Original Research Paper

Volume - 14 | Issue - 08 | August - 2024 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

General Medicine

PRIMARY SJOGREN'S SYNDROME UNVEILED BY HYPOKALEMIC PARALYSIS: A CASE REPORT

Dr. G. Srivani	MD, Asst. professor, Dept of General Medicine, Narayana Medical College.
Dr. O. Sai Lahari	MD, Asst. professor, Dept of General Medicine, Narayana Medical College.
Dr. P. Nagarjuna	Postgraduate, Dept of General Medicine, Narayana Medical College.

ABSTRACT Sjogren's syndrome is a rare, chronic autoimmune disorder primarily marked by dry eyes and dry mouth due to the immune system targeting the lacrimal and salivary glands. It often coexists with other autoimmune conditions such as rheumatoid arthritis, systemic lupus erythematosus, or thyroiditis. The syndrome can also lead to renal issues, including interstitial nephritis and glomerulonephritis, along with distal or type 1 renal tubular acidosis, which can cause serious electrolyte imbalances Renal involvement is a well-recognized extra-glandular manifestation of primary SS, occurring in 16%–67% of cases, with distal renal tubular acidosis (RTA) being common, reported in 4.3%–9% of PSS patients.4. Here, we report the case of a 30-year-old woman with quadriparesis. Her symptoms were traced back to type 1 renal tubular acidosis, which subsequently led to a diagnosis of Sjogren's syndrome. This case highlights a rare and challenging presentation of Sjogren's syndrome. The difficulty in early detection and diagnosis is compounded by the current diagnostic criteria, often resulting in missed or delayed diagnoses.

KEYWORDS : Sjogren's syndrome, hypokalemia, potassium, quadriparesis, renal tubular acidosis

INTRODUCTION

Sjogren's syndrome (SS) is a rare autoimmune condition typically characterized by chronic inflammation of exocrine glands such as the lacrimal and salivary, resulting in symptoms like dryness of eyes and mouth. [1]

Renal tubular acidosis (RTA) is characterized by renal tubular impairment in maintaining physiological acid–base balance. It often results from defects in tubular transporters that participate in the secretion or uptake of specific ions. RTA can be caused by congenital factors, exposure to nephrotoxic drugs, diuretic abuse, autoimmune diseases, or malignancies. There are three major types of RTA: distal or type 1, proximal or type 2, and hyperaemic or type 4. All three types of RTA are characterized by a positive urine anion gap, hyperchloremic non anion gap metabolic acidosis, alkalotic or acidotic urine pH, and derangements in serum potassium levels (hypo- or hyperkalaemia)..

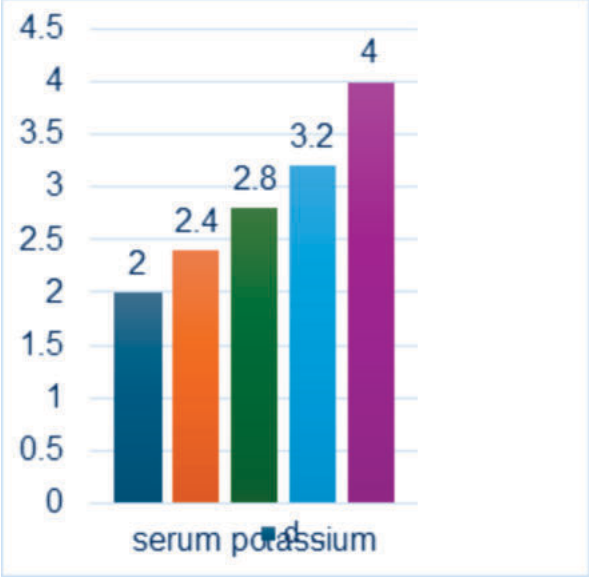
We herein reported a case of distal RTA secondary to SS that led to hypokalaemia periodic muscular weakness. [2] [3].This case report will enhance our understanding of renal involvement in patients with SS. Early identification and initiation of treatment for SS are crucial to reducing the risk of severe and potentially fatal complications. Additionally, clinicians should be aware of the potential renal involvements in these patients to provide timely and effective treatment and will provide evidence of the occurrence of renal tubular acidosis in SS patients.

CASE REPORT

A 30years old female presented to emergency department for the first time with complaints of sudden onset of weakness of both upper and lower limbs for one day. The weakness was painless and was not associated with a loss of consciousness. She denies any rash, joint pain, alopecia, dryness of eyes or mouth, renal stones, fractures or gastrointestinal problems. She had regular menstrual cycles, and two pregnancies were uneventful. There was no history of steroid use or laxative abuse. The weakness was prominent in both the proximal and distal extremities. Family history was nonsignificant for similar conditions. The patient was vitally stable and had a Glasgow coma score of 15/15. Thyroid, parotid examination was normal. She had no muscle tenderness, reflexes were 1/2, power was 1/5, the tone was 1/3 in both upper and lower limbs, with no cranial nerves deficit, sensations were intact, and plantar reflexes were mute. Schirmer test was normal suggesting normal tear secretion.

LAB INVESTIGATIONS

Laboratory investigations revealed hypokalaemia. Serum potassium of 2.0meq/l, magnesium of 2.05meq/l calcium of 8.27mg/dl ,cortisol 33.2ug/dl, renal ,thyroid ,viral screening were normal. Arterial blood gases of the patient were sent and showed normal anion gap metabolic acidosis with a pH of 7.11, bicarbonate of 10.5 mEq, and partial pressure of carbon dioxide (pCO2) of 33 mm Hg,pO2 of 75.All the other causes of hypokalaemia with normal anion gap metabolic acidosis were ruled out by the patient history and laboratory investigations. Her serum potassium values were monitored (figure 1) A suspicious distal RTA that leads to hypokalaemia was made, which was confirmed by urinary tests as shown below Urine examination: pH6.5, sodium 133meq/l, spot potassium19.7meq/l, chloride 116, anion gap 36.7meq/l Positive Usg abdomen was unremarkable with no evidence of obstructive uropathy.



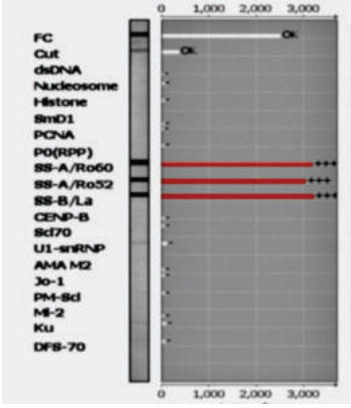


Figure1

DISCUSSION

Extra-glandular involvement in SS also occurs and includes glomerulonephritis, tubulointerstitial nephritis, arthritis, arthralgia, vasculitis, pulmonary diseases, and lymphomas [4]. The condition can affect an individual of any age but is more common in elderly population. Renal, pulmonary, and haematological involvements are more common among the Asian population. Renal involvement is a well-recognized extra-glandular manifestation of primary SS, occurring in 16%–67% of cases, with distal renal tubular acidosis (RTA) being common, reported in 4.3%–9% of PSS patients

CONCLUSION

Distal RTA as a cause for hypokalaemia is the gateway to the diagnosis of Sjogren's syndrome because distal RTA is frequently associated with autoimmune disorders such as Sjogren's.

REFERENCES:

1. Ramos-Casals, M. (2004). Primary Sjogren's syndrome: New clinical and therapeutic concepts. *Annals of the Rheumatic Diseases*, *64*(3), 347–354. https://doi.org/10.1136/ard.2004.025676
2. Khanal, P., Paudel, S., Chapagain, S., Thapa, S., & Gyawali, M. (2023). Hypokalemic paralysis revealing primary Sjogren's Syndrome: A case report. Journal of Nepal Medical Association, 61(265), 735–737. https://doi.org/10.31729/jnma.8266
3. Hamada, S., Takata, T., Yamada, K., Yamamoto, M., Mae, Y., Iyama, T., Sugihara, T., Takata, M., & Isomoto, H. (2023). Renal tubular acidosis without interstitial nephritis in Sjögren's syndrome: A case report and review of the literature. *BMC Nephrology*, *24*(1). https://doi.org/10.1186/s12882-023-03290-3
4. Evans, R., Zdebik, A., Ciurtin, C., & Walsh, S. B. (2015). Renal involvement in primary Sjögren's syndrome. Rheumatology, 54(9), 1541–1548. https://doi.org/10.1093/rheumatology/kev223.
5. Harrisons principle of internal medicine 21st edition.