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Oral manifestation of systemic lupus erythematosus: A case report

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Abstract
Systemic lupus erythematous (SLE), one of the rare dermatoses shows desquamative lesions as the oral manifestation Periodontal disease and SLE are both multifactorial conditions that share several pathogenic characteristics. The similar mechanisms of tissue destruction for periodontitis and other autoimmune diseases have stimulated the study of potential associations between these conditions. Hence, in the present study we present a case of a 70-year-old female suffering from SLE with oral manifestations.

Keywords: Autoimmune disease, lupus erythematosus, periodontal disease

Introduction
Systemic lupus erythematosus (SLE) is a systemic, chronic inflammatory condition with diverse clinical manifestations, primarily affecting the joints, internal organs, and the skin [1]. It commonly affects patients in the fourth decade of life, especially women with a ratio 7 to 10:1.Periodontitis is characterized by chronic gingival inflammation that leads to destruction of the periodontal tissues supporting the teeth, and subsequently, may lead to tooth loss [2]. Although it is primarily initiated by bacteria, the host immune response plays a significant role in its development. Oral manifestations of SLE are frequently encountered and may include oral ulceration, honeycomb plaque, raised keratotic plaque, nonspecific erythema, purpura, petechiae, and cheilitis [3]. Periodontitis and SLE are both multifactorial conditions that share several pathogenic characteristics, such as elevated serum levels of beta 2-glycoprotein I-dependent anti-cardiolipin, the IgG Fc receptor, and proinflammatory cytokines. The similar mechanisms of tissue destruction for periodontitis and other autoimmune diseases have stimulated the study of potential associations between these conditions [4]. In spite of presenting different etiologies, the existence of similar destructive mechanisms could explain an eventual association between periodontitis and SLE. These potential mechanisms in common may involve deregulation, especially in the innate immune system, with action of phagocyte cells and of proinflammatory cytokines, such as IL-1β and IL-18, in the pathogenesis of both conditions, contributing to tissue destruction [5, 6].

Case report
A 70-year-old woman reported to the Department of Periodontology of the Adesh Institute of Dental Sciences & Research with chief complaint of fever, dry mouth and generalized redness of gums with associated burning sensation in the gums from one month which had started spontaneously. She also had bleeding gums for the past one month, while brushing and eating. Her medical history revealed that she was diagnosed with systemic lupus erythematosus about 10 years ago. Her vital signs were monitored which were normal. Clinical examination revealed facial cutaneous depigmented malar rash. [Fig 1]
Her laboratory examination revealed WBC count at 2300 (neutrophils 30%, lymphocytes 45%, monocytes 25%), high ESR and normal urinalysis. Intraoral examination revealed erythematous marginal, attached gingiva and interdental papilla in maxillary and mandibular anterior region. Nikolsky's sign was positive. [Fig 2] Generalized BOP (bleeding on probing) was positive; however, no periodontal pockets or furcation involvement was seen.

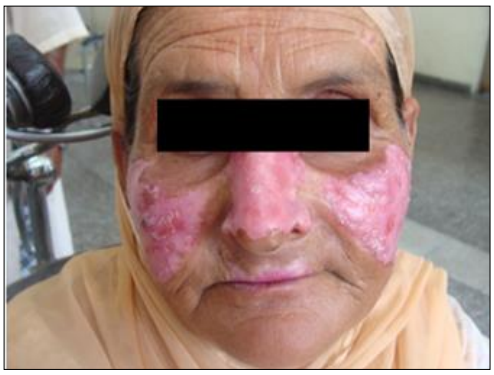

Fig 1: Erythematous rash on malar region of face

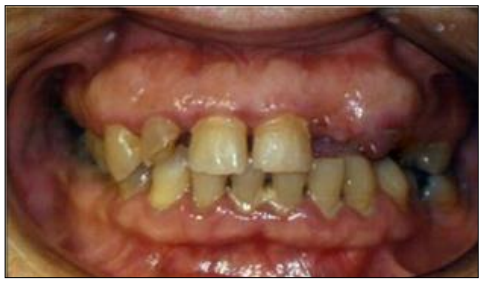

Fig 2: Clinical picture showing gingivitis

In accordance to her medical situation and oral examination, oral lesions were diagnosed to be SLE manifestations. Differential diagnosis included oral lichen planus. Clinical diagnosis was established by incisional biopsy, performed under local anesthesia in lesional mucosa, involving clinically healthy mucosa.

Histological examination reveals atrophic areas of covering epithelium with underlying connective tissue shows moderate inflammation consisting of lymphocytes, plasma cells and a few neutrophils. Hydropic degeneration of basal epithelium layer exists focally, along with presence of lymphocytes and a few colloid bodies. (Figure 3)All the above findings were consistent with SLE.

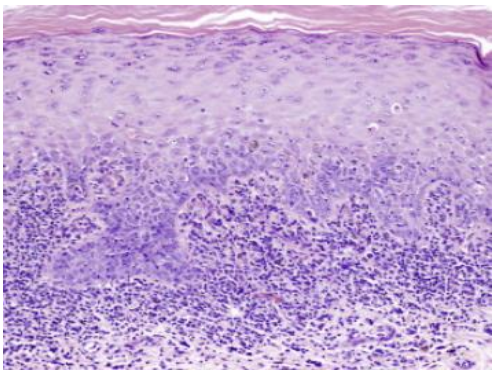

Fig 3: Histopathologic photograph showing hydropic degeneration and inflammation.

We performed hand scaling on the patient, as gently as possible. She was recommended to maintain oral hygiene and was prescribed a soft-bristle toothbrush with Triamcinolone acteonide oral paste and Chlorhexidine mouthwash. The patient was advised to follow up in 7 days. She was asked to avoid excessive exposure to sunlight because ultraviolet light may precipitate disease activity.

Discussion

Systemic lupus erythematosus (SLE) is a serious multisystem disease with variety of cutaneous and oral manifestations. It is an auto immune disease, where the patient develops auto antibodies to many of their cells and cell components and tissues. Some of the manifestations appear to result from deposition of antigen antibody complex in the tissues. SLE often presents in non-specific, vague fashion, frequently with periods of remission and exacerbation. Cutaneous manifestations are erythematous patches on the face which coalesce to form a roughly symmetrical pattern over the cheeks across the bridge of the nose in a butterfly distribution. Skin over the neck, arms, shoulders and fingers are also affected. Patient may complain of itching or burning sensation. Some may present with areas of hyperpigmentation. Oral lesions of SLE develop in 20-50% of patients. The oral mucosa may be involved either prior to or following the development of skin lesions or even in the absence of skin manifestations. Oral lesions begin as erythematous areas, without induration and with white spots. The margins of the lesions are not sharply demarcated but frequently show the formation of narrow zone of keratinization. Hyperemia with edema will be there and there may be a tendency for bleeding [7].

Immunological pathways and predisposing genetic factors common to periodontal disease and rheumatic diseases, including systemic lupus erythematosus, have been studied. Genetic pleomorphism is a very important factor in both SLE and periodontal disease. In fact, a relationship has been found between both diseases in certain genotypes. Moreover, periodontal treatment was shown to improve the SLE activity of patients on immunosuppressive medication [8-12]. Sales *et al*. showed that a relationship does exist between SLE activity and periodontal status, along with a relationship between the latter and levels of CRP in serum. In an earlier study, Rhodus and Johnson showed a high prevalence of oral lesions among SLE patients, including angular cheilitis, ulcers, mucositis, and glossitis. A high prevalence of oral complaints such as dysphagia, dysgeusia, and glossodynia was also present [13]. Fabbri *et al*. showed that treatment of periodontal disease among SLE patients on immunosuppressive therapy is beneficial in controlling disease activity [14]. A more recent study as well showed that treatment of periodontal disease aids in reducing the symptoms of SLE [15]. Two studies examined inflammatory cytokines in gingival crevicular fluid (which can be found in periodontal pockets) and in serum. Souza found higher crevicular fluid levels of total and free elastase and lower crevicular fluid levels of IL-18, but higher serum levels of IL-18 in patients with juvenile SLE *versus* control group. CAL showed a negative correlation with crevicular fluid IL-18 levels, suggesting that this cytokine could have a protective effect on the tissue destruction associated with PD [16]. In the study by Miceli *et al*., healthy adolescents had higher levels of IL-1-β in crevicular fluid *versus* patients with juvenile SLE who were evaluated for presence of periodontal disease [17]. Al-Mutairi KD *et al* compared periodontal findings in systemic lupus erythematosus (SLE) patients and healthy controls, and to determine, whether there is a correlation between periodontal parameters and SLE biomarkers. Twenty-five participants diagnosed with SLE and 50 healthy controls were selected. Periodontal assessment consisted of clinical attachment level (CAL), probing depth (PD), bleeding on probing, and plaque scores. For the SLE group, several laboratory tests were obtained, such as, white blood cell count, hemoglobin level, platelet count, anti-nuclear antibody, anti-double-stranded DNA antibody, calcium level, and vitamin D. Periodontal

findings in SLE patients and controls were not significantly different. The SLE patients who had no flare-ups for more than a year showed significant bleeding on probing and deeper PD compared with those who had flare-ups less than a year before starting the study. They concluded that periodontal health was not different between SLE patients and healthy controls. In SLE patients however, flare-ups and presence of arthritis had a significant relation with periodontal health [18].

In the present case report, an association between SLE and periodontal disease as desquamative gingivitis was seen.

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