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Case Report

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Regressed lichen planus: a case report

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ABSTRACT

Lichen planus is a chronic inflammatory autoimmune disease. The incidence of the disease is higher than other dermatoses. Clinically, it may present in different forms, the most common being the reticular form. Lichen planus may be precancerous and may develop into a malignant lesion. Although topical corticosteroids are the first choice for treatment, many different topical or systemic agents can be used. The aim of this case report is to discuss the clinical features of lichen planus.

Keywords: Regressed lichen planus, malignant transformation, etiology of LP

INTRODUCTION

Lichen planus (LP) is a chronic inflammatory disease of unknown etiology that commonly affects mucous membranes, skin, genital mucosa, scalp and nails. It was first described by Erasmus Wilson in 1869.[1] Individuals aged fifty and over are more affected.[2] The disease affects women twice as often as men. Lichen planus affecting the oral mucosal area is called oral lichen planus (OLP) and can be seen alone or in combination with skin lesions. The incidence of LP is 1.9%, which is more common than the cutaneous form (0.23%). Clinically, LP can be seen in six types: papules, reticular, plaque-like, atrophic, erosive and bullous.[3] The most common type is the reticular pattern of fine white lines known as "*Wickham's striae*". LP may be associated with systemic diseases such as diabetes mellitus, hepatitis-C and hypertension. Lichenoid lesions can be caused by various medications such as antibiotics, antihypertensives, anti-inflammatory drugs and antimalarial drugs. Metal restorations can also trigger lichenoid reactions in the adjacent oral mucosa.[4] Treatment of LP is usually performed when erosive lesions or ulcerations are present. Before starting a local or systemic treatment, it is important to eliminate all factors that may be responsible. Topical or systemic corticosteroid therapy is recommended for erosive lesions.[4]

CASE

A 28-year-old woman presented to Kırıkkale University, Faculty of Dentistry, Department of Periodontology with complaints of pain and redness in the buccal mucosa and gingiva. In the anamnesis, it was learned that he started complaining of pain and burning in the mouth after having a dental scaling 3 months ago and that he did not have any systemic disease. The patient does not smoke. Intraoral examination revealed lesions and white striae on the gums (Figure 1A, 1B).

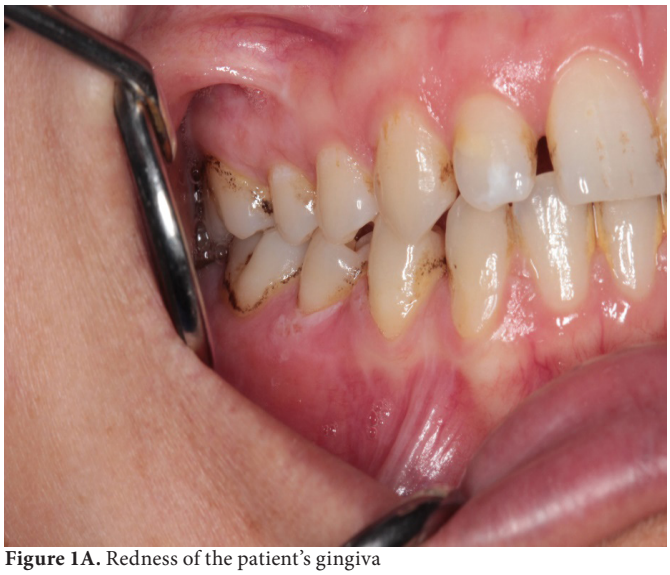

Figure 1A. Redness of the patient's gingiva

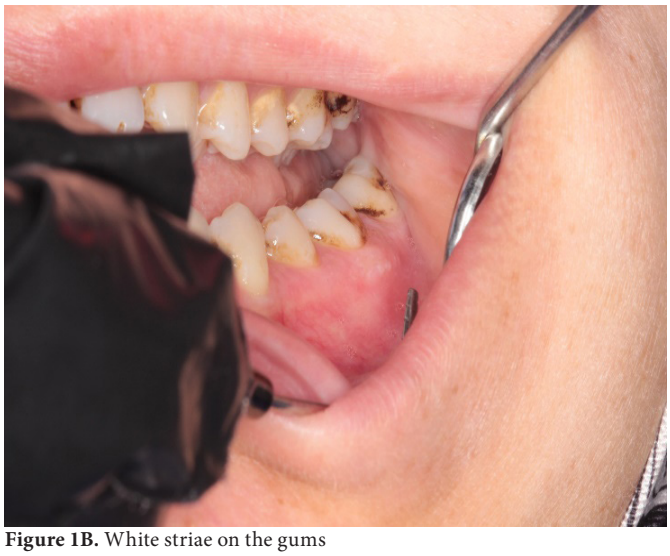

Figure 1B. White striae on the gums

For definitive diagnosis, one histopathology and one direct immunofluorescence were performed by punch biopsy from the gingival area where the lesion was most severe.

Histopathologic examination of the tissue showed mucosal tissues in which epithelium and connective tissue were seen separately from each other. In the fragment of parakeratinized mucosal epithelium, degeneration and loss of basal layer cells were observed and inflammatory cells penetrated into the epithelium. In the connective tissue fragment, there was band-shaped lymphocytic infiltration in the lamina propria. In the material sent for frozen section, limited connective tissue was seen under the edematous epithelium. Immunofluorescence studies revealed focal positive fibrinogen, negative C3, IgG, IgM and IgA. As a result of clinical and histopathologic evaluations, regressed lichen planus was diagnosed. The patient was recommended periodontal treatment with tartar removal, oral hygiene education and mouthwash containing 0.2% chlorhexidine gluconate.

DISCUSSION

Although the etiology is unknown, the disease is thought to be caused by a specific antigenic mechanism or non-specific mechanisms including autoimmune response triggered by epithelial basal cell modification and multifactorial factors. Antigen-specific mechanisms may include limited antigen presentation by lesional keratinocytes including MHC class-I and MHC class-II, activation of antigen-specific CD4+helper T cells and CD8+cytotoxic T cells, clonal expansion of antigen-specific T cells and keratinocyte apoptosis triggered by antigen-specific CD8+cytotoxic T cells. Many non-specific mechanisms may play a role, including heat shock proteins, reactive oxygen products, stress, mast cell chemotaxis.[5] The incidence of the disease is higher than in other dermatoses and is more common in older women than in men. It is reported that the lesion may be precancerous and may transform into a malignant lesion. The incidence of malignant transformation varies between 0.5-2.5% in studies. However, there are some authors who do not consider lichen planus lesions as malignant.[6]

LP affecting the gingiva is characterized by the presence of diffuse erythematous areas that are desquamative or ulcerated. Lesions may occur along keratinized gingiva, hyperkeratotic reticulated lines may be present on the periphery of erosive areas and may facilitate the diagnosis. This clinical appearance is not specific only for LP. It is possible to find similar clinical appearance in many diseases such as cicatricial pemphigoid, lupus erythematosus, pemphigus vulgaris, linear IgA dermatosis. In addition, diseases such as hormonal dysfunction, candidiasis, lichenoid lesions and vulvo-vaginal-gingival syndrome should be kept in mind in the differential diagnosis of lichen planus.[7,8]

Our definitive diagnosis was made after biopsy. In our case, direct immunofluorescence (DIF) was performed for the differential diagnosis of pemphigus group diseases (paraneoplastic pemphigus) and pemphigoid (mucous membrane pemphigoid).[9] IgG, C3 were negative. Pemphigus group diseases can be diagnosed by detection of antibodies against keratinocyte cell surface. Detection of specific antibodies against the squamous intercellular intermediate (matrix) in the tissue and serum of the patient with pemphigus is necessary for definitive diagnosis. Immunofluorescence methods are currently one of the most important diagnostic methods of immunobullous diseases.[10] In IgA pemphigus, intercellular IgA deposition is observed in 50% of cases in DIF. In our case, IgA was negative in direct immunofluorescence for linear IgA.

The aim of treatment of symptomatic LP is to relieve painful ulcerations or burning sensation. A stepwise approach should be adopted. Topical corticosteroid therapy is the mainstay of treatment for ulcerative disease. There is limited evidence from randomized controlled trials on the definitive efficacy of the various commonly used preparations. In addition to treatment, patients should also be counseled about the need to maintain a high standard of oral hygiene and eliminate any causes of mucosal trauma, such as unsuitable dentures, sharp spikes and poor dental restorations. Patients should be informed that there is a very small risk of malignancy associated with LP and that long-term follow-up is appropriate.[11]

CONCLUSION

As the cause of LP is currently unknown, there are no specific preventive regimens for this disease. The pathogenesis of LP may involve both antigen-specific and non-specific mechanisms. However, regular clinical follow-ups should be performed against the risk of malignancy. Careful, regular and long-term follow-up of patients with LP is necessary for early detection of oral squamous cell carcinoma. Follow-up intervals of 2 months or 12 months can be adjusted according to the patient.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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