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Case Report

Rare presentations of Sjogren syndrome

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ABSTRACT

Sjögren's syndrome is a chronic systemic autoimmune disease characterized by lymphocytic infiltration of exocrine glands. It most commonly presenting with sicca symptoms. Sicca refers to dryness most often involving the eyes and mouth due to inflammation and resultant pathology of the lacrimal and salivary glands. Up to one-half of affected individuals also develop extra-glandular involvement in organs such as the joints, skin, lungs, gastrointestinal (GI) tract, nervous system, and kidneys. This condition is frequently associated with other autoimmune disorders including rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) - called the secondary Sjogrens syndrome. Evaluation of a patient with suspected Sjogren syndrome should include an evaluation of oral and ocular dryness and function. In addition to the history, this may include the performance of a Schirmer test, slit-lamp exam with vital dye staining, salivary flow rate, and/or nuclear scintigraphic evaluation of the salivary glandular function. Assessment of autoantibodies (ANA, RF, SS-A, and SS-B) should also be performed. Of these, SS-A is probably the most sensitive and specific antibody for Sjogren's but alone is not diagnostic since it may be present in other autoimmune disorders and may be absent in up to a third of Sjogren cases. The most specific single test is a minor salivary gland (lip) biopsy which will demonstrate focal lymphocytic sialadenitis (FLS). Therapies are directed toward replacing moisture at affected glandular sites and suppressing the autoimmune response locally as well as systemically. This activity reviews the evaluation and management of Sjogren syndrome and explains the different rare presentations in which it can present.

Keywords: Neuromyelitis optica, Hypokalemic periodic paralysis, Keratoconjunctivitis sicca

INTRODUCTION

Primary Sjogren syndrome is a systemic autoimmune disorder commonly presenting with dryness involving the eyes and mouth due to inflammation and resultant pathology of the lacrimal and salivary glands.[1]

Neuromyelitis optica is an auto-immune demyelinating disorder which present with optic neuritis, transverse myelitis, or other varied presentations like nausea, vomiting (the area postrema syndrome). Acute hypokalemic paralysis is a fatal medically reversible condition caused by, either due to excessive loss of potassium in urine like excessive use of diuretics, drugs like amphotericin, renal tubular acidosis or trans cellular shift of potassium in hypokalemic periodic paralysis.

We present 2 rare cases of Sjogren's syndrome which present with different presentations, one with neuromyelitis optica and other with acute hypokalemic paralysis secondary to distal renal tubular acidosis.

CASE REPORT

Case 1

A 22-year female, who came to the hospital with complaints of gastritis, hiccoughs and nausea and vomiting

since 1 year underwent OGD scopy, which revealed lax LES, large hiatus hernia, esophageal and gastric ulcers with H. pylori testing positive on RUT. Patient received treatment for 14days of HP kit.

Now 2-months later patient came with complaints of vomiting, dysphagia, cough associated with whitish mucoid expectoration, giddiness since 2 days, and high grade fever since 1 day. On examination, the patient was febrile (102.9 F), pulse of 110 bpm, blood pressure (BP) of 110/70 mmHg, saturation of 90 percent with RA and respiratory rate of 28/min. The chest had bilateral crackles. Patient was started on lines of treatment of aspiration pneumonia given her past history. Patient was shifted to intensive care unit (ICU), sputum and urine culture sent and patient started on Piperacillin+Tazobactam and clindamycin and O_2 support given. Initial complete blood count (CBC) was 10.5/14500/4.1 and erythrocyte sedimentation rate (ESR) 35. Creatinine was 1.0. SGOT/PT 493/283. ABG showed 7.391/35.6/314.1/21.1/99.7%

On detailed examination, patient had bilateral upbeat nystagmus, power was reduced on left side (3/5) than right (5/5), absent gag reflex on left side, palatal palsy, reduced sensations on left upper and lower limb. Suspecting lateral medullary syndrome, a magnetic resonance imaging (MRI) brain was done- s/o hyperintensity in medulla demyelination versus infarct. Workup for young stroke sent, Hams sucrose lysis test, APLA profile- all negative. Aspirin and statins started.

Patient had hiatus hernia and clinically found to have diaphragmatic paralysis based on paradoxical breathing. RNS study done to rule out myasthenia gravis- negative. AchR antibody <0.11.

Considering family history (mother having SLE), an ANA-IF was sent, which was very weak positive (cytoplasmic fluoroscence). Patient's tachypnea worsened and O_2 requirement increased, ABG at the time 7.257/52.5/66.6/22.9/90%. She was stated on pulse steroid – injection MPS 1 gm IV. She was electively intubated and sedated. Suspecting autoimmune demyelinaton- MS NMO- area postrema syndrome, patients CSF was done after ruling out papilledema (fundus normal, no optic neuritis) and sent for oligoclonal bands, which was negative. Routine analysis showed 5 white blood cells (WBCs), 28.4 mg/dl proteins and 104 mg/dl sugars.

Serum MOG antibody negative and anti NMO antibody was positive for aquaporin 4. ANA blot showed positivity for anti SSA Ro 60.

Liver function test (LFT) repeated- SGOT/PT 22/102. CBC 12.1/10.13/49, creatinine 0.4, and electrolyte 137/4.7/101.

Neurology review taken who advised to continue pulse steroid for 5 days and then switch to oral prednisolone 1 mg/kg body weight. Patient responded dramatically to steroid and was extubated on day 4 of pulse steroid as her breathing improved, and ABG normal- 7.534/26.7/87.9/21.5/97.7%. She was kept on non-invasive ventilation and eventually weaned off ventilator. With Extensive chest physiotherapy and suctioning, the patient was weaned off oxygen and was shifted to ward. Sputum culture showed *Pseudomonas* and *Klebsiella* species, injection cefepime 1 gm iv BD was started i/v/o increasing WBC counts (28,000).

Patients condition improved and she was started on tablet prednisolone 60 mg for 1 week, and then tapered by 10 mg. Patient started to tolerate liquids and then solids and ryles tube was removed. WBC counts showed reducing trend and was 9,500 at the time of discharge. Patient was discharged on prednisolone 40 mg and MMF 500 mg BD.

Table 1: Laboratory parameters of case report 1.

Date	Investigation	Report
15 April 2023	OGD SCOPY (in previous admission)	Lax LES, large hiatus hernia, lower esophageal and gastric ulcers, RUT done-positive for *H. pylori*, biopsy taken-HPE- chronic gastritis
02 May 2023	Routine labs	CBC-10.5/14.5/418, ESR-35, creatinine-1.0, electrolytes- 138/3.6/102, bilirubin T/D-1.2/0.7, SGOT/PT-283/357, urine routine-normal
02 May 2023	ABG	7.492/25.7/59.9/19.3/93.3%
02 May 2023	MRI brain plain	Altered signal intensity in posterior aspect of medulla oblongata appearing hyperintense on T2/T2 IR and isointense on T1 with partial diffusion restriction s/o demyelination versus acute infarct
03 May 2023	ANA-IF	Very weak positive- cytoplasmic fluorescence
04 May 2023	SGOT/PT	27/102
04 May 2023	Lumbar puncture	Protein 28.4, sugar 104, WBC 5 (100% lymphocytes), oligoclonal bands negative
05 May 2023	S. TSH	0.34
06 May 2023	APLA profile (beta2 glycoprotein, lupus anticoagulant, anti-cardiolipin)	Negative

Continued.

Date	Investigation	Report
	Sucrose lysis test	Negative
06 May 2023	Achr antibody	<0.11 (negative)
	NCV-RNS	Normal study
07 May 2023	CBC	9.8/15.45/405
08 May 2023	Sputum culture	*Pseudomonas, Klebsilla*
08 May 2023	2D echo	Bradycardia during study, ef 45-50%, mildly global LV hypokinesia
08 May 2023	ABG	7.534/26.7/87.9/21.5/97.7%
09 May 2023	ANA by immunoblot	Anti SSA Ro 60 positives
10 May 2023	MRI whole spine	No plaques/demyelination – no significant abnormality
11 May 2023	CBC	12.5/21.46/578
	CRP	3.30
12 May 2023	70-degree scopy	Normal
13 May 2023	24-hour urine protein	301.53 mg
	Shcirmer test	Negative
15 May 2023	USG abdomen	Normal
20 May 2023	CBC	12.2/9.51/574

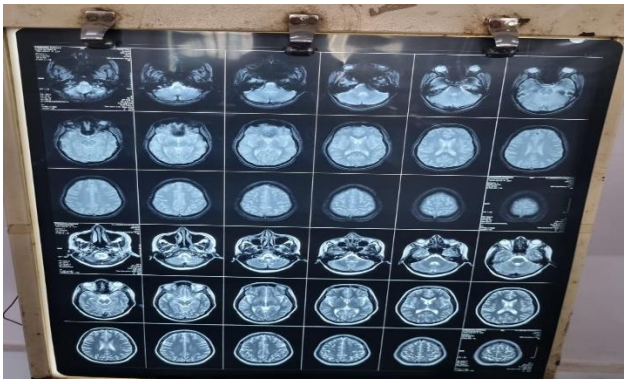

Figure 1: MRI brain showing altered signal intensity in posterior aspect of medulla oblongata appearing hyperintense on T2/T2 IR and isointense on T1 with partial diffusion restriction suggestive of demyelination versus acute infarct.

Case 2

A 40-year-old female patient came to the casualty, presented with complaints of sudden onset of weakness in both upper and lower limbs bilaterally which gradually progressed within a period of 12-15 hours. Patient also developed difficulty in swallowing after 15 hours of onset of symptoms, and she had not passed urine since morning. She was conscious and oriented although she had difficulty in neck holding and speaking. (Informant is reliable being the patient's husband)

There is no history of trauma, recent vaccination, any intoxication with drug, fever, arthralgia, morning stiffness, loss of appetite and loose stools. There is no history of similar complaints in the past. There is no history of remitting or relapsing of symptoms. There is no history of diurnal variation of the symptoms. There is no history of diabetes mellitus, hypertension, tuberculosis, bronchial asthma, hypothyroidism, rheumatic valvular heart disease.

General examination

On presentation, pulse was 86 bpm and regular, blood pressure 90/60 mmHg on right arm in supine position, SpO_2 98% on room air, respiratory rate 18/min, random blood sugar was 110 mg/dl. Mild pallor was present with no icterus, cyanosis, clubbing, lymphadenopathy or pedal edema.

Systemic examination

On the CNS examination, patient was conscious and oriented but with slowness of speech. Cranial nerves examination showed gag reflex was bilaterally sluggish with difficulty in swallowing with the rest of the cranial nerves being normal. Motor system examination-neck holding was absent, flickering movements were present in all 4 limbs in all the muscle groups and joints (power 1/5). Hypotonia was present in all 4 limbs. Deep tendon reflexes; knee reflex was bilaterally normal with the rest of them being bilaterally diminished. Plantar was bilateral flexor. On sensory system examination, it was intact. Cerebellar function was normal. Romberg's test could not be assessed. No neck rigidity and no Brudzinski's sign seen. Another systemic exam (respiratory, CVS, abdominal) was normal.

Course in the hospital

After admission, Foley's insertion was done and 800 ml urine came out within a few minutes. Supportive treatment was started. Patient was found to have hypokalemia with severe metabolic acidosis and was treated for the same with injectable potassium chloride and injectable sodium bicarbonate. Her power then improved from 1/5 to 2/5 in all 4 limbs. Six hours after starting potassium correction the patient developed carpopedal spasm which was then treated with injectable magnesium sulphate and injectable calcium gluconate.

Injectable potassium chloride and magnesium sulphate infusion was continued for 36 hours. Patient improved dramatically and could walk without support and was able to hold her neck and talk normally. Her power improved to 5/5 in all 4 limbs. On further questioning the patient, it was found that the patient had a history of dryness of the eyes since the last 6 months, she also gave the history of decreased salivation and dyspareunia since the last 6 months. Schirmer test was done in both the eyes and it was suggestive of 9 mm of wetness on the filter thus, suggesting decreased tears secretion.

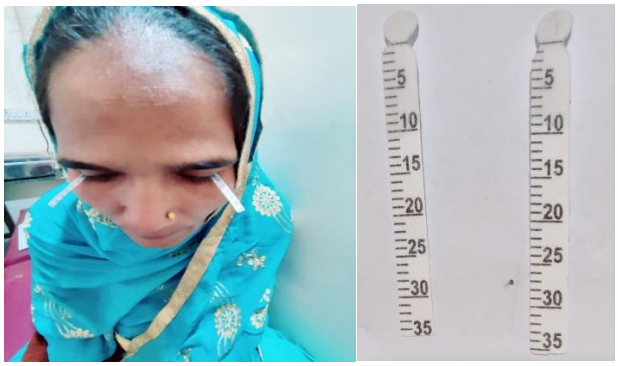

Figure 2 (a and b): Schirmer's test.

Figure 3: Carpopedal spasm.

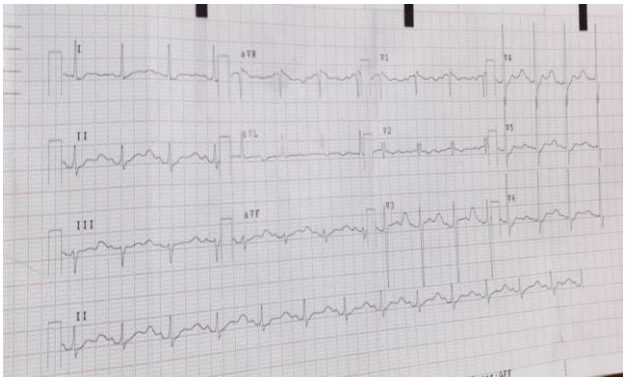

Figure 4: ECG suggestive of QTC prolongation.

Urinary potassium more than 15 was suggestive of renal loss. TTKG was calculated to be 9, suggestive of increased distal potassium secretion.

Laboratory investigations came showing hypokalemia with metabolic acidosis with hypomagnesemia and ANA blot showing SS-A and SS-B significantly positive suggestive of Sjogren's syndrome. Due to financial constraints and considering patient's drastic improvement with treatment, further investigations were not done.

Table 2: Laboratory parameters of case report 2.

Laboratory parameters	Result
Serum potassium (mEq/liter)	1.9
Serum sodium (mEq/liter)	154
PCO_2 arterial (mmHg)	26
pH arterial (mmHg)	7.16
HCO^{3-} arterial (mEq/liter)	8.9
Hemoglobin (gm/dl)	9.5
Total leukocyte count (cells/ cmm)	6000
Platelet count (platelets/microliter)	2 lakhs
Total bilirubin (mg/dl)	1
SGOT/SGPT (mg/dl)	40/38
Alkaline phosphatase (mg/dl)	110
Serum creatinine (mg/dl)	1.2
Serum calcium (mg/dl)	8.4
Serum magnesium (mEq/dl)	1.1
TSH (mIU/ml)	2.24
Serum osmolality (mosmol/kg)	325
Urine pH	5.6
Urine albumin	Trace
24-hour urine protein	Total protein 14.8 mg/dl, total urine volume 3500 ml, total protein/day 518 mg (N<140)
Urine osmolality (mosmol/kg)	399
Urinary potassium (mg/dl)	19.6
ANA screen	Positive
ANA titer	1:1000
Anti SSA antibody (U/ml)	141
Anti SSB antibody (U/ml)	58
RO (kD)	52 (150)
USG (abdomen + pelvis)	Normal bilateral kidney
Chest X-ray	Normal

She was discharged after 72 hours with tablet hydroxychloroquine 300 mg OD, tablet azathioprine 50 mg OD and carboxymethyl cellulose sodium eye drops and was asked to follow up after 5 days.

Lab investigations

All routine investigations had been sent and the patient was planned for MRI brain with whole spine screening, nerve conduction studies, electromyography and cerebrospinal fluid study. The electrocardiograph was suggestive of prolonged QT interval.

DISCUSSION

Sjögren syndrome is a chronic, slowly progressing autoimmune disease characterized by lymphocytic infiltration of the exocrine glands resulting in xerostomia and dry eyes (keratoconjunctivitis sicca). Female: male ratio is 9:1.[1] The prevalence of primary Sjögren syndrome is ~0.5-1% while 5-20% patients associated with other autoimmune diseases is called secondary Sjogren's syndrome such as RA, SLE, scleroderma, MCTD, primary biliary cirrhosis, autoimmune thyroid disease, chronic active hepatitis.[1]

Autoantibodies to Ro/SS-A and La/SS-B antigens are usually present prior to diagnosis and are associated with earlier disease onset, longer disease duration, salivary gland enlargement, extra-glandular (systemic) manifestations, and more intense lymphocytic infiltration of minor salivary glands. The extra-glandular manifestations of primary Sjögren syndrome include fatigability/ myalgia, arthralgia/arthritis, Raynaud's phenomenon, lung involvement which includes diseases such as small airway disease/lymphocytic interstitial pneumonitis, renal involvement such as interstitial kidney disease clinically manifested by hyposthenuria and renal tubular dysfunction with or without acidosis, liver involvement including primary biliary cirrhosis. Primary Sjogren patients have an increased relative risk for the development of B-cell non-Hodgkins's lymphoma. Many of these lymphomas are extranodal and may involve the salivary glands. Risk factors include persistent salivary gland swelling, enlarging lymph nodes, leukopenia, palpable purpura and low complement C4 at presentation.[1] Based on biopsy reports in the available literature, tubulointerstitial nephritis (TIN) is the most common histological abnormality.[2]

Treatment for Sjogren's syndrome includes mainly symptomatic relief for dry eyes by avoiding smoky areas and drugs like diuretics and anticholinergics, artificial tears without preservatives for lubrication, cyclosporine 2% olive or castor oil solution for local stimulation, pilocarpine (5 mg thrice daily thrice) for systemic stimulation and in case of severely dry eyes, soft contact lenses or corneal transplantation is required. For dry mouth, oral hygiene after each meal, topical application of fluoride and topical nystatin or clotrimazole for oral candidiasis. Persistent dry cough because of xero-trachea may respond to humidification, secretagogues and guaifenesin (1200 mg twice a day).

For cases with parotid gland enlargement antibiotics treat superinfection and in hard, persistent glands ruling out lymphoma is important. Hydroxychloroquine (200-400 mg/dl) or methotrexate (0.2-0.3 mg/kg body weight weekly) plus prednisolone (<10 mg daily orally) is given to the patients presenting with arthritis. For patients with Raynaud's phenomenon and renal tubular acidosis, cold protection with gloves and replacement with bicarbonate is given respectively. Vaginal dryness is treated with propionic acid gel. Combination of anti-CD-20 with a classic CHOP regimen [cyclosporine, adriamycin (hydroxydaunorubicin), vincristine (oncovin), and prednisone] leads to increased survival rates among patients with high-grade lymphomas. Rituximab appears to be effective in patients with systemic disease, particularly in those with purpura, arthritis, and fatigability.[3]

According to the European Community Study Group, the diagnosis of Sjögren syndrome should be based on the results of: biopsies of the minor salivary glands and/or lachrymal glands; examination of the oral cavity consisting of sialography or the Saxon test (in which a sponge is chewed for 2 min by the patient and then weighed) combined with salivary-gland scintigraphy; eye examination consisting of the Schirmer test plus the Bengal rose test or the fluorescein test; and assays for anti-Ro/SSA or anti-La/SSB antibodies. For a definitive diagnosis, at least two of the four criteria must be met.[4]

Neuromyelitis optica (NMO) also known as Devic syndrome is a rare relapsing auto-immune disease of the central nervous system (CNS) which is sometimes found in association with other autoimmune disorders, including Sjogren syndrome.[5] Based on the revised criteria by Wingerchuck et al, a diagnosis of NMO can be made in the presence of both absolute and two of three supportive criteria.[6] The absolute criteria include optic neuritis and myelitis; while the supportive criteria are MRI evidence of a contiguous spinal cord lesion (3 or more segments in length), MRI brain non-diagnostic for multiple sclerosis and serological evidence of NMO-IgG or aquaporin 4 (AQP4) antibodies. NMO spectrum disorders (NMOSD) includes a wide range of neurologic conditions that express NMO antibody and share features with NMO but do not meet the strict diagnostic criteria specified previously, example is the area postrema syndrome.[7]

NMO is characterized by recurrent episodes of myelitis and optic neuritis and most patients have a unique antibody against NMO IgG/ AQP4. Patients who have NMO IgG/AQP4 antibodies have frequent relapses compared to those who do not have the antibodies. NMOSD is manifested by loss of AQP4, which is a bidirectional water channel found in the cell membrane of foot processes of the astrocytes and on the ependymal cells in the CNS.[8] NMO IgG antibodies cause destruction of the AQP4 channels on the astrocyte foot processes by complement mediated cytotoxicity via the lytic complex C5b9, thus causing de-myelination.[9] Studies have shown that an inflammatory process such as an infection (viral or bacterial) can be a trigger in developing immunogenicity.[10] It may also play a role in damaging the blood brain barrier allowing NMO IgG penetration.[11]

The treatment options are empiric and based on disease severity ranging from plasmapheresis, corticosteroids, CTX and rituximab to oral agents such as azathioprine, MMF and methotrexate. Early initiation of systemic

steroids alone or in combination with other immunosuppressive therapies is most often used.

Distal renal tubular acidosis (RTA), also known as type 1 RTA or classic RTA, is a complex entity characterized by an inability to acidify the urine; a process that occurs in the distal parts of the nephron, including the connecting tubule and the collecting duct.[12] The most common form of dRTA is due to selective failure of activity or expression of the H^+-ATPase. The decreased transit through the proton pump inhibits urine acidification and reduces the electrical dissipation of the membrane potential. The latter has been suspected to be a driving force for K^+ secretion and eventual potassium wasting. As hypokalemia progresses, storage tissues such as the skeletal muscle compensate by releasing K^+ to the extracellular compartment, for which laboratory data may fail to uncover a K^+ imbalance. However, a serum K^+ <3 mEq/l is related to a total body deficit of >200 mEq, which varies with weight. Patients with Sjögren's syndrome and dRTA can present life-threatening complications owing to massive intracellular potassium depletion, including rhabdomyolysis, respiratory paralysis, or malignant arrhythmias. There is a downregulated expression of the vacuolar H^+-ATPase in the A-intercalated cells in patients with SS with concomitant under-expression of AE1 (pendrin) in the B-intercalated cells. It is hypothesized that the reduced secretion of H^+ is the primary dysfunction in SS, whereas the under-regulation of pendrin is a compensation to suppress HCO_3^- secretion and prevent further acidosis. Despite the presence of H^+-ATPase and AE1 in other parts of the nephron, patients with Sjögren's Syndrome present selective under-expression of these proteins in the collecting duct.[12]

While the immunomodulatory effect of hydroxychloroquine relays in suppressing toll-like receptors in a wide range of cells, azathioprine is an antimetabolite that inhibits synthesis of DNA, RNA, and proteins predominantly in T cells and B cells. Although both medications have been used for the management of this condition, outcomes have been variable in the literature.[12]

CONCLUSION

Many NMO patients have a systemic autoimmune disorder, often systemic lupus erythematosus, Sjögren's syndrome, pANCA associated vasculitis, myasthenia gravis, Hashimoto's thyroiditis, or mixed connective tissue disease. The past and family history is very important in auto-immune conditions and all auto-immune conditions are linked to each other. Rheumatologic evaluation is necessary in these patients to rule out the coexistent connective tissue disease for appropriate management. De-myelination should be kept in mind when such complex neurological involvement is there. Similarly, all patients with hypokalemic periodic paralysis should be evaluated rheumatologically as there is association with auto-immune disorders like Sjogren syndrome.

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