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Case Report

# Recurrent Fatigue and Hypokalemia in a Patient with Sjögren's Syndrome Complicated by Renal Tubular Acidosis: A Case Report

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## Abstract

***Background:*** *This case report highlights the diagnostic challenge of unexplained fatigue and recurrent hypokalemia, emphasizing the importance of considering autoimmune etiologies, specifically Sjögren's syndrome complicated by renal tubular acidosis, in such presentations. While this association is documented, cases presenting primarily with persistent fatigue and electrolyte disturbances underscore the need for a broad differential diagnosis.*

***Case Presentation:*** *A 55-year-old female presented with a two-month history of progressive fatigue and intermittent muscle twitching. Initial evaluations revealed recurrent hypokalemia, which improved only transiently with supplementation. Comprehensive workup, including positive anti-SSA/SSB antibodies and findings suggestive of renal potassium wasting, led to the diagnosis of Sjögren's syndrome with associated renal tubular acidosis. Treatment involved potassium correction, hydroxychloroquine, and corticosteroids. At discharge and follow-up, her fatigue resolved, potassium levels normalized, and no symptom recurrence was observed.*

***Conclusions:*** *This case illustrates that Sjögren's syndrome can manifest with significant metabolic disturbances like hypokalemic renal tubular acidosis, even when classic sicca symptoms are subtle. It underscores the necessity of a thorough, interdisciplinary diagnostic approach for patients with unexplained fatigue and electrolyte imbalances to identify underlying autoimmune disorders, thereby enabling targeted therapy and improving outcomes.*

**Keywords:** Sjogren's syndrome, Hypokalemia, Fatigue, Renal tubular acidosis, Case report

## Abbreviations

RTA: Renal Tubular Acidosis
ECG: The electrocardiogram
SS: Sjögren's Syndrome

## 1. Introduction

Fatigue is a common clinical symptom. It can result from a wide range of underlying conditions, ranging from benign to life-threatening disorders [1]. It is characterized by persistent tiredness or low energy that is not relieved by rest. The differential diagnoses of fatigue are extensive, encompassing conditions such as anemia, thyroid dysfunction, chronic infections, and metabolic disorders [2]. Among these causes, hypokalemia is notable. It is defined as a serum potassium level below 3.5 mmol/L and can lead to significant muscle weakness and fatigue because potassium plays a critical role in neuromuscular function and cellular metabolism [3].

Hypokalemia can cause various symptoms, such as muscle cramps, weakness, and severe paralysis. The pathophysiology of hypokalemia often involves excessive renal excretion, insufficient dietary intake, or gastrointestinal loss [4]. In particular, renal tubular acidosis (RTA) can cause hypokalemia by impairing tubular function and reducing potassium reabsorption [5]. The association between RTA and autoimmune conditions, such as Sjögren's syndrome, is well-documented because the latter can cause renal tubular dysfunction and subsequent electrolyte imbalances [6].

This case report presents a 55-year-old female patient who experienced persistent fatigue and recurrent hypokalemia. She was ultimately diagnosed with Sjögren's syndrome complicated by renal tubular acidosis. Although this condition is relatively rare, it highlights the importance of considering autoimmune disorders in patients who present with unexplained fatigue and electrolyte disturbances. The case highlights the necessity of thorough evaluation and interdisciplinary collaboration in managing complex presentations, especially if multiple conditions coexist, such as hypertension and electrolyte imbalances.

## 2. Case Presentation

The patient is a 55-year-old woman admitted to Shenzhen People's Hospital on September 8, 2022, for persistent fatigue and recurrent hypokalemia. She had generalized fatigue for over two months, worsening in the last month. About two months before admission, the patient began to feel fatigue during activities, describing it as unexplained weakness. This fatigue was not accompanied by symptoms such as loss of appetite, dizziness, headache, fever, nasal congestion, cough, runny nose, nausea, vomiting, abdominal distension or pain, diarrhea, low back pain, or dysuria. Nevertheless, she was able to continue her daily activities and initially did not seek medical attention. One month before admission, her fatigue intensified and became persistent, accompanied by intermittent muscle twitching in her limbs, dizziness, abdominal distension, and decreased appetite. She continued to report no major symptoms, such as headaches, loss of consciousness, chest tightness, or palpitations.

On August 9, 2022, she visited the Department of Neurology at Huizhou People's Hospital. Her serum potassium level was 3.01 mmol/L. She was diagnosed with dizziness and hypokalemia. She received intravenous potassium supplementation and hydration, which provided slight symptom relief. However, her symptoms recurred, leading to her visit to our hospital on August 28, 2022. At that time, her potassium level was recorded at 3.15 mmol/L, with other biochemical markers remaining unremarkable. An electrocardiogram showed normal sinus rhythm, and a brain CT scan revealed no significant abnormalities. After outpatient potassium supplementation, her symptoms temporarily improved; however, she still experienced generalized fatigue and muscle twitching. The patient also reported depressed mood, sleep disturbances, and appetite changes. In addition, she has a history of chronic constipation requiring glycerin suppositories for the past three months. She has experienced difficulty urinating, increased nocturia, and urinary frequency, along with a significant weight loss of 1.5 kg over the past two months. Her medical history was generally unremarkable. She had no chronic illnesses or significant medication use. She underwent sterilization surgery in 2000. She also has a family history of breast cancer in her sister.

The physical examination revealed mild malnutrition, elevated blood pressure, and no significant abnormalities on neurological examination. Laboratory tests showed recurrent hypokalemia, with potassium levels ranging between 3.01 to 3.43 mmol/L. Imaging studies revealed chronic inflammation in the left upper lobe and nodules in the left lower and right middle lobes, recommending follow-up. This case highlights the complexity of diagnosing fatigue and electrolyte disturbances. It emphasizes the need for comprehensive evaluation and interdisciplinary collaboration in managing such cases.

### 2.1. Therapeutic Intervention and Outcomes

Upon admission, the patient underwent a comprehensive evaluation to determine the underlying causes of her persistent fatigue and recurrent hypokalemia. Initial laboratory tests revealed a serum potassium level of 3.01 mmol/L, consistent with the diagnosis of hypokalemia. Following this, she received intravenous potassium supplementation and hydration, which provided slight improvement in her symptoms. On August 28, 2022, the patient returned for further assessment. Her potassium level was recorded at 3.15 mmol/L, and other biochemical markers showed no significant abnormalities. The electrocardiogram (ECG) showed normal sinus rhythm, and the brain CT scan revealed no significant findings. However, despite potassium supplementation, her generalized fatigue and muscle twitching recurred.

Further investigations included a series of laboratory tests conducted on September 9, 2022. These tests revealed potassium levels of 3.43 mmol/L, sodium at 138.5 mmol/L, chloride at 101.7 mmol/L, calcium at 2.23 mmol/L, and phosphorus at 1.17 mmol/L. A urinalysis showed light yellow, clear urine with a specific gravity of 1.008 and a pH of 7.50, indicating no significant abnormalities. A 24-hour urine electrolyte test was performed, revealing potassium excretion of 84.12 mmol/24 h, which was above the normal threshold, suggesting renal loss of potassium. The aldosterone suppression test showed a plasma aldosterone level of 8.07 ng/dL and a direct renin concentration of 0.8451 mIU/L, resulting in a calculated aldosterone-to-renin ratio of 9.55. Additionally, autoimmune screening showed positive results for nuclear homogeneous type antibodies (1:100-1:320) and granular type antibodies (1:320-1:1000). The presence of anti-SSA and anti-SSB antibodies was also noted, which indicates a potential autoimmune etiology.

Overall, the investigations identified renal tubular acidosis as a significant contributor to the patient's hypokalemia. Additionally, a possible diagnosis of Sjögren's syndrome was considered and later confirmed through rheumatological evaluation. The combination of persistent fatigue, recurrent hypokalemia, and positive autoimmune markers underscore the complexity of her condition and the need for a multidisciplinary approach to her management.

Given her clinical presentation and laboratory findings, the patient was evaluated upon admission for persistent fatigue and recurrent hypokalemia. A treatment plan was established focusing on symptomatic management and treating the underlying autoimmune condition. Initially, potassium was

given intravenously to correct hypokalemia, and her serum potassium levels were closely monitored. After admission, her levels improved to 3.43 mmol/L by September 9, 2022, but ongoing observation remained essential due to her history of recurrent hypokalemia.

To investigate the underlying causes of her symptoms, we conducted several diagnostic tests, including a 24-hour urine electrolyte test and an aldosterone suppression test. The results indicated renal tubular dysfunction consistent with renal tubular acidosis caused by Sjögren's syndrome. After diagnosis confirmation, the patient was referred to rheumatology for specialized care. Treatment includes hydroxychloroquine 200 mg twice daily and corticosteroids (prednisone) 30 mg daily. This regimen aims to control her autoimmune symptoms and improve her overall condition. Throughout her hospital stay, the patient's electrolyte levels and clinical symptoms were closely monitored; adjustments to her treatment plan were made as necessary. On September 27, 2022, the patient was stable, with no recurrence of fatigue or hypokalemia. She was discharged with a continued treatment plan that included reducing corticosteroids to 5 mg daily, maintaining hydroxychloroquine, and monitoring her condition. This comprehensive approach not only addressed her immediate symptoms but also aims to improve her long-term health outcomes by targeting the underlying autoimmune disorder through effective management strategies.

After 17 days of hospitalization, the patient demonstrated significant improvement in fatigue and muscle twitching. Furthermore, her serum potassium levels stabilized, reaching 4.17 mmol/L by September 18, 2022. Additionally, no recurrence of hypokalemia was observed during her hospital stay. Upon discharge, the patient was provided with a comprehensive treatment plan that included hydroxychloroquine (200 mg twice daily) and prednisone (30 mg daily), to manage her Sjögren's syndrome and associated renal tubular acidosis. She was instructed to continue potassium supplementation if symptoms of hypokalemia occur and to monitor her electrolyte levels regularly. Follow-up appointments were scheduled to ensure ongoing evaluation of her blood pressure, renal function, and autoimmune markers. The patient was advised to return for routine check-ups every month for the first three months post-discharge, followed by visits every two months thereafter. During these follow-ups, her blood pressure, renal function, and autoimmune markers were closely monitored. At her first follow-up visit one month after discharge, the patient reported improved energy levels and no significant side effects from her medication regimen. Her blood pressure was stable, and she showed no signs of recurrent fatigue or hypokalemia. At the three-month follow-up, the patient's condition remained stable, with her symptoms well-managed under the current treatment plan. She was encouraged to maintain regular follow-ups to monitor her autoimmune condition and to promptly report any new symptoms, which contributed to the successful management of her health. Overall, the patient's treatment and follow-up strategy effectively addressed her complex health issues, including fatigue, joint pain, and inflammation, leading to an improved quality of life and stabilization of her condition (For timeline, see table 1 and figure 1).

### 2.2. Timeline

| Date | Event/Action |
|---|---|
| July 2022 | Patient began experiencing generalized fatigue and unexplained weakness during activities. |
| August 9, 2022 | Visited Department of Neurology at Huizhou People's Hospital; potassium level recorded at 3.01 mmol/L; diagnosed with dizziness and hypokalemia; received intravenous potassium supplementation and hydration. |
| August 28, 2022 | Returned to hospital with potassium level recorded at 3.15 mmol/L; other biochemical markers unremarkable; ECG showed normal sinus rhythm; brain CT scan revealed no significant abnormalities. |
| September 9, 2022 | Comprehensive laboratory tests conducted; potassium level at 3.43 mmol/L; sodium at 138.5 mmol/L; chloride at 101.7 mmol/L; calcium at 2.23 mmol/L; phosphorus at 1.17 mmol/L; urinalysis showed no significant abnormalities; 24-hour urine electrolyte test revealed potassium excretion of 84.12 mmol/24 h; aldosterone suppression test results indicated a potential autoimmune etiology. |
| September 18, 2022 | After 17 days of hospitalization, patient's potassium levels stabilized at 4.17 mmol/L; significant improvement in fatigue and muscle twitching; no recurrence of hypokalemia observed. |
| September 27, 2022 | Patient discharged with a treatment plan including hydroxychloroquine (200 mg twice daily) and prednisone (30 mg daily) for Sjögren's syndrome and renal tubular acidosis; instructed to continue potassium supplementation as needed. |
| October 2022 | First follow-up appointment; patient reported improved energy levels and no significant side effects; blood pressure stable; no signs of recurrent fatigue or hypokalemia. |
| December 2022 | Three-month follow-up appointment; patient's condition remained stable; symptoms well-managed under current treatment plan; encouraged to maintain regular follow-ups and report any new symptoms |

**Table 1: Timeline of Clinical Events and Management for a Patient with Sjögren's Syndrome and Renal Tubular Acidosis**

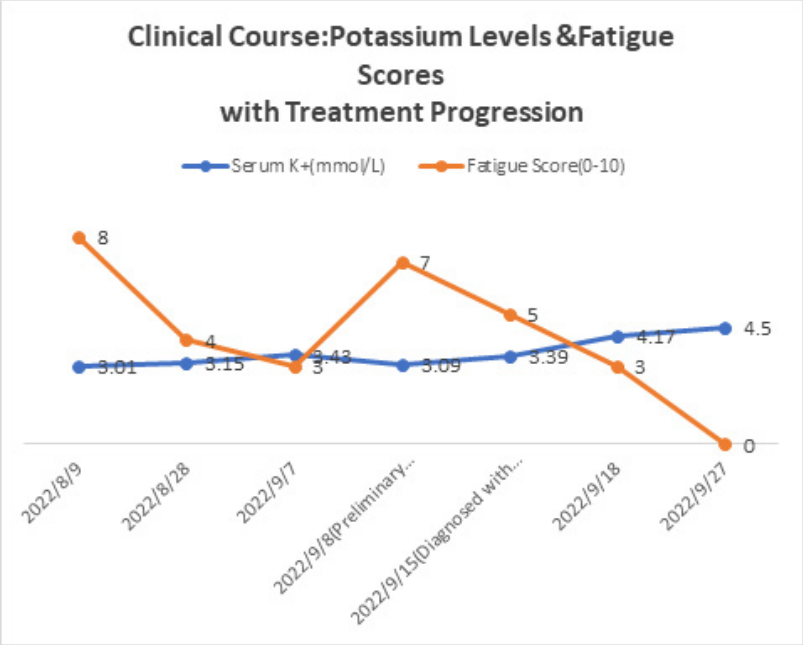


**Figure 1: Case report timeline: patient's diagnosis, treatment and symptom improvement process**

## 3. Discussion

The case of a 55-year-old female patient presenting with persistent fatigue and recurrent hypokalemia is clinically significant due to the complexity of her condition and the interplay of multiple contributing factors. This case underscores the necessity for thorough evaluation in patients exhibiting non-specific symptoms such as fatigue, particularly when electrolyte disturbances are involved. The potential for underlying autoimmune disorders to contribute to these symptoms cannot be overlooked.

Existing literature indicates that fatigue is a common complaint, stemming from various underlying conditions, including metabolic, endocrine, and autoimmune disorders [7,8]. Notably, fatigue in patients with autoimmune conditions like Sjögren's syndrome can often be attributed to systemic inflammation and the impact of the disease on overall health. The relationship between hypokalemia and symptoms such as muscle weakness and fatigue are well documented, as low potassium levels can disrupt neuromuscular function, leading to significant clinical manifestations, including paralysis and arrhythmias [9,10]. In this case, the patient's recurrent episodes of mild hypokalemia, with potassium levels ranging from 3.01 mmol/L to 3.43 mmol/L, suggested an underlying renal tubular dysfunction, particularly given her persistent muscle weakness and fatigue despite potassium supplementation [5].

The diagnostic process for this patient was particularly complex due to the presence of multiple comorbidities, including suspected primary hypertension and possible autoimmune disease, as suggested by her dry eye symptoms and positive autoimmune markers. This complexity highlights the importance of a comprehensive diagnostic approach, which may include not only standard biochemical tests but also more specialized assessments such as renal function tests and autoimmune panels. Differentiating between various causes of hypokalemia, such as renal potassium loss versus dietary potassium deficiency, proved challenging [11]. This was addressed through comprehensive laboratory evaluations, including 24-hour urine electrolyte measurements, which revealed elevated potassium excretion and supported the hypothesis of a renal potassium loss etiology [12].

Ultimately, a diagnosis of Sjögren's syndrome with associated renal tubular acidosis was established. This diagnosis highlights a rare yet clinically significant connection between autoimmune conditions and electrolyte imbalances, which, while infrequent, has important implications for patient care [13,14]. The novelty of this case lies in the recognition of how Sjögren's syndrome can manifest not only through typical symptoms but also through significant metabolic disturbances, thus emphasizing the need for clinicians to maintain a broad differential when evaluating patients with unexplained fatigue and electrolyte disturbances [15,16]. Moreover, this case illustrates the importance of interdisciplinary collaboration in diagnosing and managing complex cases; the involvement of rheumatology was crucial in confirming the diagnosis and guiding treatment [17-19].

The treatment plan for this patient involved symptomatic management of hypokalemia while specifically addressing Sjögren's syndrome. The administration of hydroxychloroquine and corticosteroids aligns with current therapeutic strategies for managing Sjögren's syndrome and its renal manifestations [20-23]. Additionally, continuous monitoring and adjustment of treatment based on the patient's response and electrolyte levels are essential to prevent recurrence of symptoms and improve overall quality of life [24-26].

## 4. Conclusions

In conclusion, this case reminds us that fatigue is a complex and multifaceted clinical symptom. It also highlights the need for a comprehensive approach to diagnosis and management. It underscores the importance of clinicians remaining vigilant for underlying autoimmune processes in patients presenting with electrolyte imbalances, as early recognition and intervention can significantly improve clinical outcomes. Future clinical practice should integrate these insights and advocate thorough evaluations in similar cases to optimize patient care and ensure effective resource utilization.

## Declarations

**Ethics approval and consent to participate:** Ethical approval for this case report was obtained from the Ethics Committee of Shenzhen People's Hospital. All procedures were performed in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from the patient for participation in the study.

**Consent for publication:** Written informed consent was obtained from the patient for publication of anonymized case details and associated images within the manuscript.

**Availability of data and material:** The datasets generated during and/or analysed during the current study are not publicly available due to privacy and ethical restrictions but are available from the corresponding author on reasonable request.

**Competing interests:** The authors declare that they have no competing interests.

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**Authors' contributions:** ZY and XC contributed to study concept and design, performed the data analyses, and wrote the manuscript. CC and JH contributed to study supervision. All authors (ZY, XC, CC, JH, FL, SL, MX, XC, QL and YJ) have full access to all data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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