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PARALYSIS BEFORE THE DRYNESS: A DIAGNOSTIC CLUE TO UNDERLYING SJÖGREN SYNDROME

General Medicine

Dr. Gaurav Sharma* Post Graduate Resident in General Medicine at RKDF Medical College Hospital and Research Centre, Bhopal, M.P. *Corresponding Author

Dr. Priyanka Mishra Post Graduate Resident in General Medicine at RKDF Medical College Hospital and Research Centre, Bhopal, M.P.

Dr. Akash Wardiya Post Graduate Resident in General Medicine at RKDF Medical College Hospital and Research Centre, Bhopal, M.P.

ABSTRACT

Sjögren syndrome (SS) is a chronic systemic autoimmune disorder primarily affecting the salivary and lacrimal glands; However, extraglandular involvement may appear before conventional sicca features and predominate in the clinical presentation. Distal renal tubular acidosis (dRTA), which can cause severe hypokalemia and, in rare cases, acute flaccid paralysis, is frequently caused by tubulointerstitial nephritis. We describe a 45-year-old woman who, over the course of 12 hours, developed sudden paralysis in all four limbs without any prior infection, fever, gastrointestinal symptoms, or history of vaccinations. Examination showed no involvement of the cranial nerves and flaccid areflexic quadriparesis (upper limbs 2/5, lower limbs 1/5) with preserved sensory functions. A laboratory analysis revealed normal anion gap metabolic acidosis (pH 7.21, HCO_3^- 12 mmol/L, anion gap 12) with hyperchloremia and severe hypokalemia (serum K^+ 1.5 mmol/L). Urine pH was abnormally alkaline (7.5), which is consistent with dRTA, and the urine potassium–creatinine ratio confirmed renal potassium loss. Renal function, (rule out thyrotoxic periodic paralysis), thyroid profile, and glycemic parameters were normal, and nerve conduction studies showed no evidence of neuropathy, thereby excluding primary neurological causes such as Guillain–Barré syndrome. The diagnosis of underlying SS was confirmed by a reduced Schirmer test (3–4 mm/5 min). Within 24 hours of receiving intravenous potassium chloride and sodium bicarbonate, the patient's motor strength dramatically improved with positive SSA/Ro antibody, indicating hypokalemic paralysis caused by dRTA. For long-term care, corticosteroids and immunomodulators were then started. This case emphasizes the significance of taking into account metabolic and autoimmune etiologies in acute flaccid paralysis to prevent misdiagnosis and guarantee prompt, targeted therapy. It also shows that "paralysis before dryness" may be an early clue to SS.

KEYWORDS

Sjögren Syndrome, Hypokalemic Paralysis, Distal Renal Tubular Acidosis, Guillain–Barré Syndrome Mimic, Autoimmune Disease, Extra Glandular Manifestation

INTRODUCTION

Sjögren syndrome (SS) is a chronic systemic autoimmune disease characterized by lymphocytic infiltration and progressive destruction of the exocrine glands, predominantly the salivary and lacrimal glands (Zhan et al., 2023). It usually manifests as keratoconjunctivitis sicca and xerostomia in middle-aged women (Sanjeevani et al., 2025; Berman et al. 2019). According to epidemiological research, the prevalence peaks in the fourth and fifth decades of life, with a female predominance of up to 9:1 (Fox, 2005; Ramos-Casals et al., 2012). However, extraglandular signs may arise in around one-third to half of patients during the course of the disease, and SS is now commonly acknowledged as a multisystem disorder (Vitali et al., 2002).

The most typical manifestation of renal involvement in SS is chronic tubulointerstitial nephritis, which results in abnormalities in tubular acidification (Sanjeevani et al., 2025). The most common kidney symptom, distal renal tubular acidosis (dRTA), is caused by decreased hydrogen ion production in the distal nephron, which results in hypokalemia and normal anion gap (hyperchloremic) metabolic acidosis (Evans et al., 2015; Ren et al., 2008). Acute flaccid paralysis is an uncommon symptom of severe hypokalemia, which can be brought on by persistent potassium loss in the urine. Hypokalemic paralysis is an uncommon initial presentation of SS and may occur before overt sicca symptoms, despite being a documented consequence of dRTA (Morović-Vergles et al., 2007).

When areflexic quadriparesis is present, acute hypokalemic paralysis might clinically resemble neurological emergencies like Guillain-Barré syndrome (GBS) (Guo et al. 2025). In contrast to GBS, metabolic paralysis is characterized by quick recovery after electrolyte correction, maintained sensory function, and frequently no preceding infection. Therefore, early detection of metabolic acidosis with hypokalemia is essential to prevent misdiagnosis and guarantee prompt treatment.

This case emphasizes how crucial it is to take SS into account when a patient presents with hypokalemic paralysis that cannot be explained, especially if there is laboratory evidence of dRTA and subtle sicca symptoms.

Case Presentation

Over the course of 12 hours, a 45-year-old woman experienced acute, progressive weakness in all four limbs, making it impossible for her to stand and carry out daily tasks. The weakness was symmetrical and unrelated to bladder/bowel problems, cranial nerve involvement, or sensory loss. There was no history of toxin exposure, recent vaccinations, respiratory or gastrointestinal infections, or preceding fever. For several weeks, she complained of generalized fatigue, minor gritty eye sensation, and dry mouth. She denied experiencing similar events in the past and denied using any drugs that could lead to dryness symptoms and electrolyte imbalance .

Examination

The patient had stable vital signs, was afebrile, and had pallor. She was aware, focused, and awake. The evaluation of the cranial nerves was normal. A motor examination showed symmetrical flaccid quadriparesis, with upper limb power of 2/5 and lower limb power of 1/5. Deep tendon reflexes were absent, and plantar responses were diminished bilaterally. Sensory examination was intact. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

Investigation

Laboratory evaluation revealed moderate normocytic anemia with preserved leukocyte and platelet counts. Renal function parameters were within normal limits (Table 1). However, severe hypokalemia with biochemical evidence of renal potassium wasting and normal anion gap metabolic acidosis was noted (Table 2). Urine alkalinity and a shortened Schirmer test suggested distal renal tubular acidosis (dRTA) in the background of autoimmune disease (Table 4). Autoimmune serology showed markedly elevated ANA and SSA/Ro antibodies (Table 3).

Table 1. Hematological and Biochemical Parameters

Parameter	Result	Reference Range	Interpretation
Hb	8.5 g/dL	12–15 g/dL	Moderate anemia
MCV	86 fL	80–100 fL	Normocytic indices
Platelets	1.6 lakh/µL	1.5–4.0 lakh/µL	Normal

TLC	7,600/µL	4,000–11,000/µL	Normal
Creatinine	0.9 mg/dL	0.6–1.2 mg/dL	Normal renal function
Urea	36 mg/dL	15–45 mg/dL	Normal
Serum Potassium (K^+)	1.5 mmol/L	3.5–5.0 mmol/L	Severe hypokalemia
Urine K^+/Creatinine Ratio	16 mEq/g	<13 mEq/g	Renal potassium loss
Urine pH	7.5	<5.5 (in acidosis)	Inappropriately alkaline
Thyroid Profile	Normal		Normal
HbA1c	Normal		No diabetes

Table 2. Arterial Blood Gas (ABG) Analysis

Parameter	Result	Interpretation
pH	7.21	Metabolic acidosis
HCO_3^-	12 mmol/L	Reduced
Anion Gap	12	Normal anion gap metabolic acidosis

Inference: Findings consistent with distal renal tubular acidosis (dRTA).

Table 3. Autoimmune Profile

Test Name	Result	Reference Range	Interpretation
SSA/Ro Antibody (EIA)	77.00 Units	<20.00	Positive
ANA Screen	390.0 (H) AU/mL	Negative <40.0; Positive >40.0	Strongly Positive

Method: Chemiluminescence immunoassay (ANA).

Table 4. Ophthalmologic Test

Test Name	Result	Reference Range	Interpretation
Schirmer Test	3-4mm/5 min	>10mm/5 min	Reduced tear production

DISCUSSION

Extraglandular symptoms may appear before traditional sicca symptoms in Sjögren syndrome (SS), a systemic autoimmune illness. Distal renal tubular acidosis (dRTA) is often caused by renal involvement, most commonly tubulointerstitial nephritis (Ramos-Casals et al., 2012; Evans et al., 2015). Impaired distal hydrogen ion secretion is a characteristic of dRTA, which causes hypokalemia and normal anion gap metabolic acidosis. Rarely, acute flaccid paralysis due to severe hypokalemia may be the first sign of SS (Morović-Vergles et al., 2007).

Guillain-Barré syndrome (GBS) and acute hypokalemic paralysis can appear with symmetrical weakness and areflexia. However, GBS frequently involves sensory or autonomic dysfunction with aberrant nerve conduction studies and typically follows an infectious trigger (Yuki & Hartung, 2012). Hypokalemic paralysis, on the other hand, is characterized by isolated motor weakness, maintained sensory functions, normal electrophysiology, and quick recovery following potassium supplementation.

Distal tubular acidification dysfunction is caused by autoimmune lymphocytic infiltration of the renal interstitium in the pathogenesis of dRTA in SS. In order to prevent misdiagnosis and guarantee prompt electrolyte correction and immunomodulatory therapy, early identification of this reversible metabolic cause is crucial.

Treatment and Clinical Outcome

In order to treat metabolic acidosis, the patient was given intravenous potassium chloride (10–20 mEq/hour) with cardiac monitoring and sodium bicarbonate (1-2 mEq/kg/day). Within a day, there was a noticeable recovery in motor strength, indicating that distal renal tubular acidosis was the cause of hypokalemic paralysis.

Corticosteroids and immunomodulators, dose-titrated based on patient symptoms, organs involvement and medication toxicity, as well as supportive care for sicca symptoms, were part of long-term therapy for underlying Sjögren syndrome.

CONCLUSIONS

"Paralysis before dryness" could be a sign of Sjögren syndrome. Distal renal tubular acidosis-related hypokalemic paralysis can mimic Guillain-Barré syndrome and precede sicca symptoms. For a precise diagnosis and prompt treatment, early metabolic assessment and autoimmune screening are essential.

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