Oral Lichen Planus

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KEYWORDS

• Lichen planus • Oral • Autoimmune disease • Reticular • Erosive

KEY POINTS

- Lichen planus is an immunologically mediated mucocutaneous disease, affecting 0.1% to 4% of the general population.
- Several reports suggest an association of hepatitis C virus and human papilloma virus with oral lichen planus.
- Oral lichen planus predominately affects females, with most patients aged between 30 and 70 years.
- Common treatment options include systemic and topical corticosteroids, topical retinoids, cyclosporine, tacrolimus, and pimecrolimus.
- The potential malignant transformation of lichen planus remains highly controversial; periodic observation of these lesions for dysplastic changes remains prudent.

INTRODUCTION

Lichen planus is an immunologically mediated mucocutaneous disease. A complex series of immunologic events is purported to cause the initiation and perpetuation of the condition. It is a common disease, affecting 0.1% to 4.0% of the general population. Patients often have concomitant cutaneous and oral lesions (**Fig. 1**).[1,2] Oral lesions may be chronic in nature, remitting and relapsing with varying degrees of morbidity; they range from asymptomatic to debilitating pain.

Clinically and histologically similar entities, including lichenoid drug reaction (**Fig. 2**), lichenoid mucositis (**Fig. 3**), and lichenoid dermatitis, can make the diagnosis of lichen planus challenging. These lesions are associated with the administration of a drug or direct contact with a metal and often, but not always, resolve when the offending agent is removed. Antibiotics, antihypertensives, gold, diuretics, antimalarials, and nonsteroidal antiinflammatory drugs may precipitate these conditions.[3] Oral lichenoid reactions in chronic graft-versus-host disease are also well recognized.[4,5] It is not possible to distinguish such lesions from oral lichen planus (OLP) clinically or histologically; there is, however, a significantly higher frequency of CD25+ cells in the epithelium and the connective tissue of OLP than in chronic graft-versus-host disease. This variance in frequency indicates differences in regulatory mechanisms of the immunologic response in the two conditions.[6] Genetic involvement in OLP is yet to be determined.[7,8]

Several reports suggest an association of hepatitis C virus and human papilloma virus with OLP.[9,10] In Spain and Japan, there is a reported incidence of coinfection of 20% and 62%, respectively, with HCV; but this has not been shown in the American population.[11–13]

CLASSIFICATION

The classification of lichen planus is based on clinical presentation and is divided into 3 main

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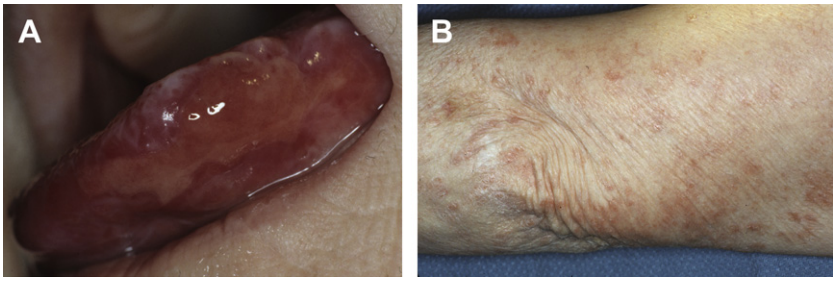

Fig. 1. (*A*) Superficial ulcer of erosive lichen planus on the lateral border of the tongue. Note atrophic appearance of the tongue. (*B*) Skin lesions on the extensor surface of the arm and forearm in the same patient as (*A*).

forms: reticular, erosive, and atrophic (or erythematous) lesions. Many other descriptors have also been used, including bullous, plaquelike, and papular. There is often overlap between types, with a combination of reticular, erosive, and erythematous lesions.

CLINICAL FEATURES

OLP predominately affects females, with most patients aged between 30 and 70 years.[11] It is a rare occurrence in children; but in men, lesions often develop at an earlier age. The presentation is varied in clinical appearance, with most lesions being bilateral and located on the buccal mucosa. Lesions can appear, however, on the tongue, in the vestibule, and on the gingivae. Isolated gingival lichen planus may be seen in up to 8.6% of patients.[12,13]

Malignant transformation of lichen planus is highly controversial.[14–16] The term *premalignant* implies eventual malignant transformation, but lichen planus may better be described as having "malignant potential."[16] There has been a reported incidence of 0.4% to 1.5% malignant transformation to squamous cell carcinoma in patients with lichen planus.[17] The World Health Organization's (WHO) criteria describe lichen planus as a condition predisposed to malignant transformation.[18]

Others have suggested that another entity, known as lichenoid dysplasia, is responsible for the conversion to malignancy. These lesions are dysplastic leukoplakias with a secondary lichenoid infiltrate but are often misdiagnosed as lichen planus.[15] It is also purported that patients with erosive lichen planus are more susceptible to known carcinogenic agents because of the lack of an epithelial barrier. Regardless, most investigators advocate periodic observation for dysplastic changes.

Reticular Lichen Planus

Reticular lichen planus is the most common type and is often found incidentally. Lesions are asymptomatic and located on the buccal mucosa, tongue, gingivae, or in the vestibule.[2] The lesions present as white, slightly raised plaques or papules with interlacing white lines described as Wickham striae on an erythematous background.

Erosive Lichen Planus

Erosive lichen planus appears atrophic, with areas of ulceration, erythema, and keratotic white striae. There can be pseudomembranes, and in the gingival region it often appears similar to desquamative gingivitis. There is a range of symptoms, from a mild burning sensation to debilitating pain. Lesions can interfere with speech, chewing, and

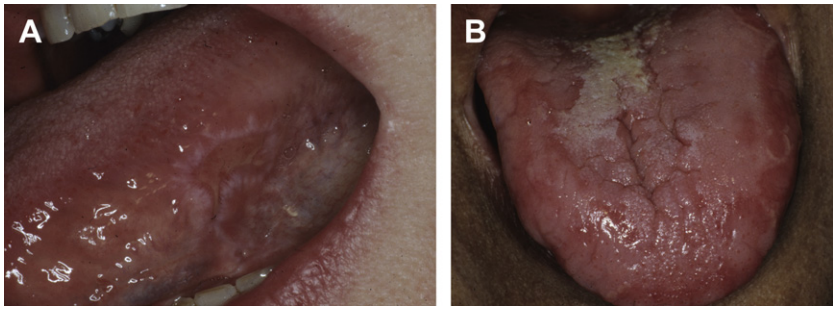

Fig. 2. (A) Superficial ulceration mimicking lichen planus in a patient taking nonsteroidal antiinflammatory medication. (*B*) Lichenoid lesions in a patient using nonsteroidal antiinflammatory medication. The lesions resolved after stopping the drug.