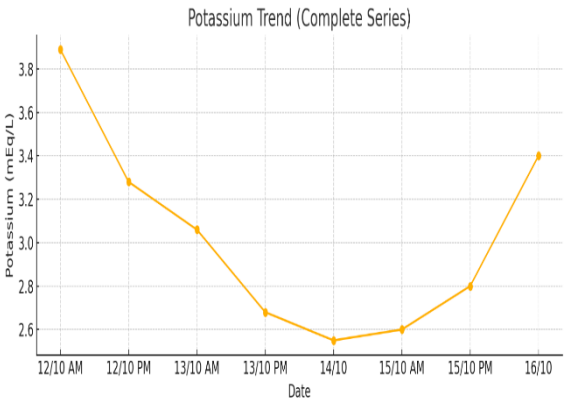
Patient Profile and Initial Presentation: A 22-year-old woman, six months postpartum, presented to the emergency department with a seven-day history of progressively worsening weakness involving all four limbs. The weakness began in the lower limbs, gradually ascended to the upper limbs, and was associated with severe aching pain in both lower limbs. One day prior to admission, she experienced a brief episode of loss of consciousness at home, after which she was noted to be markedly weak and unable to stand without support. On arrival, she was drowsy but responsive, and her blood pressure measured 90/60 mmHg. Due to her postpartum status, hypotension, and transient altered sensorium, an initial working diagnosis of Sheehan's syndrome was considered.

History and Symptom Review: There was no history of postpartum hemorrhage, lactation failure, seizures, fever, or recent infections. She denied exposure to toxins, alcohol, recreational drugs, or any medications that could precipitate electrolyte disturbances. There were no preceding gastrointestinal, respiratory, or genitourinary symptoms. She reported no dryness of eyes or mouth at presentation, and no family history of autoimmune disorders. Her obstetric history was unremarkable, with a normal vaginal delivery six months earlier.

Physical Examination: General examination revealed a conscious but fatigued patient with stable respiratory parameters. Neurological examination showed symmetric quadriparesis. Muscle strength was significantly reduced in both upper and lower limbs, with more pronounced involvement of proximal muscles. Deep tendon reflexes were preserved throughout, and there were no sensory deficits, cranial nerve abnormalities, or signs of meningeal irritation. Gait could not be assessed due to inability to stand. Cardiovascular and respiratory examinations were unremarkable, apart from hypotension.

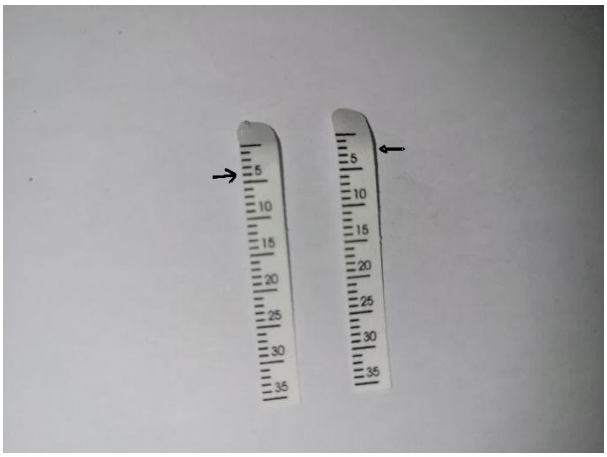
Laboratory Investigations: Initial biochemical evaluation revealed persistent hypokalemia, with serum potassium levels progressively declining to as low as 2.55 mEq/L. Serial serum electrolyte measurements demonstrated fluctuating but consistently low potassium levels despite oral and intravenous replacement, accompanied by hyperchloremia and normal anion gap metabolic acidosis, suggestive of distal renal tubular acidosis (dRTA). Sodium levels remained within normal limits. Renal function tests were normal, ruling out acute kidney injury.

Autoimmune workup revealed positive antinuclear antibodies (ANA) along with anti-Ro/SSA and anti-La/SSB antibodies. Schirmer's test performed subsequently showed reduced tear secretion (4 mm in the right eye and 3 mm in the left eye), confirming ocular dryness. Hormonal evaluation and pituitary imaging were within normal limits, effectively excluding Sheehan's syndrome.



Date	Na	K	HCO_3^-	Cl
12/10 AM	142	3.89	12	-
12/10 PM	136	3.28	-	-
13/10 AM	141	3.06	19.9	106
13/10 PM	139	2.68	29.8	-
14/10	-	2.55	-	-
15/10 AM	140	2.6	-	96
15/10 PM	-	2.8	-	-
16/10	-	3.4	-	-

Schirmer's Test: Positive – Right Eye – 4mm, Left Eye – 3mm



Cardiac and Imaging Studies: Echocardiography revealed normal resting left ventricular systolic function with no regional wall motion abnormalities, indicating preserved cardiac contractility. The mitral valve showed annular calcification with trivial mitral regurgitation, while the aortic valve appeared thickened with trivial aortic regurgitation. Trivial tricuspid regurgitation was also noted, with a pressure gradient of 27 mmHg. The estimated pulmonary artery pressure measured 32 mmHg, consistent with mild pulmonary arterial hypertension. Additionally, grade 1 diastolic dysfunction was observed, suggesting early diastolic relaxation abnormality without clinical significance. Overall, these findings were incidental and not contributory to the patient's neuromuscular presentation. No structural cardiac lesions or imaging abnormalities were identified that could explain the episode of quadriparesis or hypotension.

Treatment and Clinical Course: The patient received aggressive potassium supplementation along with bicarbonate therapy to correct metabolic acidosis. Intravenous fluids were administered for hemodynamic stabilization. Over the next 48 to 72 hours, her muscle strength began to improve significantly. Repeat electrolyte measurements showed normalization of serum potassium levels, and her blood pressure stabilized without vasopressor support. By the fifth day of hospitalization, she regained the ability to stand and walk with near-complete resolution of weakness.

Follow-Up: She was discharged on oral alkali therapy with advice for regular rheumatology follow-up. At follow-up review, she remained clinically stable with no recurrence of weakness. Early diagnosis and targeted therapy prevented long-term neuromuscular or renal complications.

DISCUSSION

Sjögren's syndrome is a chronic autoimmune exocrinopathy with diverse extraglandular manifestations that may overshadow the classical symptoms of xerostomia and keratoconjunctivitis sicca. Among these, renal involvement particularly distal renal tubular acidosis (dRTA) is a recognized

but frequently underdiagnosed feature, especially when it precedes overt glandular involvement (11). In the present case, distal RTA manifested primarily through persistent hypokalemia, which in turn produced acute flaccid quadriparesis. Such a presentation in a young postpartum woman can easily be misinterpreted as a neurological or endocrine emergency, underscoring the complexity of diagnostic evaluation in such settings.

Hypokalemia-induced paralysis results from reduced resting membrane potential and impaired muscle fiber excitability. The degree of weakness typically correlates with the severity of hypokalemia, and serum potassium levels below 2.5 mEq/L may lead to profound paralysis (12). In this patient, potassium levels fluctuated between 2.55 and 2.8 mEq/L despite replacement therapy, suggesting ongoing renal potassium losses. The presence of normal anion gap metabolic acidosis with hyperchloremia supported the diagnosis of dRTA. The postpartum period can act as a confounding factor because clinicians often first suspect Sheehan's syndrome in the presence of hypotension and altered consciousness. However, the absence of postpartum hemorrhage, preserved lactation, and normal pituitary hormonal evaluation made Sheehan's syndrome unlikely (13).

Neurological differentials such as Guillain–Barré syndrome, periodic paralysis, myopathies, and spinal cord lesions were considered but ruled out based on preserved deep tendon reflexes, absence of sensory deficits, and spontaneous clinical improvement with potassium correction. This reinforces the need for a thorough metabolic work-up in any patient with acute flaccid paralysis, particularly when initial findings do not support primary neurological pathology (14).

The autoimmune evaluation played a crucial role in establishing the underlying etiology. Strong positivity for ANA, Anti-Ro/SSA, and Anti-La/SSB antibodies, along with an abnormal Schirmer's test, confirmed the diagnosis of Sjögren's syndrome (15). Interestingly, the patient did not initially present with dryness of eyes or mouth a finding consistent with literature reporting that extraglandular manifestations may precede classical symptoms in a significant proportion of cases. Renal tubular acidosis is often the earliest manifestation of autoimmune renal involvement and can present years before glandular disease becomes clinically apparent.

Early recognition of dRTA is critical, as untreated metabolic acidosis leads to nephrocalcinosis, osteomalacia, nephrolithiasis, and progressive renal impairment. Moreover, recurrent episodes of hypokalemia may predispose patients to life-threatening arrhythmias and repeated paralytic attacks (16). In this case, prompt initiation of potassium and bicarbonate therapy resulted in rapid symptomatic improvement and normalization of biochemical parameters. This highlights the importance of recognizing renal tubular pathology early, especially in atypical clinical presentations.

This case emphasizes the need for heightened awareness of autoimmune causes of dRTA in postpartum patients presenting with acute neuromuscular weakness. A multidisciplinary approach involving internal medicine, nephrology, and rheumatology is essential to ensure accurate diagnosis and long-term management. With early detection and appropriate therapy, patients can achieve excellent neurological recovery and prevent chronic renal complications.

CONCLUSION

This case illustrates the diagnostic challenges posed by atypical presentations of Sjögren's syndrome, particularly in postpartum women. The patient presented with acute quadriparesis due to severe hypokalemia resulting from distal renal tubular acidosis, initially mimicking endocrine and neurological emergencies. The absence of classical sicca symptoms and the confounding postpartum context contributed to diagnostic delay. However, persistent hypokalemia with normal anion gap metabolic acidosis guided evaluation toward renal tubular pathology. Autoimmune serology and Schirmer's test ultimately confirmed Sjögren's syndrome. Timely correction of electrolyte disturbances and acidosis led to rapid improvement and complete neurological recovery. This case reinforces the importance of considering autoimmune dRTA in postpartum patients presenting with unexplained weakness and underscores the value of early metabolic assessment. Prompt diagnosis and treatment are crucial to preventing long-term renal and neuromuscular complications.

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