

Oral Lichen Planus

Rafael Tomaz Gomes, Felipe Paiva Fonseca, João Figueira Scarini, and Lara Maria Alencar Ramos Innocentini

Lichen planus is a common disorder of the stratified squamous epithelium and is accepted as a chronic mucocutaneous disease of an autoimmune, T-cell-mediated nature. The disease affects approximately 1-2% of the general adult population and has a slight predilection for female patients (1.4:1.0) over the age of 40 years.

Clinically, it presents in a very varied manner, either through whitish streaks or papules, erythema, and erosion or even through blisters that rupture easily, usually with multifocal and symmetrical manifestations. Intraoral involvement occurs more frequently in the buccal mucosa, tongue, and gingiva. In some cases, simultaneous cutaneous manifestations may be observed, typically presenting as flattened polygonal, violaceous, pruritic papules, preferentially located on the wrists, ankles, and lumbar region. Nail involvement may be noted as the first manifestation of the disease with the formation of longitudinal grooves or ridges, pitting, darkening, and/or ungual dystrophy.

Although several hypotheses have been suggested, the etiology of lichen planus remains unknown, and there are several controversies regarding the pathogenesis of

R. T. Gomes (🖂)

F. P. Fonseca

J. F. Scarini

Department of Pathology, School of Medical Sciences, University of Campinas (FCM/ UNICAMP), Campinas, SP, Brazil

L. M. A. R. Innocentini

Department of Medicine, Universidade Federal de São Paulo (UNIFESP), São Paulo, SP, Brazil e-mail: rafael.tomaz@unifesp.br

Department of Oral Surgery and Pathology, School of Dentistry, Universidade Federal de Minas Gerais (UFMG), Belo Horizonte, MG, Brazil

Department of Oral Diagnosis, Piracicaba Dental School, University of Campinas (FOP/ UNICAMP), Piracicaba, SP, Brazil

Dentistry and Stomatology Division, Ophthalmology, Otolaryngology and Head and Neck Surgery Department, Clinical Hospital of Ribeirão Preto, School of Medicine, University of São Paulo (USP), Ribeirão Preto, SP, Brazil

[©] The Author(s), under exclusive license to Springer Nature Switzerland AG 2023 A. R. Santos-Silva et al. (eds.), *Clinical Decision-Making in Oral Medicine*, https://doi.org/10.1007/978-3-031-14945-0_17

this disease. However, a wide range of evidence points to an important role played by immune system dysregulation, and thus, different mechanisms have been considered, including (1) cellular immune response to specific antigens, (2) nonspecific mechanisms, (3) autoimmune response, and (4) humoral immunity.

1 Clinical Characteristics

- Usually, adults over 40 years of age.
- The predominance in the female gender.
- Buccal mucosa, tongue, and gingiva are the most affected sites.
- Variable clinical presentation, with six distinct patterns, described below.

1.1 Reticular

- Presence of whitish reticulate striations (Wickham's striations) (Figs. 1 and 2).
- Minimal symptomatology in most cases.

Fig. 1 Reticular white lines (Wickham's striae) involving the buccal mucosa





Fig. 2 Reticular lichen planus involving the vermilion of the lower lip and the lateral surface of the tongue

1.2 Papular

- Rare and often neglected variant.
- Small (0.5–1 mm) elevated white papules with fine white streaks at the periphery of the lesion.
- It usually coexists with another variant.

1.3 Plaque-Like

- Homogeneous white plaques resembling leukoplakia.
- Presence of white striae on the surface.
- It generally affects the dorsum of the tongue and oral mucosa.
- Lesions may appear multifocal.

1.4 Erosive

- Ulcerated central area.
- Presence of a fibrin network or pseudomembrane over the ulcer.
- Pain when chewing or swallowing.

1.5 Atrophic

- Erythematous areas with very fine white streaks on the surface.
- Burning, burning sensation, and sensitivity.

1.6 Bullous

- Rare variant.
- Presence of blisters a few millimeters to centimeters.
- The blisters break rapidly, causing painful erosions or ulcers.
- The buccal mucosa is the most affected in this variant.

2 Diagnosis

2.1 Clinical Evaluation

- Investigate the presence of dental materials near lesions.
- Check for recent or current use of medication (non-steroidal anti-inflammatory drugs, antihypertensives, hypoglycemic agents, or antimalarials).
- Search for other underlying diseases (graft versus host disease, liver disease, or HIV).

2.2 Incisional Biopsy

• Histopathological findings are unspecific, however, essential for a proper clinicopathological correlation and include hyperkeratosis, irregular "sawtooth" appearance suggestive of acanthosis, basal layer liquefaction, and lymphocytic banded infiltrate (lichenoid pattern).

3 Treatment

- Asymptomatic lesions: Patient education and clinical follow-up.
- Lesions associated with an underlying cause: management of the systemic condition.
- Symptomatic lesions:
 - Clobetasol propionate 0.05%, topical, two times a day, during the symptomatic period of the lesion, or for a maximum of 2 months (See Chap. 8) (Fig. 3).
 Apply on dry mucosa and do not ingest drinks or food for 30 min.
 - Triamcinolone acetate 0.1%, topical, three times daily for up to 3 months, two times daily in the fourth month, one time daily in the fifth month, and applications on alternate days in the sixth month.
 - Cyclosporin A 1.5%, topical in hydroxyethyl cellulose gel, two times daily, for up to 2 months.
 - Prednisone 0.5–1.0 mg/kg per day in the morning. Prolonged treatments require progressive reduction of the daily dose according to clinical response, suggesting medical follow-up.
 - Antifungal prophylaxis with topical miconazole, 1 time daily for 14 days, or chlorhexidine 0.12% (alcohol-free); oral rinses 3 times daily for 7 days.

Side effects of prolonged use of systemic corticotherapy

- 1. Cushingoid appearance
- 2. Hypertension
- 3. Tachycardia
- 4. Fluid retention
- 5. Hyperglycemia
- 6. Dyslipidemia
- 7. Opportunistic fungal infections
- 8. Corticosteroid induced myopathy
- 9. Osteopenia and osteoporosis
- 10. Thromboembolic complications
- During treatment, assess the following:
 - Weight gain or loss
 - Blood pressure
 - Fasting blood glucose
 - Serum electrolytes
 - Serum urea and creatinine
 - Liver function



Fig. 3 Use of individual trays with clobetasol

Acknowledgments The São Paulo State Research Foundation (FAPESP, São Paulo, Brazil, grant number JFS 19/09419-0) and the Coordination of Training of Higher Education Graduate Foundation (CAPES, Brasilia, Brazil, finance code 001).

Sources

- Carrozzo M, Porter S, Mercadante V, Fedele S. Oral lichen planus: a disease or a spectrum of tissue reactions? Types, causes, diagnostic algorhythms, prognosis, management strategies. Periodontol. 2000;2019(80):105–25.
- Daniel BS, Murrell DF. Review of autoimmune blistering diseases. J Eur Acad Dermatol Venereol. 2019;33:1685–94.
- Malhotra AK, Khaitan BK, Sethuraman G, Sharma VK. Betamethasone oral mini-pulse therapy compared with topical triamcinolone acetonide (0.1%) paste in oral lichen planus: a randomized comparative study. J Am Acad Dermatol. 2008;58:596–602.
- Proliferative verrucous leukoplakia may initially mimic lichenoid reactions. World J Clin Cases. 2015;3(10);861–3. https://doi.org/10.12998/wjcc.v3.i10.861.
- Roopashree MR, Gondhalekar RV, Shashikanth MC, George J, Thippeswamy SH, Shukla A. Pathogenesis of oral lichen planus: a review. J Oral Pathol Med 2010;39:729–734.
- Zhang J, Zhou G, Du GF, Xu XY, Zhou HM. Biologics, an alternative therapeutic approach for oral lichen planus. J Oral Pathol Med. 2011;40:521–4.