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Sjogren Syndrome: A Case Report

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ABSTRACT

The chronic, systemic autoimmune disease known as Sjogren syndrome is typified by lymphocytic infiltration of the exocrine glands. The lacrimal and salivary glands are intricately involved, and the result is Keratoconjunctivitis Sicca and xerostomia. It can manifest in two ways: primary or secondary, linked to another autoimmune condition, most frequently rheumatoid arthritis. There have been many criteria put forth for the diagnosis of Sjogren syndrome. The international classification criteria for Sjogren's syndrome that were developed by American and European groups are the most widely accepted. Ocular symptoms, oral symptoms, ocular signs, histopathology, involvement of the salivary glands, and x-ray are some of these criteria. Four of the six items must be met for the classification to be complete; one of the requirements must be a positive minor salivary gland biopsy or positive antibody test. Prompt diagnosis is essential to stop additional problems. This paper aims to highlight oral changes, advanced diagnosis, and Sjogren's syndrome management.

KEYWORDS: *Autoimmune diseases, salivary gland, Sjogren's syndrome*

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INTRODUCTION

The chronic, systemic autoimmune disease known as Sjogren syndrome is typified by lymphocytic infiltration of the exocrine glands. Delicate involvement of the lacrimal and salivary glands thus culminates in xerostomia and keratoconjunctivitis sicca.[1] There have been many criteria put forth for the diagnosis of Sjogren syndrome. The international classification criteria for Sjogren's syndrome that were developed by American and European groups are the most widely accepted.[2] It is known that there is a tendency toward femininity in the fourth and fifth decades of life, and that this tendency is uncommon in children, despite clinical studies that have been linked to these circumstances, even though there is currently no known treatment for Sjogren's syndrome. Laboratory tests, clinical signs, and anamnesis are used to diagnose the syndrome.[3] The set of symptoms and signs, in addition to the clinical identification linked to the complementary exams, should form the basis of the differential diagnosis. Since the disease has distinct oral manifestations, including dry mouth followed by hyposalivation, burning of the lingual and jugal mucosa, altered taste, fissured and despapilated tongue, erythematous and atrophic mucosa, and a propensity for fungal infections, dentistry is crucial to the early diagnosis of the condition. mostly erythematous candidiasis in the mouth.[4]

Case Report

A 50-year-old female patient reported to the Department of intensive care unit, Saveetha Medical College and Hospital, Chennai, Tamil Nadu, with complaining of: Inability to eat completely due to loss of teeth, fatigue, joint pain, neck swelling, Along with that patient also complained of dryness of mouth, since 1 year, and dryness of eyes since 5-6 years. Extra oral examination showed bilateral parotid gland enlargement present on the right (Figure 1) and left side of the parotid region (Figure2).

On an inspection – swelling on right and left of the parotid gland, measuring about 2.5 cm × 2 cm in diameter. Surface texture over the swelling was normal. Swelling extending superior up to the inferior

border of the mandible at the angle region. Ear lobe was everted on the right side.

On palpation it was diffuse, firm, non-movable, warm, tender on palpation, overlying surface texture was normal, along with that dryness of eyes, fever was also noted. Bilateral submandibular lymph nodes were palpable.

Intra-oral examination showed upper and lower well-formed edentulous alveolar ridges. Diffuse black pigmentation present on right and left buccal mucosa and palatal mucosa. Depapilation was present on the anterior 2/3rd and lateral border of the tongue (Figure 3). Buccal mucosa was thin and friable. After milking of the left side of the parotid gland pus discharge was coming through parotid duct (Figure 4).

Culture and sensitivity test showed organism isolated Escherichia coli grown. Complete hemogram showed increased erythrocyte sedimentation rate. Schirmer test and Rose Bengal dye test was positive. Serum immunoglobulin – SS-A RO positive for Sjogrens syndrome. RA factor was positive for rheumatoid arthritis. Ultrasonography shows bilateral submandibular and parotid gland enlargement with multiple hypoechoic lesions within showing very high vascularity, likely to present systemic disorder like Sjogren's syndrome. X-ray shows diffuse foci of sialectasis. Incisional biopsy of lower lip was not done because the patient was not ready for the biopsy.

Based on history, clinical presentation of the patient, and above investigatory findings, a confirmatory diagnosis of primary Sjogren syndrome was given.

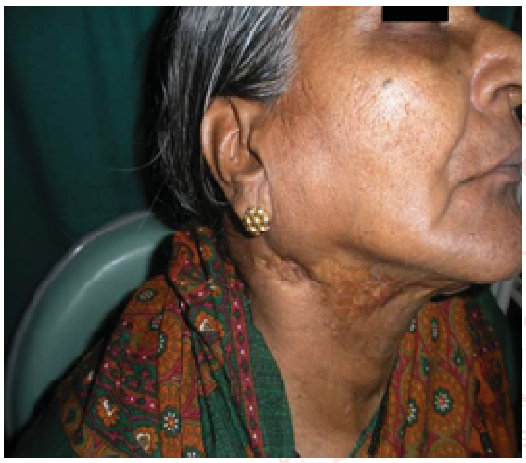

Figure 1: Right parotid gland enlargement

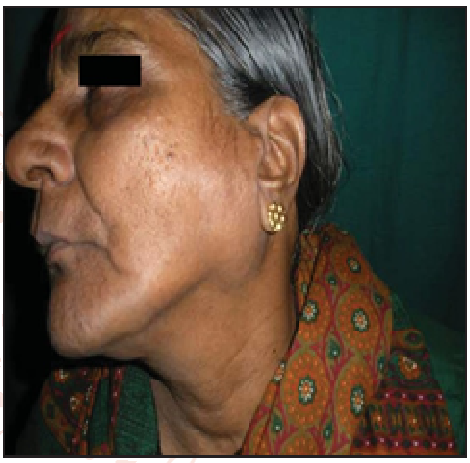

Figure 2: Left parotid gland enlargement.

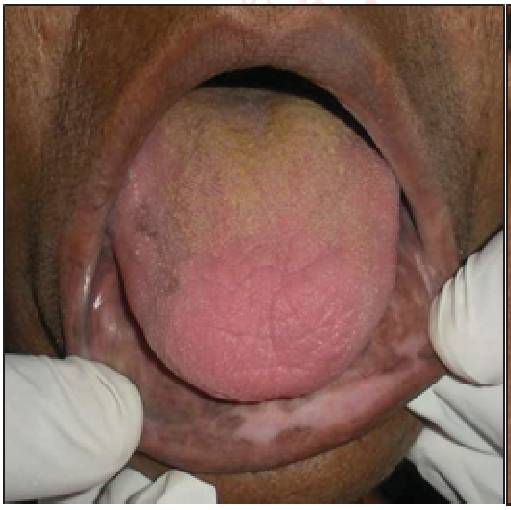

Figure 3: Depapillation on lateral border and anterior 2/3rd of tongue.

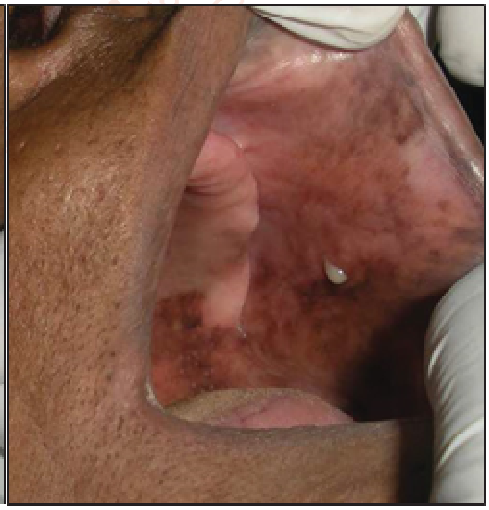

Figure 4: Pus discharge through the parotid duct.

Discussion

A clinical symptom complex is the definition of Sjogren's syndrome. The primary salivary and lacrimal exocrine glands are destroyed by an autoimmune process that results in keratoconjunctivities sicca, dry mouth, and, in over 50% of cases, hypertrophy of the parotid glands.[5] When the condition is restricted to this pattern of involvement, primary Sjogren syndrome is diagnosed. This pattern of involvement, however, might be a sign of another autoimmune disease that is more well defined, including primary biliary cirrhosis, systemic lupus erythematosus, or rheumatoid arthritis. It is known as secondary Sjogren syndrome in this setting.[6] Women over 40 are predominantly affected by Sjogren syndrome. However, it has been detected in

kids and teens using more recent diagnostic methods such parotid biopsies and antibody identifications. Typically, the parotid enlargements are painless and asymmetrical.[7] Our patient underwent a thorough physical examination both at the time of presentation and throughout the three-month follow-up, but no signs of a related connective tissue disease were found. The degree and severity of the clinical symptoms determine how best to treat Sjogren's syndrome, and a multidisciplinary approach is preferable. Artificial tears and salivary substitutes are used as symptomatic treatments to alleviate symptoms and prevent local infectious complications such as corneal inflammation and conjunctivitis. To prevent periodontal disease and caries, a comprehensive preventive program should be followed in all cases. Only those cases with substantial leukopenia, severe clinical symptoms, or indications of organ damage should receive corticosteroid treatment. Although the pathogenesis of Sjogren syndrome is unclear and complex, it is believed to be comparable to that of benign lymphoephtal lesions. Due to lymphocytic cell invasion, 6–10% of cases develop into lymphomas.[8]

The patient reported for the first and second visit shows value added scale (VAS). VAS scale-5 for burning sensation with hot and spicy foods. Pus collected from stensen's duct send for culture and sensitivity. Drainage of pus like the exudate after milking of the left parotid gland. Prescribed medicine was tablet doxycycline 100 mg – on first day BID, followed by OD for 5 days. Tablet paracetamol 500 mg BID for 1 week. Continuous sipping of water throughout the day, orbit VAS scale-4 for burning sensation of hot and spicy foods. After second week, drainage of pus like the exudate through left side Stensons duct after milking of the parotid gland. Tablet augmentin 625 mg – OD for 7 days. Tablet paracetamol 500 mg sos. Continues sipping of water throughout day, orbit chewing gums 3-4 times per day. Aquet spray (lubricating and moisten spray) – 3-4 times per day. Maintenance therapy was continued for next 3 weeks. Capsule menopause was also advised after consultation with a gynecologist from Bharati Hospital for about 1 month. This hormonal replacement therapy shows 10-20% reduction in xerostomia.

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