Oral lichen planus.

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The cause of lichen planus is not known. Its treatment depends on the specific type—reticular, erosive, or bullous—and usually includes topical or systemic corticosteroids and topical antifungal agents. Patients require lifelong monitoring and/or therapy.

Lichen planus (LP) is a chronic, self-limited, inflammatory disorder of unknown etiology that involves mucous membranes, skin, nails, and hair. It is postulated that there is an abnormal T-cell-mediated immune response that results in disruption of the basement membrane. Several drugs are known to be associated with the onset of LP, but the exact mechanism is unknown.

LP develops in about 1 to 2% of the general population, with a peak in middle-aged adults and with women affected more often than men (3:2). Three major types are recognized in the oral cavity: reticular, erosive, and bullous.

Reticular LP is usually asymptomatic, affects multiple sites, and can be recognized by white papules that can coalesce to form plaques. There may be fine, white, lace-like striae (Wickham striae) on the buccal mucosa, gingiva, and lips. Cutaneous LP may be seen in up to 44% of patients with oral LP. Erosive LP usually presents with pain while eating, especially with spicy foods. There is usually atrophic, erythematous mucosa with ulcerations. Bullous LP is uncommon, resulting in bullae formation with epithelial separation. This type may show a positive Nikolsky sign.

Treatment varies depending on the specific type of LP, usually including topical or systemic corticosteroids and topical antifungal agents. Symptoms usually come and go over the patient's lifetime, requiring lifelong therapy or monitoring after the initial presentation.

Histologically, the lesions usually show both atrophy and acanthosis of the squamous epithelium, with variable degrees of both ortho- and parakeratosis. The classic appearance is a "sawtooth" pattern to the rete, with a hydropic degeneration of the basal layer (figures 1 and 2). A rich, band-like, predominantly T-cell lymphocytic infiltrate results in blurring of the epithelial-to-stromal junction.
Plasma cells may also be seen. Isolated, degenerated keratinocytes (Civatte, or hyaline, bodies) are present at the epithelial-stromal junction (figures 2 and 3). Erosive LP may show ulceration or a sub-basal separation of the epithelium from the stroma. It is not uncommon to have a secondary, superimposed candidiasis. Direct immunofluorescence of perilesional tissue may show linear or granular deposits of fibrin or fibrinogen (figure 2). Importantly, there is no dysplasia, although reactive atypia may be present.

Figure 3. High-power photograph shows disruption of the junctional zone, with bright pink Civatte bodies (arrow) at the basal zone. Note the inflammatory cells throughout.

The pathology differential diagnosis for lichen planus includes mucous membrane pemphigoid, pemphigus vulgaris, lichenoid reaction to drugs, lupus erythematosus, chronic graft-versus-host disease, linear IgA disease, and cinnamon-induced stomatitis.

Figure 2. A: This view shows dissolution and hydropic change at the junction between the epithelium and stroma, with inflammatory cells filling the dermis and sprinkled throughout the epithelium. Civatte bodies are noted (arrow). B: Granular deposition of fibrinogen is seen by immunofluorescence.
Figure 3. High-power photograph shows disruption of the junctional zone, with bright pink Civatte bodies (arrow) at the basal zone. Note the inflammatory cells throughout.

**Suggested reading**


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