VOLUME - 14, ISSUE - 05, MAY - 2025 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

SJOGREN'S SYNDROME PRESENTING AS HYPOKALEMIC PARALYSIS SECONDARY TO RENAL TUBULAR ACIDOSIS - A CASE REPORT

Dr Latchumana Kumaran M — Post Graduate, Department Of General Medicine, Aarupadai Veedu Medical College, Vinayaga Mission's Research Foundation (VMRF-DU), Puducherry, India

Dr PM Karthik — Assistant Professor , Department Of General Medicine , Aarupadai Veedu Medical College, Vinayaga Mission's Research Foundation (VMRF-DU), Puducherry, India

ABSTRACT A chronic autoimmune disease that mainly affects exocrine glands, Sjogren's syndrome can also cause extraglandular complications. We describe hypokalemic paralysis brought on by distal renal tubular acidosis (dRTA), a rare manifestation of Sjogren's syndrome. A 41-year-old woman had severe hypokalemia and weakness that started suddenly. The diagnosis of Sjogren's syndrome with dRTA was confirmed by additional testing, which showed non-anion gap metabolic acidosis and positive serology for anti-SSA/Ro antibodies. Significant clinical improvement resulted from prompt electrolyte imbalance correction and immunomodulatory therapy initiation. This case emphasizes how crucial it is to take into account autoimmune causes when dealing with unexplained hypokalemic paralysis.

KEYWORDS :

INTRODUCTION:

Sjögren's syndrome is a chronic autoimmune disease that primarily involves exocrine glands, and leads to xerostomia and keratoconjunctivitis sicca. The pathological process includes lymphocyte infiltration within the exocrine tissues such as salivary and lacrimal glands. In addition to exocrine involvement, Sjögren's syndrome can have significant systemic features including skin, joints, lungs, liver and kidneys. Kidney involvement is an established but often underappreciated feature of Sjögren's syndrome with varying presentations from renal tubular dysfunction and asymptomatic to overt renal tubular acidosis (RTA) and interstitial nephritis. Out of these, distal (type 1) RTA is the hallmark presentation encountered most frequently. These cases also tend to have mild hypokalemia. However, the severity of hypokalemia that can lead to clinical complications such as hypokalemic paralysis is unusual and rarely documented. Finding this clinical picture early on is paramount in preventing delayed treatment and misdiagnosis.

Case Presentation: We describe a 41 year old female who presented with acute onset of generalized weakness that was described as quadriparesis. The weakness came on suddenly and was bilateral without fever, trauma, or significant exercise.

The patient displayed an extreme muscular deficiency that inhibited him from being able to stand or walk, yet he was still able to perceive sensation. Neither bowel nor bladder functions were impaired. Aside from lack of intake which resulted in dry mouth and dry eyes, which was previously disregarded, there was no remarkable past medical history.

After examining the patient, it was noticed that he was wide awake and looking around. However, he had detected very severe muscular consequences in all four of his limbs. After testing his muscle power, he was able to achieve 2/5 in both his upper and lower limbs. No signs of cranial nerve infiltration were present, yet deep tendon flexes were limited. Based on the effect of acute quadriplegia, a cyberspace paralysis or a physical cause was presumed for the time being. Blood tests were performed, including a CT scan which yielded no concerning results. This was followed by serum electrolyte analysis which showed hypokalemia.

With the initiation of muscular paralysis diagnosis, he was placed on intravenous potassium therapy. Post removal of potassium hope from muscles, patients are often able to foster attaining the ability to gain muscle eighty percent whilst full flexibility within the next forty-eight hours. This symptom collectively diagnosed is known as hypokalemic paralysis. At risk of diving into ramification of severe muscular flimsy will enable flexed posture, complemented with limited food intake and no visible signs of waterfall will enable sight. This will deepen the examination in hopes of finding the reason behind shallow muscle expenditure.

An investigation of urine showed an anion gap that was raised and a positive urinary anion gap as well which suggests excretion of hydrogen ions is impaired, aligning with distal type 1 renal tubular acidosis.

Taking into account that the patient has keratoconjunctivitis and Sjogren's syndrome, there is an autoimmune origin. An ophthalmological examination was performed and results from Schirmer's test showed the production of tears from every eye was significantly decreased, measuring just 1 mm which aligns with keratoconjunctivitis sicca. Further serological tests were done alongside autoimmune markers. The level of Anti Ro SSA was oustandingly high while anti La SSB was low, although still high. In relation to the rest of the body alongside supporting aids, these results confirm primary Sjögren's syndrome and renal involvement with distal renal tubular acidosis.

The model of conservative management was adopted for this patient. Bicarbonate therapy was initiated in a bid to rectify the acidosis, and potassium supplementation was continued orally. Dry eyes were treated with artificial tears to dull the symptoms. This led to myopathy relieving most of his symptoms meaning he remained asymptomatic and improving alongside stronger feeling. By the end of his treatment period my potassium levels stabled and my compliance to the medications meant I had no symptoms.

DISCUSSION:

Schjogren is a paired syndrome that heavily effects middle aged women for autoimmune diseases.

The diagnostic criteria integrates a mix of subjective symptoms (like dry eyes and dry mouth), objective evaluation (such as Schirmer's test), histopathological findings (lymphocytic infiltration of minor salivary glands), and

serological indicators (anti-Ro/SSA and anti-La/SSB antibodies). Glandular manifestations are hallmark features of the disease, and while systemic involvement may be significant in its own right, contribute to both morbidity and complexity in diagnosis.

A variable proportion of patients with Sjögren's syndrome exhibit renal involvement, with studies estimating prevalence between 2% to 67%, depending on the population and diagnostic methods used. Chronic interstitial nephritis, resulting in renal tubular dysfunction like distal RTA, is the most common renal manifestation. In distal (type 1) RTA, the distal nephron's capability to secrete hydrogen ions diminishes, resulting in non-anion gap metabolic acidosis with hypokalemia and inappropriately elevated alkaline urine pH. Histological studies suggest the pathogenesis involves immune mediated injury of the renal tubular cells, driven by lymphocytic and plasma cell infiltration in the renal interstitium.

Mild to moderate is typically the case in RTA distal hypokalemia. There is however life threatening potential in severe cases, clinically manifesting as arrhythmias, muscle cramping, and in rarer case, hypokalemic paralysis.

Hypokalemic paralysis is an uncommon emergency that typically occurs due to additional factors within the body. In this case, it is triggered by low potassium levels in the body and manifests with instant flaccid paralysis, also known as acute paralysis. Early recognition of Sjögren's syndrome is critical, as it could represent a secondary symptom. Diagnosis may be overlooked and lead to frequent complications.

In the presented case, preliminary diagnosis of Sjögren's syndrome was made based on cross-sectional clinical data under ophthalmology, along with blood tests for antibodies. Decreased renal function and the case of dystrophic paralysis provided critical information that guided further diagnosis. Treatment focuses on correcting electrolyte imbalances and the underlying autoimmune condition. In the absence of profound systemic effects, conservative treatment with supplementation and management of dry eye may be adequate. Advanced cases may require immunosuppressants.

This example is crucial for the understanding autoimmune disorders when accompanied with unexplainable electrolyte imbalance and neuromuscular conditions. Autoimmunity adds to the already existing dry symptoms while creating other unexplained conditions. We also want to emphasize the unique manifestations of Sjögren's syndrome that require deeper investigation in non-typical cases.

Outcomes can be ideal with rapid diagnosis and prompt treatment, and this patient's successful outcome reasserts that claim.

CONCLUSION:

Sjögren's syndrome primarily affects the exocrine glands, but expanded observation shows renal tubular atrophy as a secondary effect, confirming the condition's broad underlying syndrome.

The condition is well known as a renal manifestation of Sjögren's syndrome, however, having distal renal tubular acidosis with severe hypokalemia and paralysis is not frequently encountered. This illustrative case reinforces the fact that Sjögren's syndrome should be actively sought in those who have unexplained RTA with hypokalemic paralysis along with prominent dry eyes or mouth. Precision diagnosis and thorough care tend to require renal, rheumatology, and ophthalmology specialists working as multidisciplinary one team. Timely diagnosis and appropriate management of the electrolyte disorder together with the underlying autoimmune disorder are critical for avoiding persistent problems, optimizing, and improving patient care.

REFERENCES:

1. Sjögren's syndrome, Mavragani CP, Moutsopoulos HM. 2014; 9:273-285; Annu Rev Pathol. 10.1146/annurev-pathol-012513-104621 is the doi.
2. Maripuri S, Hogan MC, Fervenza FC, Grande JP, Osborn TG, and Matteson EL. A clinicopathologic investigation of renal involvement in primary Sjögren's syndrome. 2009;4(9):1423–1431; doi:10.2215/CJN.01710309; Clin J Am Soc Nephrol.
3. Yaqoob MM, Emtage J, Saif I, Nair H, and Ting SM. severe hypokalaemia and distal renal tubular acidosis as a consequence of primary Sjögren's syndrome. 2006;43(Pt 4):360-363; Ann Clin Biochem. 10.1258/000456306777698256 is the doi.
4. Tzioufas AG, Voulgarelis M. pathogenetic processes in the development and maintenance of Sjögren's syndrome. 2010;6(9):529-537; doi:10.1038/nrrheum.2010.116; Nat Rev Rheumatol.
5. Distal renal tubular acidosis, Miller JA, Sebastian A. In: Schrier RW, ed. Kidney and Urinary Tract Diseases, 8th ed., Philadelphia: Lippincott Williams & Wilkins, 2007.6. Fox RI. Sjögren's syndrome.