

ORAL LICHEN PLANUS

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Lichen planus is an immune-mediated disease involving skin and mucosa. Diverse knowledge is essential for diagnosing this case because of its potential for malignant transformation. This article reports the case of oral lichen planus (OLP) along with the pathogenesis and importance of inflammatory infiltrate in the diagnosis of OLP and oral lichenoid dysplasia (OLD).

A 29-year-old male patient from Visakhapatnam reported to the hospital with the chief complaint of a burning sensation in the mouth for 3 years. Its intensity had increased since 1 month. The patient also complained of frequent dryness of mouth and bad breath. On general examination, scaly patches and macules were revealed on the scalp and both the knees respectively. Erythematous and edematous swelling of labial gingiva which was soft in consistency was observed in the maxilla and mandible. Generalized recessions and bleeding on probing were noted along with poor oral hygiene status. No restorations are evident and the patient was not taking any medication. Based on this background, atrophic lichen planus was considered a provisional diagnosis by surgeons. An incisional biopsy was sent to the Oral Pathology department. Under microscopy, the hematoxylin and eosin-stained sections revealed saw tooth rete ridges and lymphohistiocytic infiltrate at the epithelial connective tissue interface. Vacuolar degeneration of basal keratinocytes was observed along with clusters of homogenous eosinophilic ovoid bodies at the basement membrane zone resembling Civatte bodies. Mast cells were also observed in the inflammatory infiltrate (Fig1).



The case was diagnosed as “ORAL LICHEN PLANUS”, considering clinical and histological correlations.

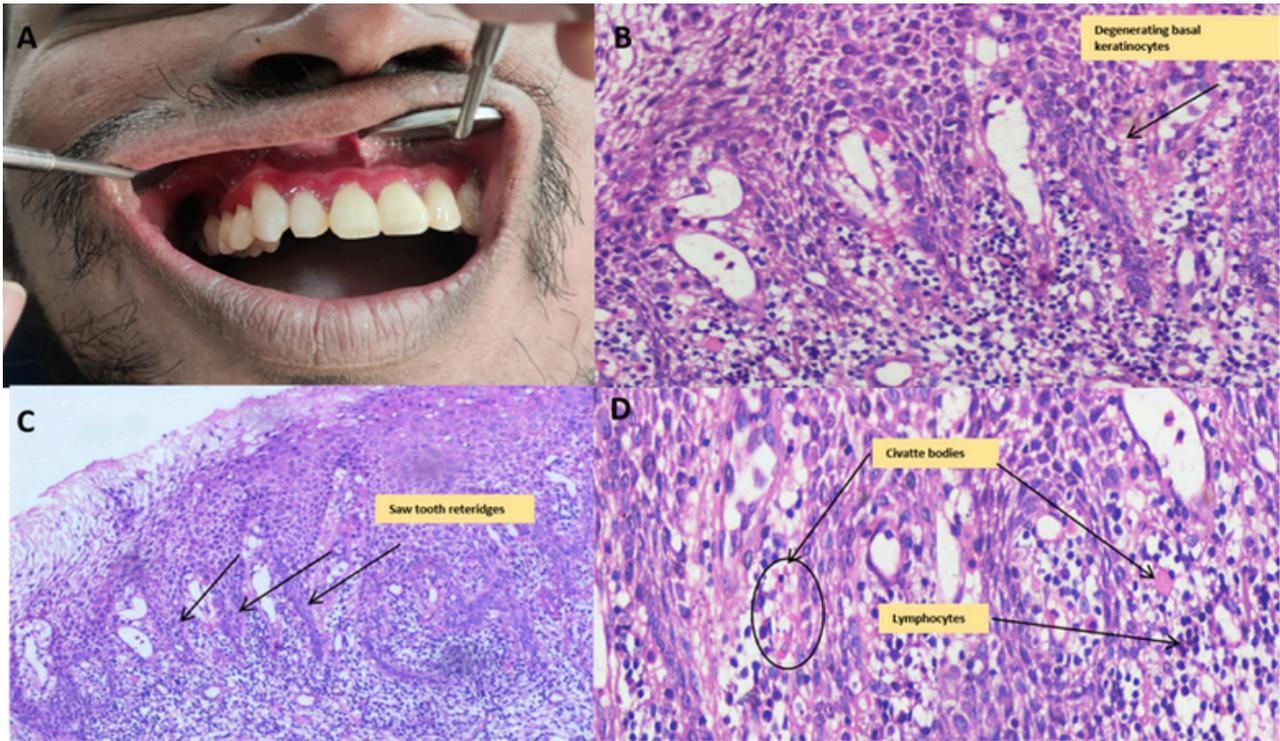
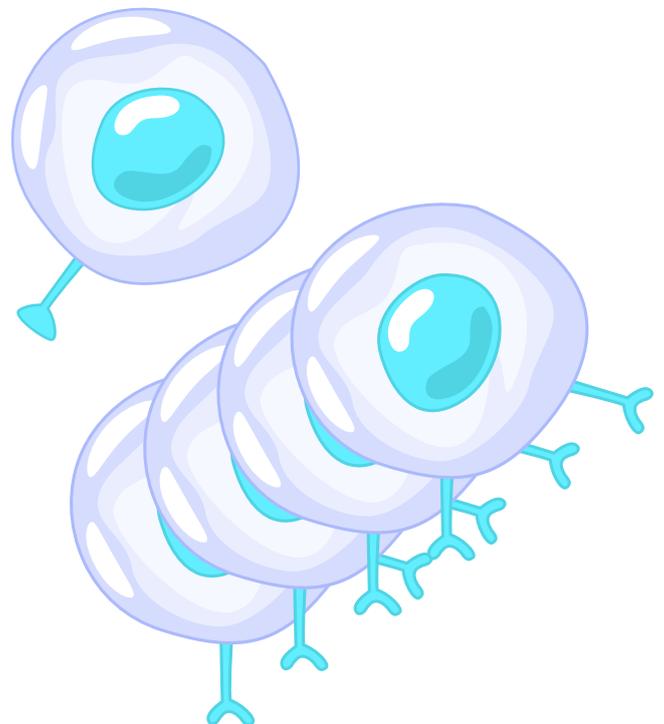


Fig 1: A) Erythematous lesion on the gingiva. H&E stained tissue sections showing B) degenerating basal keratinocytes (40x), C) saw tooth rete ridges (10x), and D) Civatte bodies and lymphocytic infiltrate (40x).

Cell-mediated immunity plays a major role in its pathogenesis. CD8+ cells are the cause of the initiation and progression of the lesion (Fig 2).¹

Many differentiating features of OLP and OLD are reported in the literature. Considering the type of cells in the inflammatory infiltrate makes the differentiation quite meaningful as they reflect the pathogenesis of the lesion. The Lichenoid immune response is characterized by lymphohistiocytic infiltrate which is seen in OLP. Even though OLP is predominantly T cell-mediated, if there are a lot of plasma cells in the infiltrate, search for dysplastic characteristics in the epithelium. If dysplastic features are evident then the case should be diagnosed as “Oral epithelial dysplasia” rather than Lichenoid dysplasia. Hence, clinical correlation is important.^{2,3}

A thorough inspection is crucial while diagnosing cases with lichenoid immune reactions.



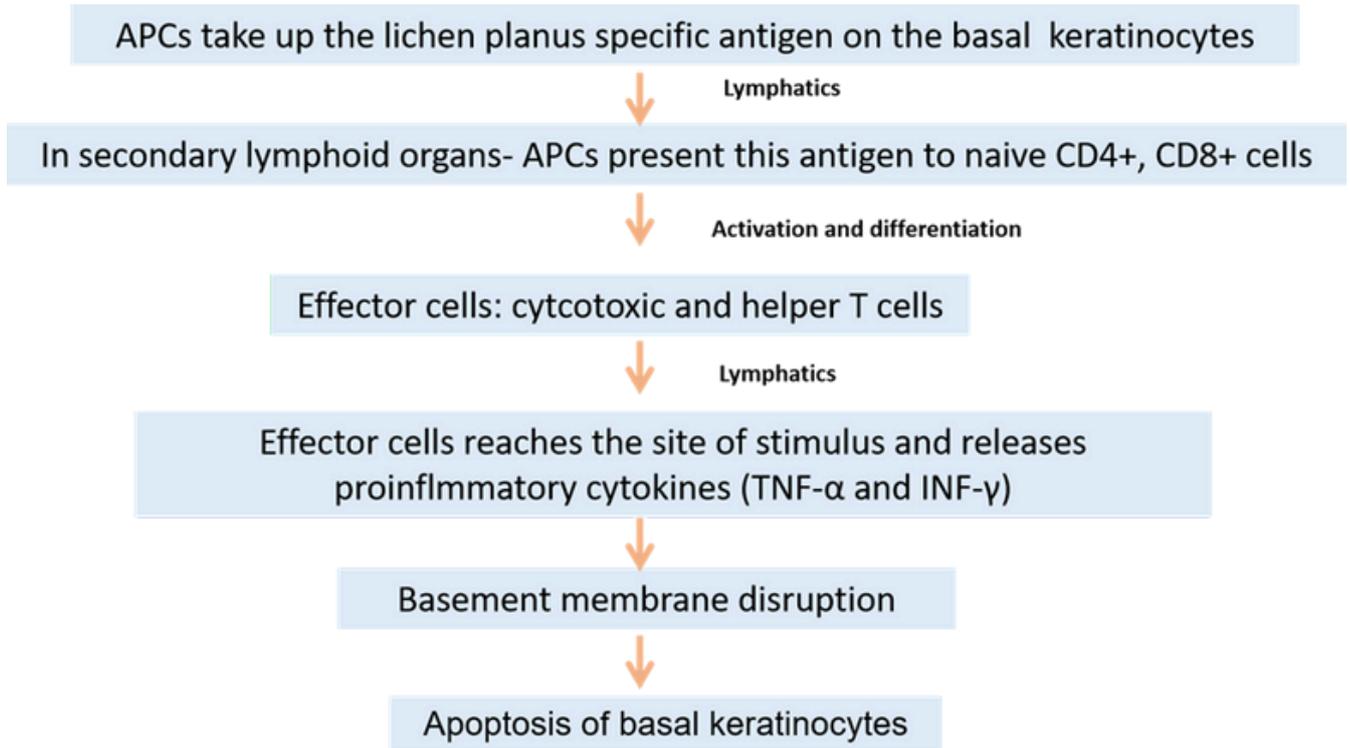


Fig 2: Pathogenesis of lichen planus: APCs present the lichen planus-specific self-antigen on the basal keratinocytes to the naive CD4+ and CD8+ cells. Post-activation, these cells proliferate and differentiate into effector cytotoxic T cells and helper T cells. These cells release many proinflammatory cytokines, among them, the most important roles are played by TNF- α and INF- γ . They disrupt the basement membrane via the increased transcription of MMP9 protein and pave the way for basal keratinocyte apoptosis.

APC: Antigen-presenting cell, TNF- α : Tumor necrosis factor-alpha, INF- γ : Interferon-gamma

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