Hindawi
Case Reports in Rheumatology
Volume 2017, Article ID 7509238, 7 pages
https://doi.org/10.1155/2017/7509238

Case Report

Hypokalemic Paralysis due to Primary Sjögren Syndrome: Case Report and Review of the Literature

A. Garza-Alpirez, A. C. Arana-Guajardo, J. A. Esquivel-Valerio, M. A. Villarreal-Alarcón, and D. A. Galarza-Delgado

Servicio de Reumatología, Departamento de Medicina Interna, Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Monterrey, NL, Mexico

Correspondence should be addressed to A. C. Arana-Guajardo; ana.aranag@gmail.com

Received 29 March 2017; Revised 14 June 2017; Accepted 2 July 2017; Published 1 August 2017

Academic Editor: Syuichi Koarada

Tubulointerstitial nephritis (TIN) is the main renal involvement associated with primary Sjögren syndrome (pSS). TIN can manifest as distal renal tubular acidosis (RTA), nephrogenic diabetes insipidus, proximal tubular dysfunction, and others. We present a 31-year-old female with hypokalemic paralysis due to distal RTA (dRTA). She received symptomatic treatment and hydroxychloroquine with a good response. There is insufficient information on whether to perform a kidney biopsy in these patients or not. The evidence suggests that there is an inflammatory background and therefore a potential serious affection to these patients, such as hypokalemic paralysis. We found 52 cases of hypokalemic paralysis due to dRTA in pSS patients. The majority of those patients were treated only with symptomatic medication. Patients who received corticosteroids had stable evolution even though they did not have another symptomatology. With such heterogeneous information, prospective studies are needed to assess the value of adding corticosteroids as a standardized treatment of this manifestation.

1. Introduction

Sjögren's syndrome is an autoimmune disease with glandular (salivary and lacrimal) and extraglandular (neurologic, renal, hepatic, respiratory, vascular, and cutaneous) manifestations. Tubulointerstitial nephritis (TIN) is the main renal involvement associated with primary Sjögren syndrome (pSS). TIN can manifest as distal renal tubular acidosis (RTA), nephrogenic diabetes insipidus, proximal tubular dysfunction, and others [1], of which RTA is the main clinical presentation [2]. RTA has been reported in 4.3 to 9% of pSS patients; it is more common in middle-aged women, and two-thirds of them will develop symptoms [2, 3]. Hypokalemic paralysis is the initial symptom in seven percent of patients with Sjögren's syndrome [4]. We present a case of paralysis due to RTA in a pSS patient and also discuss the treatment in these patients.

2. Case Report

A 31-year-old female presented to the emergency room due to a 3-day history of progressive weakness and pain of the upper and lower extremities until walking was impossible. Two days before admission, cramps and generalized dysesthesias were evidenced. On admission, the patient presented mild dyspnea. Her past medical record was significant for polyarthralgias in carpal, metacarpophalangeal, and proximal interphalangeal joints and dry mouth for the past three months. She denied use of alcohol, illicit drugs, or herbal medicines. Her vital signs on admission were a temperature of 36.3°C, a heart rate of 54 beats per minute, a respiratory rate of 20 breaths per minute, oxygen saturation of 97% at room air, capillary blood glucose of 103 g/dL, and blood pressure of 100/60 mmHg. On physical examination, the deep tendon reflexes were globally diminished, her muscle strength, both proximal and distal, was 3/5 on Lovett's scale, and her tongue was dry and the infralingual salivary pooling was absent. Remarkable laboratory tests are shown in Table 1. A panoramic photo of minor salivary gland biopsy is shown in Figure 1. With all lab results, a distal RTA (dRTA) diagnosis due to pSS was made. Hypokalemia and metabolic acidosis were treated with intravenous potassium chloride and

Table 1: Laboratory investigation.

Laboratory investigation	Result
CBC	Hemoglobin: 14.7 g/dL, WBC: 8.7×10^3, lymphocytes: 0.683×10^3, platelets: 159 K/μL
Serum electrolytes	Sodium: 138.2 mmol/L, potassium: 2.7 mmol/L, chloride: 101 mmol/L
Serum chemistry	Glucose: 123 mg/dL, creatinine: 0.8 mg/dL, urea nitrogen: 13 mg/dL
Liver panel	AST: 19 IU/L, ALT: 13 IU/L, albumin: 4.2 g/dL, total bilirubin: 0.7 mg/dL
Urinalysis	pH: 8, leucocytes: 0–2/HPF, erythrocytes: 0/HPF, tubular cells: 0/HPF
Urinary electrolytes	Sodium: 114 mmol/L, potassium: 32 mmol/L, chloride: 57.3 mmol/L, creatinine: 31.8 mg/dL
Urinary anion gap	76 mmol/L
Blood gas	pH: 7.12, HCO_3: 11 mmol/L, pO_2: 31 mmHg, pCO_2: 37 mmHg, saturation: 37%
Serum anion gap	10 mEq/L
Thyroid panel	TSH: 2.06 μIU/mL, free T4: 0.94 ng/dL
Acute phase reactants	ESR: 31 mm/h, CRP < 0.5 mg/L
Virus panel	HIV-negative, HBV-negative, HCV-negative
Rheumatoid factor	IgM: 155.7 IU/mL, IgG: 6.7 IU/mL, IgA: 12.2 IU/mL
ANAs by IFA	1 : 5120 fine speckled
SSA/SSB by ELISA	200.14/19.67 IU/mL
Unstimulated whole saliva flow, without anesthesia	1.4 mL/15 minutes
Minor salivary gland biopsy*	Positive, focus score of 5
Schirmer's test	Right eye: 7 mm, left eye: 10 mm

*According to [5].

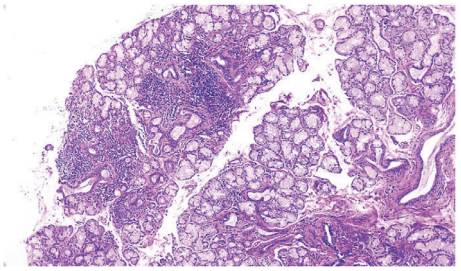

Figure 1: A panoramic photo of minor salivary gland biopsy. A chronic lymphocyte focal sialadenitis was observed.

sodium bicarbonate. Then, we initiated hydroxychloroquine. The patient was discharged and we followed her up in our clinic every two months for the next eight months. She was reported to be asymptomatic with the use of potassium citrate only.

3. Discussion

A recent set of classification criteria for pSS were published by the ACR/EULAR in 2016 [6] and this applies to the individual that has a score of ≥4. According to this, the diagnosis of this autoimmune disease was made in our patient (labial salivary gland with a focus score of ≥1, anti-SSA positive, and an unstimulated whole saliva flow of less than 0.1 mL/min). Renal involvement in pSS is the result of two distinct pathophysiological processes: TIN and glomerulopathy [1]. The tubulointerstitial inflammation is the most common renal lesion described by Talal et al. [7]. dRTA prevalence fluctuates between 5 and 70%, according to population studies [4, 8, 9]. dRTA can be classified as complete or incomplete; the former is characterized by metabolic acidosis with morning urine pH > 5.5 and a positive urinary anion gap. The incomplete form presents with normal serum bicarbonate levels but urinary pH fails to fall to <5.3 after ammonium chloride loading [10]. The pathogenic mechanism of this complication is not completely understood. Antibodies to vacuolar H+-ATPase and anion exchanger 1, as well as antibodies to carbonic anhydrase II, have been implicated in the pathogenesis [11–13]. Another hypothesis is a defective S-phase-kinase-associated protein-1, a component of the regulator of the ATPase of vacuolar and endosomal membranes that could induce a defective V-ATPase assembly [14]. Also, a possible relation between antibodies anti-SSA/Ro and dRTA has been described as one pathogenic mechanism of development [15].

Hypokalemia is the most common electrolyte abnormality in patients with dRTA. The causes of hypokalemia include decreased distal tubular Na delivery, secondary hyperaldosteronism, defective H-KATPase, and bicarbonaturia [16]. Hypokalemic paralysis may precede sicca syndrome from three months to four years in patients with a final diagnosis of pSS [17, 18].

Renal biopsy is not mandatory in these patients [2], but it may help us evidence the inflammatory mechanisms that trigger the disease. As has been demonstrated by Evans et al. in twelve patients with TIN secondary to pSS, they observed CD4+ T-cell predominance in biopsies, similar to those seen

Table 2: Comparative treatment in hypokalemic paralysis due to distal RTA in Sjögren syndrome patients.

Reference	Type of study	Number of patients	Age (years) mean	Extraglandular manifestations besides dRTA	Treatment	Follow-up	Outcome
Goroshi et al.	Case series	13	33.1	Arthritis, arthralgias, vasculitis	Symptomatic Extraglandular: HCQ and MTX	2.8 years (0.5–4)	No improvement in reduction of HCO_3 or K requirements
Khadgawat et al.	Report of cases	2	20.5	No	Symptomatic	Not reported	—
Soy et al.	Case report	1	39	Arthralgia, myalgia, nephrolithiasis	Symptomatic and methylprednisolone	2 years	Stable clinical evolution
Cheng et al.	Report of cases	2	76	Nephrocalcinosis	Symptomatic and prednisolone	5–12 months	Stable clinical evolution
Kawashima et al.	Case report	1	39	Osteomalacia, interstitial nephritis	Symptomatic and prednisolone	2 years	Relapse after stopping treatment
Comer et al.	Case report	1	43	No	Symptomatic	2 years	Stable clinical evolution
Seirafian et al.	Case report	1	64	No	Symptomatic, prednisolone, and HCQ	Not reported	—
Vaidya and Ganeshpure	Case report	1	23	No	Symptomatic	1.5 years	Stable clinical evolution
Sarah et al.	Report of cases	2	35	No	Symptomatic	Not reported	—
Rao et al.	Report of cases	3	37	Not reported	Symptomatic	Not reported	—
Nail et al.	Case report	1	65	No	Symptomatic	Not reported	—
Rajagopala et al.	Case report	1	36	Medullary nephrocalcinosis, recurrent CNS demyelination, neuromyelitis optica Secondary APS with thrombosis	Symptomatic, methylprednisolone, prednisolone, CYC, and AZA	3 months	Stable clinical evolution
Palkar et al.	Case report	1	58	Low-grade fever	Symptomatic	Not reported	Stable clinical evolution
Dasari et al.	Case report	1	40	No	Symptomatic and prednisolone	6 months	Stable clinical evolution
Singhvi et al.	Case report	1	30	No	Symptomatic and prednisolone	6 months	Stable clinical evolution
Chang et al.	Report of cases	2	10	One patient: carotid artery stenosis	Symptomatic	3–6 years	One patient: four relapses
Eriksson et al.	Report of cases	6	64.6	Not reported	No reported	Not reported	—
Taylor and Parsons	Case report	1	55	No	Symptomatic	Not reported	—
Carminati et al.	Case report	1	32	No	Symptomatic	Not reported	—

Table 2: Continued.

Reference	Type of study	Number of patients	Age (years) mean	Extraglandular manifestations besides dRTA	Treatment	Follow-up	Outcome
Muthukrishnan et al.	Case report	1	39	No	Symptomatic and prednisolone	2 years	Stable clinical evolution
Prakash et al.	Case report	1	49	No	Symptomatic, methylprednisolone, prednisolone	16 days	Died
Skalova et al.	Case report	1	16	No	Symptomatic, methylprednisolone, CYL	Not reported	Stable clinical evolution
Liao et al.	Case report	1	49	Not reported	Symptomatic	Not reported	—
Sengul et al.	Case report	1	48	No	Symptomatic, prednisolone, HCQ	Not reported	—
Yilmaz et al.	Case report	1	53	No	Symptomatic, methylprednisolone, HCQ, AZA	10 days	Stable clinical evolution
Logan and Ahmed	Case report	1	36	No	Symptomatic, HCQ	3 years	Stable clinical evolution
Fujimoto et al.	Case report	1	27	Kidney lithiasis	Symptomatic	4 months	Stable clinical evolution
Mugundhan et al.	Case report	1	38	Nephrocalcinosis	Symptomatic and prednisolone	Not reported	—
Garza-Alpirez et al.	Case report	1	31	Polyarthralgias	Symptomatic, HCQ	8 months	Stable clinical evolution

Symptomatic: potassium (K) and bicarbonate (HCO_3); HCQ: hydroxychloroquine; MTX: methotrexate; CYC: cyclophosphamide; AZA: azathioprine; MM: mycophenolate mofetil; CYL: cyclosporine A; APS: antiphospholipid syndrome; extraglandular manifestation: arthritis, arthralgia, and vasculitis.

in lip salivary glands [19]. Also, similar lymphocytic infiltrates around renal tubules have been observed [20]. More data from prospective studies of pSS biopsies are needed in order to enhance knowledge in these subsets of patients and also to determine the best treatment.

dRTA treatment includes potassium restitution before alkali therapy, because the last might aggravate hypokalemia by enhancing the shift of potassium into cells and bicarbonaturia [21]. In the beginning, hydroxychloroquine was started in the suspicion of a secondary cause of Sjögren's syndrome but it was later discontinued.

RTA is not a usual indication for immunomodulatory therapy in pSS, even though it is an extraglandular manifestation [22]. Steroid therapy in cases that are nonresponsive to replacement therapy and in those with recurring hypokalemic paralysis attacks is indicated [23].

We searched in MEDLINE, IMBIOMED, and Google Scholar for clinical cases of hypokalemic paralysis due to pSS. We included only articles written in English or Spanish. In Table 2, we describe each one of them: number of cases, age of patients, extraglandular manifestations besides dRTA, treatment, and outcome. We found fifty-two cases for analysis but we included only cases with a complete report of treatment [15, 21–47]. We observed the highest frequency of this clinical presentation in young adults of the female gender. It is important to note that, in some cases, dRTA was present before the diagnosis of pSS. All patients received symptomatic treatment. We noted that 25% (13/52) received corticosteroids. Of these patients, 61% (8/13) did not report extraglandular manifestations, besides dRTA. The outcomes (at different duration) were clinically stable in 61% (8/13), 8% (1/13) had a relapse after treatment was stopped, 8% (1/13) died from an infectious cause, and 23% (3/13) did not report the outcome.

On the other hand, 32.6% (17/52) received only symptomatic treatment. Of these patients, 41% (7/17) did not report extraglandular manifestations. Only in six (35%) patients was the outcome reported, of whom 83% (5/6) were clinically stable, and in 17% (1/6) four relapses occurred.

In the early diagnosis era of autoimmune diseases (like in rheumatoid arthritis), the importance of recognizing kidney involvement before glandular symptoms appear has been observed previously [21, 25, 28]. Also, we consider it important to determine whether some factors can trigger the beginning of this manifestation. This association has been observed by Logan and Ahmed. They described in a patient the use of *Echinacea* as a trigger of pSS [45]. Perhaps this means that the immunological tolerance is already lost, and some infections or substances can precipitate the clinical disease. We agree with the recommendation given by François and Mariette to screen all pSS patients according to manifestations every six to twelve months [10].

With such heterogeneous information, prospective studies are needed to assess the value of adding corticosteroids as a standardized treatment of this manifestation. We may consider that, in cases of hypokalemic paralysis in which there is a potentially life-threatening presentation, the treatment with corticosteroids could be justified.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

References

[1] R. Evans, A. Zdebik, C. Ciurtin, and S. B. Walsh, "Renal involvement in primary Sjögren's syndrome," *Rheumatology*, vol. 54, no. 9, pp. 1541–1548, 2015.

[2] M. Ramos-Casals, P. Brito-Zerón, R. Seror et al., "Characterization of systemic disease in primary Sjögren's syndrome: EULAR-SS Task Force recommendations for articular, cutaneous, pulmonary and renal involvements," *Rheumatology*, vol. 54, no. 12, pp. 2230–2238, 2015.

[3] M. Ramos-Casals, P. Brito-Zerón, R. Solans et al., "Systemic involvement in primary Sjögren's syndrome evaluated by the EULAR-SS disease activity index: Analysis of 921 spanish patients (GEAS-SS registry)," *Rheumatology (United Kingdom)*, vol. 53, no. 2, Article ID ket349, pp. 321–331, 2014.

[4] H. Ren, W. M. Wang, X. N. Chen, W. Zhang, X. L. Pan Wang et al., "Renal involvement and followup of 130 patients with primary Sjögren's syndrome," *The Journal of Rheumatology*, vol. 35, pp. 278–284, 2008.

[5] D. M. Chisholm and D. K. Mason, "Labial salivary gland biopsy in Sjögren's disease," *Journal of Clinical Pathology*, vol. 21, no. 5, pp. 656–660, 1968.

[6] C. H. Shiboski, S. C. Shiboski, R. Seror et al., "2016 American College of Rheumatology/European League Against Rheumatism classification criteria for primary Sjogren's syndrome A consensus and data-driven methodology involving three international patient cohorts," *Annals of the Rheumatic Diseases*, vol. 76, no. 1, pp. 9–16, 2016.

[7] N. Talal, E. Zisman, and P. H. Schur, "Renal tubular acidosis, glomerulonephritis and immunologic factors in Sjogren's syndrome," *Arthritis and Rheumatology*, vol. 11, no. 6, pp. 774–786, 1968.

[8] S. Maripuri, J. P. Grande, T. G. Osborn et al., "Renal involvement in primary sjögren's syndrome: a clinicopathologic study," *Clinical Journal of the American Society of Nephrology*, vol. 4, no. 9, pp. 1423–1431, 2009.

[9] N. Bossini, S. Savoldi, F. Franceschini et al., "Clinical and morphological features of kidney involvement in primary Sjögren's syndrome," *Nephrology Dialysis Transplantation*, vol. 16, no. 12, pp. 2328–2336, 2001.

[10] H. François and X. Mariette, "Renal involvement in primary Sjögren syndrome," *Nature Reviews Nephrology*, vol. 12, no. 2, pp. 82–93, 2016.

[11] B. Bastani, L. Haragsim, S. Gluck, and K. C. Siamopoulos, "Lack of h-atpase in distal nephron causing hypokalaemic distal rta in a patient with sjögren's syndrome," *Nephrology Dialysis Transplantation*, vol. 10, no. 6, pp. 908-909, 1995.

[12] F. Takemoto, J. Hoshino, N. Sawa et al., "Autoantibodies against carbonic anhydrase II are increased in renal tubular acidosis associated with Sjögren syndrome," *American Journal of Medicine*, vol. 118, no. 2, pp. 181–184, 2005.

[13] P. E. DeFranco, L. Haragsim, P. G. Schmitz, B. Bastani, and J. P. Li, "Absence of vacuolar H+-ATPase pump in the collecting duct of a patient with hypokalemic distal renal tubular acidosis and Sjögren's syndrome," *Journal of the American Society of Nephrology*, vol. 6, no. 2, pp. 295–301, 1995.

[14] P. Sandhya and D. Danda, "Role of vacuolar ATPase and Skp1 in Sjögren's syndrome," *Medical Hypotheses*, vol. 82, no. 3, pp. 319–325, 2014.

[15] P. Eriksson, T. Denneberg, S. Eneström, B. Johansson, F. Lindström, and T. Skogh, "Urolithiasis and distal renal tubular acidosis preceding primary Sjögren's syndrome: A retrospective study 5-53 years after the presentation of urolithiasis," *Journal of Internal Medicine*, vol. 239, no. 6, pp. 483–488, 1996.

[16] S.-H. Lin, S. Cheema-Dhadli, M. Gowrishankar, E. B. Marliss, K. S. Kamel, and M. L. Halperin, "Control of excretion of potassium: lessons from studies during prolonged total fasting in human subjects," *American Journal of Physiology—Renal Physiology*, vol. 273, no. 5, pp. F796–F800, 1997.

[17] R. Shioji, T. Furuyama, S. Onodera, H. Saito, H. Ito, and Y. Sasaki, "Sjögren's syndrome and renal tubular acidosis," *The American Journal of Medicine*, vol. 48, no. 4, pp. 456–463, 1970.

[18] K.-K. Pun, C.-K. Wong, E. Y.-L. Tsui, S. C.-F. Tam, A. W.-C. Kung, and C. C.-L. Wang, "Hypokalemic periodic paralysis due to the Sjogren syndrome in Chinese patients," *Annals of Internal Medicine*, vol. 110, no. 5, pp. 405-406, 1989.

[19] R. D. R. Evans, C. M. Laing, C. Ciurtin, and S. B. Walsh, "Tubulointerstitial nephritis in primary Sjögren syndrome: clinical manifestations and response to treatment," *BMC Musculoskeletal Disorders*, vol. 5, no. 17, p. 2, 2016.

[20] N. Talal, "Sjögren's syndrome, lymphoproliferation, and renal tubular acidosis," *Annals of Internal Medicine*, vol. 74, no. 4, pp. 633-634, 1971.

[21] C.-J. Cheng, J.-S. Chiu, C.-C. Chen, and S.-H. Lin, "Unusual cause of hypokalemic paralysis in aged men: Sjögren syndrome," *Southern Medical Journal*, vol. 98, no. 12, pp. 1212–1215, 2005.

[22] M. Goroshi, S. Khare, T. Jamale, and N. Shah, "Primary Sjogren's syndrome presenting as hypokalemic paralysis: a case series," *Journal of Postgraduate Medicine*, vol. 63, no. 2, p. 128, 2016.

[23] M. Soy, Ö. N. Pamuk, M. Gerenli, and Y. Çelik, "A primary Sjögren's syndrome patient with distal renal tubular acidosis, who presented with symptoms of hypokalemic periodic paralysis: report of a case study and review of the literature," *Rheumatology International*, vol. 26, no. 1, pp. 86–89, 2005.

[24] R. Khadgawat, N. Tandon, D. Khandelwal, S. Bhattacharya, S. Kaur, and A. Ammini, "Hypokalemic paralysis as a presenting manifestation of primary Sjögren's syndrome: a report of two cases," *Indian Journal of Endocrinology and Metabolism*, vol. 16, no. 5, pp. 853–855, 2012.

[25] M. Kawashima, T. Amano, Y. Morita, M. Yamamura, and H. Makino, "Hypokalemic paralysis and osteomalacia secondary to renal tubular acidosis in a case with primary Sjögren's syndrome," *Modern Rheumatology*, vol. 16, no. 1, pp. 48–51, 2006.

[26] D. M. Comer, A. G. Droogan, I. S. Young, and A. P. Maxwell, "Hypokalaemic paralysis precipitated by distal renal tubular acidosis secondary to Sjögren's syndrome," *Annals of Clinical Biochemistry*, vol. 45, no. 2, pp. 221–225, 2008.

[27] S. Seirafian, M. Shafie, A. Abedini, B. Pakzad, and P. Roomizadeh, "Recurrent attacks of hypokalemic quadriparesis: an unusual presentation of primary Sjögren syndrome," *Internal Medicine*, vol. 55, no. 13, pp. 1797–1800, 2016.

[28] G. Vaidya and S. Ganeshpure, "Sjogren's syndrome with distal renal tubular acidosis presenting as hypokalaemic paralysis," *BMJ Case Reports*, vol. 19, 2012.

[29] S. Sarah, G. Lijo, E. Sukanya, and D. Rajasekaran, "Renal tubular acidosis due to Sjogren's syndrome presenting as hypokalemic quadriparesis: a report of two cases," *Indian Journal of Nephrology*, vol. 25, no. 6, pp. 386-387, 2015.

[30] N. Rao, M. John, N. Thomas, S. Rajaratnam, and M. S. Seshadri, "Aetiological, clinical and metabolic profile of hypokalaemic periodic paralysis in adults: a single-centre experience," *National Medical Journal of India*, vol. 19, no. 5, pp. 246–249, 2006.

[31] M. Nail, T. Bhat, M. Naqash et al., "Hypokalemic quadriparesis in an elderly female," *Indian Journal of Nephrology*, vol. 22, no. 5, pp. 402-403, 2012.

[32] S. Rajagopala, G. Danigeti, and D. Subrahmanyan, "An unusually dry story," *Indian Journal of Critical Care Medicine*, vol. 19, no. 9, pp. 550–553, 2015.

[33] A. V. Palkar, S. Pillai, and G. C. Rajadhyaksha, "Hypokalemic quadriparesis in Sjogren syndrome," *Indian Journal of Nephrology*, vol. 21, no. 3, pp. 191–193, 2011.

[34] S. Dasari, K. Naha, G. Vivek, V. Acharya, and M. Hande, "Primary presentation with acute flaccid quadriparesis in Sjogren's syndrome sans sicca," *BMJ Case Reports*, vol. 2013, 2013.

[35] J. Singhvi, A. Ganguli, and B. Kaur, "Primary Sjogren's syndrome presenting as Acute Flaccid Quadriplegia," *Annals of Neurosciences*, vol. 17, no. 2, 2010.

[36] Y.-C. Chang, C.-C. Huang, Y.-Y. Chiou, and C.-Y. Yu, "Retal tubular acidosis complicated with hypokalemic periodic paralysis," *Pediatric Neurology*, vol. 13, no. 1, pp. 52–54, 1995.

[37] I. Taylor and M. Parsons, "Hypokalemic paralysis revealing Sjögren's syndrome," *Journal of Clinical Neuroscience*, vol. 11, no. 3, pp. 319–321, 2004.

[38] G. Carminati, A. Chena, J. M. Orlando, S. Russo, S. Salomón, and J. A. Carena, "Distal renal tubular acidosis with rhabdomyolysis as the presenting form in 4 pregnant women," *Nefrologia*, vol. 21, no. 2, pp. 204–208, 2001.

[39] J. Muthukrishnan, S. Dawra, V. Marwaha, and C. S. Narayanan, "Sjögren's syndrome presenting as hypokalemic paralysis," *Medical Journal Armed Forces India*, vol. 71, pp. S172–S174, 2015.

[40] E. B. S. Prakash, M. E. Fernando, M. Sathiyasekaran, R. M. Bhoopathy, J. J. Jayanth, and J. Samuel, "Primary Sjögren's syndrome presenting with distal renal tubular acidosis and rhabdomyolysis," *Journal of Association of Physicians of India*, vol. 54, pp. 949-950, 2006.

[41] S. Skalova, L. Minxova, and R. Slezak, "Hypokalaemic paralysis revealing Sjogren's syndrome in a 16-year old girl," *Ghana Medical Journal*, vol. 42, pp. 124–128, 2008.

[42] C.-Y. Liao, C.-C. Wang, I.-H. Chen, J.-C. Shiang, M.-Y. Liu, and M.-K. Tsai, "Hypokalemic paralysis as a presenting manifestation of primary Sjögren's syndrome accompanied by vitamin D deficiency," *Internal Medicine*, vol. 52, no. 20, pp. 2351–2353, 2013.

[43] E. Sengul, F. Bunul, A. Yazici et al., "An unusual initial presentation of Sjögren's syndrome: severe hypokalemic paralysis secondary to distal renal tubular acidosis," *Eurasian Journal of Medicine*, vol. 45, no. 3, pp. 218–221, 2013.

[44] H. Yilmaz, M. Kaya, M. Özbek, K. Üreten, and S. I. Safa Yildirim, "Hypokalemic periodic paralysis in Sjogren's syndrome secondary to distal renal tubular acidosis," *Rheumatology International*, vol. 33, no. 7, pp. 1879–1882, 2013.

[45] J. L. Logan and J. Ahmed, "Critical hypokalemic renal tubular acidosis due to Sjögren's syndrome: association with the purported immune stimulant echinacea," *Clinical Rheumatology*, vol. 22, no. 2, pp. 158-159, 2003.

[46] T. Fujimoto, H. Shiiki, Y. Takahi, and K. Dohi, "Primary Sjögren's syndrome presenting as hypokalaemic periodic paralysis and respiratory arrest," *Clinical Rheumatology*, vol. 20, no. 5, pp. 365–368, 2001.

[47] K. Mugundhan, M. C. Mayan Vasif, P. D. Nidhin et al., "Hypokalemic paralysis in Sjogren's syndrome secondary to renal tubular acidosis," *Journal of Association of Physicians of India*, vol. 64, p. 72, 2016.