

Correlating the Histopathological Features with the Underlying Pathogenesis could Aid in Differentiating Oral Lichen Planus from Related Lesions

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Oral lichen planus (OLP) is considered as a chronic immune-mediated inflammatory condition of the oral mucosa. It has a variety of clinical presentations including reticular, erosive, papular, plaque-like, and ulcerative patterns. The major histopathological features include a subepithelial band of inflammatory infiltrates and basal cell degeneration. Examination of the subepithelial inflammation has revealed the predominance of CD8+ lymphocytes.¹ The recruitment of CD8+ cells occurs as a response to the antigenic stimuli of the oral epithelial keratinocytes. The cytotoxic activity of the CD8+ cells induces apoptosis of the antigenic keratinocytes.² Keratinocytes are responsible for maintaining the basement membrane and *vice versa*.^{3,4} Thus, apoptotic loss of keratinocytes results in loss of basement membrane integrity which in turn triggers further keratinocyte apoptosis. In addition, the loss of basement membrane integrity allows the migration of a greater number of CD8+ cells triggering large-scale keratinocyte degeneration. With each CD8+ cell attempting to breach through the basement membrane to reach its target antigen, there is an added accumulation of the CD8+ cell in the subepithelial stroma, eliciting the classic appearance of the subepithelial inflammatory band with varying degrees of keratinocyte degeneration.

Although histopathological examination reveals only the above-mentioned characteristic features, a number of other inflammatory mediators have also been identified in OLP using various diagnostic modalities including immunohistochemistry.⁵ These mediators are attributed to be the result of the wide variety of chemokines released by the apoptotic keratinocytes and the CD8+ cells. The chemokines attract additional inflammatory components that sustain the lesion, rendering a chronic course. Cases presenting with these additional inflammatory components are often misdiagnosed as oral lichenoid lesions (OLLs) and oral epithelial dysplasia with secondary lichenoid components. The latter is also referred to as oral lichenoid dysplasia (OLD) as the epithelial dysplasia is the primary pathology and the inflammation is just a reactionary component. Both OLL and OLD have shown to exhibit clinicopathological features similar to those of OLP.⁶⁻¹¹ In OLL, the antigenic stimuli are from an external factor, which could range from a restorative material to a wide range of medications. In cases of OLD, as mentioned earlier, the inflammatory components are a response to epithelial dysplasia which in turn is often due to a known risk factor such as tobacco/alcohol.⁶⁻¹¹

Unlike OLL and OLD, details of the triggering/risk factor cannot be elicited through history for OLP. Further, OLP cases could potentially have a coincidental history of oral restorations, a recent change in medications, or a known carcinogenic risk factor such as

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tobacco/alcohol.^{7,8} In such cases, if the patch test for OLL is negative and if the cessation of the associated factor does not resolve the lesion, then eliciting the self-antigen in the keratinocyte would be an objective confirmation of the disease nature. To conclude, correlating the histopathological features with the underlying pathogenesis (autoimmune response) could aid in understanding the natural history of OLP and in differentiating them from related lesions (OLL and OLD).

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