
VLC Media Player All Skin Collection 2011 By SuDHiR Chauhan [PORTABLE]

Lichen planus (LP) is a relatively common autoimmune T-cell-mediated disease of unknown aetiology affecting the mucous membranes, skin and nails. Its prevalence varies between 0.5 and 2.2% of the population in epidemiological studies with a peak incidence in the 30-60 years range and with a female predominance of 2:1. Mucosal lichen planus tends to follow a chronic course with acute exacerbations. Spontaneous remission of oral lichen planus (OLP) is uncommon, and indeed mucosal LP may become worse with time. In contrast, cutaneous lichen planus may follow a milder clinical course though some variants may be severe such as those affecting the palms and soles and the scalp and the genital tract in females (vulvovaginal gingival LP) where scarring leads to significant complications.

It is important to identify those cases that may be drug induced or be associated with a contact allergic or irritant reaction (lichenoid reaction) or the rarer oral presentation of discoid lupus erythematosus. There is a very small risk of malignancy (approximately 1:200 patients/year) associated with oral lichen planus; thus patients should be informed that long term monitoring via their general dental practitioner is appropriate. This review will focus on the clinical presentation and management of oral lichen planus.

Lichen planus (LP) is a relatively common autoimmune T-cell-mediated disease of unknown aetiology affecting the mucous membranes, skin and nails. Its prevalence varies between 0.5 and 2.2% of the population in epidemiological studies with a peak incidence in the 30-60 years range and with a female predominance of 2:1. Mucosal lichen planus tends to follow a chronic course with acute exacerbations. Spontaneous remission of oral lichen planus (OLP) is uncommon, and indeed mucosal LP may become worse with time.

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The biological basis of lichen planus is unknown. Clinical features of lichen planus are presence of inflammatory lesions and hyperkeratosis in skin. The pathogenesis and pathogenetic mechanisms are elusive. The inflammatory cells are the key players in the progress of lichen planus. In lichen planus, cytokines play a significant role in the keratinocyte activation and may be responsible for the clinical manifestation of lesions. In recent years, there have been many reports on a connection between inflammation and lichen planus. IFN-g may be involved in the pathogenesis of lichen planus. It is a pleiotropic cytokine, with some anti- and pro-inflammatory properties. It can regulate activation, proliferation and function of many cells and is involved in the immune response against microorganisms. In this study, the mRNA expression of IFN-g and IFN-g receptor were studied in skin biopsies of 20 patients with lichen planus and 20 healthy controls. The mRNA expression of IFN-g and IFN-g receptor were higher in the lesional skin of lichen planus patients. In conclusion, lichen planus is a T-cell-mediated autoimmune disease associated with a Th1 T-cell immune response, in which IFN-g plays a pivotal role in the pathogenesis. IFN-g may be a therapeutic target for lichen planus. PMID:27051651 Lichen planus, an autoimmune and inflammatory mucocutaneous disorder, is a T cell-mediated inflammatory process.

The etiology and pathogenesis of LP is not fully understood, and the exact pathogenetic mechanism by which T cells induce skin lesions is not well known. Numerous factors have been investigated for their role in the development of lichen planus. It was suggested that some of them may act on T cells which in turn can stimulate the production of additional cytokines which are key mediators in the pathogenesis of LP. This review focuses on the role of IL-17 in the pathogenesis of LP and how the treatment of lichen planus with anti-IL-17 agents could be useful.

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