



Lichen planus and lichenoid reactions as a systemic disease

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Abstract Lichen planus (LP) is a chronic disease that involves the skin, scalp, mucous membranes, and nails. The etiology of LP is still unknown; however, some external and internal factors (eg. drugs, stress, hepatitis C virus) have been suggested to trigger the disease. Many studies have investigated an immunologic pathogenesis that is probably related to T-cell autoimmunity with the keratinocyte as the target cell. Altered self-antigens on the surface of basal keratinocytes modified by viruses or by drugs are believed to be the targets of the T-cell response. Various drugs and contact allergens like amalgam may cause lichenoid reactions, which are the main differential diagnoses of LP. Clinically and histologically, LP and lichenoid reactions cannot be distinguished with certainty in many cases. Treatment is mainly symptomatic and can be difficult. The first-line therapies for LP are topical or systemic corticosteroids; however, some studies have mentioned acitretin leading to similar improvement. Medical treatment, together with patient education and psychosocial support, can significantly benefit patients' quality of life. © 2015 Elsevier Inc. All rights reserved.

Introduction

Lichen planus (LP) is a chronic disease that involves the skin, mucous membranes, and nails. The exact prevalence of LP is unknown, but it is estimated to affect 0.9% to 1.2% of the general population.^{1,2} It commonly occurs in middle-aged women,^{3,4} whereas men tend to develop LP in their 30s.⁵ LP is unusual in children, and it is more common in the African-American population.⁶

Definition and clinical features

LP by definition is a chronic idiopathic inflammatory papulosquamous disease affecting skin, scalp, mucous

membranes, and nails. Cutaneous LP and oral LP are the most common presentations.⁷ LP lesions are described using the six Ps.⁸

- Planar
- Purple
- Polygonal
- Pruritic
- Papules
- Plaques

The disease is characterized by shiny, flat-topped, pruritic, and papulosquamous eruptions involving the extremities (the flexor surfaces of the wrists, forearms, and legs), genitalia, or oral cavity, where it presents differently, as will be described later.⁹ The cutaneous lesions are typically bilateral and symmetric. The lesions are often covered by whitish reticular lines, especially visible after application of oil, known as Wickham striae. The surrounding skin is normal; however, the morphology of LP may vary

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Fig. 1 Typical cutaneous lesions of lichen planus.



Fig. 3 Polygonal lichenoid papules in lichen planus with Koebner phenomenon after scratching.

widely. Cutaneous LP has different clinical subtypes, based on the morphology of the lesions, including classic, hypertrophic, vesiculobullous, actinic, annular, atrophic, linearly oriented, pigmented, and follicular presentations¹⁰ (Figures 1 and 2). The isomorphic response of Koebner is a common occurrence in LP. The Koebner phenomenon describes the development of new lesions in previously normal skin that has been traumatized either externally or internally¹¹ (Figure 3).

Mucosal LP more commonly affects the oral mucosa but may also involve the genital area. Oral LP has reticular, erosive, atrophic, papular, plaque-like, and bullous subtypes,¹⁰ with the most common type being the reticular pattern³ (Figure 4). The buccal mucosa is involved in 80% to 90% of oral LP lesions. In the oral mucosa, the Wickham striae are typically bilateral, symmetric, and asymptomatic.¹² Mechanical trauma of dental procedures, as well as cigarette smoking, can be Koebnerogenic factors that may exacerbate oral LP.¹³ Malignant transformation of oral and genital LP is possible.¹⁴

Nail lesions are possible and are more commonly seen in children.¹⁵ Nail LP can affect only one nail but usually affects several or most nails.¹⁶ Fingernails are more commonly involved than toenails.¹⁶ Clinically, the involved nails show longitudinal ridging and splitting (onychoschizia), longitudinal striation (onychorrhexis), nail absence (anonychia), subungual hyperkeratosis, and thinning nail plate (Figure 5).² Most nail changes result from involvement of the nail matrix.² LP of the nail bed causes onycholysis and subungual hyperkeratosis and may be very difficult to distinguish from nail psoriasis or onychomycosis.¹⁶

Lichen planopilaris (LPP) is a chronic cicatricial alopecia characterized by follicular hyperkeratosis, perifollicular

erythema, and loss of follicular orificies.¹⁷ LPP is most common in women aged 30 to 60 years.¹⁸ LPP can be subdivided into three groups: Classic LPP, frontal fibrosing alopecia, and Graham-Little syndrome.¹⁷ Classic LPP shows scalp hair involvement.¹⁷ Frontal fibrosing alopecia is characterized by cicatricial alopecia of the frontal and often the temporoparietal hairlines.¹⁷ Graham-Little syndrome is a rare variant of LPP characterized by multifocal cicatricial alopecia of scalp, noncicatricial alopecia of axillae and pubic region, and keratotic follicular papules over a body¹⁹ (Figure 6).

Lichenoid drug reactions (LDR) are the main differential diagnosis of LP. LDR are inflammatory lesions with varied etiologies, including immune-mediated disorders and reactions to systemic medications and to dental materials.²⁰ The term *lichenoid* refers to papular lesions of skin diseases of which LP is the prototype.²¹ There are no standardized criteria for the diagnosis of lichenoid reactions. The condition mimics LP but may have some eczematous elements.²¹ Lichenoid lesions are a common finding in the oral cavity, mostly on the buccal mucosa, tongue, and lips.²² In most cases, the lesions are indistinguishable from idiopathic LP, clinically or histologically.

LP and LDR have been reported to be associated with different kinds of disorders and triggers, such as stress, liver diseases and viral infections, drugs, and dental materials like amalgam.



Fig. 2 Bullous lichen planus.



Fig. 4 Erosive lichen planus mucosae.

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