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Oral Lichen Planus - A Comprehensive Review

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Abstract

Oral lichen planus is an immunologically based, chronic, inflammatory, mucocutaneous disorder of undetermined etiology. It is a relatively common disorder affecting stratified squamous epithelia. It is of special importance due to its malignant potential and can be a source of morbidity. The management of oral lichen planus should therefore address both the transformation rate as well as the patient symptoms. The aim of present review of literature is to discuss the etiology, clinical feature, management of oral lichen planus in detail.

Keywords: Oral lichen planus, OLP, Management of oral lichen planus

Introduction

Oral lichen planus (OLP) is a chronic, autoimmune mucocutaneous disease, occurring most commonly in the middle-aged women. Lichen planus may also occur concurrently or independently in the skin and the genital, anal, esophageal, nasal and laryngeal mucosae. The prevalence of oral lichen planus in general population varies from 1-2%.[1] There is no racial predilection, and the disease appears to be pan racial. Andreasen reported that the average age of occurrence in males and females

is 40-49 years and 50- 59 years, respectively.[2] However, few cases have been reported in children as young as 6 months.[3]

Lichen Planus, discovered by Erasmus Wilson in 18691, is a chronic mucocutaneous disease primarily occurring due to an autoimmune mechanism. There are various mechanisms to define the pathogenesis of oral lichen planus but the broader aspect of it lies in the T-cell mediated delayed Type-4 hypersensitivity reaction, which is antigen specific. Pathogenesis of oral lichen planus is very elusive for researchers and clinicians, to clearly explain how several factors interact and are responsible for initiation, aggravation, and chronicity of oral lichen planus.[4,5] Hence, the aim of present review of literature is to discuss the etiology, clinical feature, management of oral lichen planus in detail.

Etiology: The etiology of OLP appears to be multifactorial and complicated.

Table no. 1: Etiology of Oral Lichen Planus

Factors	Interpretation
Genetic factor	Familial cases are rare. An association has been observed with HLA-A3, A11, A26, A28, B3, B5, B7, B8, DR1, and DRW9. In Chinese patients, an increase in HLA-DR9 and Te 22 antigens has also been noted.[6,7]
Dental material	Many materials commonly used in restoration treatments in the oral cavity have been identified as triggering elements for OLP, including silver amalgam, gold, cobalt, palladium, chromium and even non-metals such as epoxy resins (composite) and prolonged use of denture wear.[8,9]
Microbial agent	OLP has been suggested to be related to bacteria such as a Gram-negative anaerobic bacillus and spirochetes but this has not been confirmed. Some of the studies reveal the role of Helicobacter pylori (HP) in the etiology of OLP.
Autoimmunity	OLP may occasionally be associated with autoimmune disorders such as primary biliary cirrhosis, chronic active hepatitis, ulcerative colitis, myasthenia gravis, and thymoma.[10]
Stress	One of the factors responsible for the development of OLP is anxiety and stress. Some of the studies in literature reveal the role of the psychological stress in the etiology of OLP.[11]
Food	Food and some of food additives such as cinnamon aldehyde have been found to be associated with OLP.[12]
Malignant neoplasms	LP has been observed on the skin and/or mucosa of patients affected by a range of different neoplasms such as with breast cancer and metastatic adenocarcinoma.[13]

Pathogenesis

Although the pathogenesis of LP is not fully understood, there is strong evidence that the disease development involves an imbalance of immunologic cellular reactivity.

OLP is a T-cell mediated autoimmune disease in which the auto-cytotoxic CD8 + T cells trigger apoptosis of the basal cells of the oral epithelium. Current literature suggests that OLP is caused by cluster of differentiation 8 (CD-8) cell mediated damage to the basal keratinocytes leading to apoptosis.

An early event in the disease mechanism involves keratinocyte antigen expression or unmasking of an antigen that may be a self-peptide or a heat shock protein. Following this, T cells (mostly CD8+, and some CD4 + cells) migrate into the epithelium either due to random encounter of antigen during routine surveillance or a chemokine-mediated migration toward basal keratinocytes. These migrated CD8 + cells are activated directly by an antigen binding to major histocompatibility complex (MHC)-1 on keratinocyte or through activated CD4 + lymphocytes. In addition, the number of Langerhans cells in OLP lesions is increased along with upregulation of MHC-II expression; subsequent antigen presentation to CD4 + cells and interleukin (IL)-12 activates CD4 + T helper cells which activate CD8 + T cells through receptor interaction, interferon γ (INF-γ) and IL-2. The activated CD8 + T cells in turn kill the basal keratinocytes through tumor necrosis factor (TNF)-α, Fas-FasL-mediated or granzyme B-activated apoptosis.[14,15]

Clinical Feature

The clinical presentation of OLP varies. In some patients the onset is insidious, and patients are unaware of their oral condition. Some patients report sensitivity of oral mucosa to hot and spicy food, painful oral mucosa, red or white patches of the oral mucosa, or oral ulcerations. OLP can occur on any mucosal surface, including the lips, but most frequently occurs on the buccal mucosa, the tongue being common site.

Oral lichen planus can be present as small, raised, white lacy lesions, papules or plaques and can resemble keratotic diseases such as leukoplakia. Atrophic lesions and erosions can cause pain. Erythematous lesions that affect gingiva can cause desquamative gingivitis. It is the most common type of lichen planus. They are also presented as small, raised, white lacy papules and resemble leukoplakia or frictional keratosis.[17]

The lesions on the palate, floor of the mouth and upper lip are uncommon. In rare cases, white lesions which cannot be seen in erosive or ulcerated forms, they are difficult to differentiate clinically from other vesiculobullous lesions such as pemphigus and pemphigoid. Squamous Cell Carcinoma is the malignant transformation of oral lichen planus. Other lesions of Oral Lichen Planus that resemble clinically and histologically are oral lichenoid reactions.[18]

In 1968, Andreasen divided OLP into 6 clinical forms: reticular, papular, plaque like, atrophic, erosive and bullous. These forms may present either simultaneously or individually. Based on the predominant clinical morphology it will be labelled as specific form and the predominant morphology may change over time. Older individuals usually presents with more severe forms (erythematous/ atrophic, erosive).[2]

Diagnosis

Various diagnostic features have been described over the years. It must be noted that diagnosis of OLP cannot be made on strictly clinical or histological grounds alone. A combined clinical and histopathological investigation is required for making a diagnosis. The widely used definition for the diagnosis of OLP was the criteria introduced by World Health Organization (WHO) in 1978.

Table no. 2: World Health Organization Diagnostic Criteria of OLP (1978)

Clinical criteria	Histopathologic criteria
Presence of white papule, reticular, annular, plaque-type lesions, gray-white lines radiating from the	Normally keratinized mucosa, and if site normally is nonkeratinized this layer may be very

papules. Presence of a lace-like network of slightly raised gray-white lines (reticular pattern). Presence of atrophic lesions with or without erosion, and also in the form of bullae.	thin. Presence of civatte bodies in basal layer, epithelium and superficial part of the connective tissue. Presence of a well-defined band like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes. Signs of 'liquefaction degeneration' in the basal cell layer.

Differential diagnosis

The differential diagnosis of erosive OLP includes squamous cell carcinoma, discoid lupus erythematosus, chronic candidiasis, benign mucous membrane pemphigoid, pemphigus vulgaris, chronic cheek chewing, lichenoid reaction to dental amalgam or drugs, graft-versus-host disease (GVHD), hypersensitivity mucositis and erythema multiforme. The plaque form of reticular OLP can resemble oral leukoplakia.[19]

Management

In all OLP patients, it is important to remove local exacerbating factors. The teeth should be scaled to remove plaque and calculus, and the patient should be instructed in thorough oral hygiene. Teeth associated with oral lesions should be examined and sharp cusps or edges reduced. Dental restorations associated with oral lesions should be mirror polished or replaced if contact sensitivity is suspected.[20]

A drug history should be obtained to identify reversible causes of lichenoid eruptions as discontinuation of the offending agent is often curative. Patients with OLP who are elderly and have poor nutrition could have iron deficiency, even when they are not found to be anemic when screened.[21]

Patients with oral LP are managed with medications that were neither developed nor intended for oral diseases and, consequently, most lack adequate efficacy studies. Thus, such factors as optimal dose, duration of treatment, safety, and true efficacy remain unknown. The most commonly employed and useful agents for the treatment of LP are topical corticosteroids. A response to treatment with mid potency corticosteroids such as triamcinolone, potent fluorinated corticosteroids such as fluocinolone acetonide, fluocinonide and super potent halogenated corticosteroids such as clobetasol has been reported in 30-100% of treated patients. The greatest obstacle in using topical corticosteroids in the mouth is the lack of adherence to the mucosa for a sufficient length of time. For this reason, some investigators prefer using topical corticosteroids in adhesive pastes although there is no data that topical steroids in adhesive bases are more effective than as base preparations.[22]

Topical corticosteroids are of limited value for some cases of oral lichen planus. In such cases, it may be appropriate to use topical corticosteroids in combination with intra lesional preparations. They are used for recalcitrant or extensive lesions involve the subcutaneous injection of 0.2-0.4 mL of an l0 mg/mL solution of triamcinolone acetonide. However, intralesional corticosteroids have some contra indications, including atrophy of tissue and secondary candidiasis after frequent injections.[16]

Patients who exhibit desquamative gingivitis, wide spread oral disease, or diffuse ulcerations, may not respond adequately to topical corticosteroids alone. The

addition of potent immunosuppressants or immuno-modulatory agents such as cyclosporine, tacrolimus, tretinoin, in topical formulations, may be beneficial in this group of patients.[23]

Lycopene is a fat-soluble carotenoid. It has antioxidant activity, also acts by inhibition of cancer cell proliferation and interference with growth factor stimulation. It has shown to be effective in the management of oral leukoplakia and in chemoprevention of oral cancer. Supplementing with 8 mg/d of lycopene for 8 week showed favorable results of reduced burning sensation and decreased signs and symptoms of OLP.[24]

Photodynamic therapy (PDT) uses a photosensitizing compound like methylene blue which is activated at a specific wavelength of laser light. It is known to destroy the targeted cell via strong oxidizers, leading to membrane lysis, cellular damage, and protein inactivation. PDT has shown positive results in management of head and neck tumors. PDT have immunomodulatory properties which may induce apoptosis in the hyper proliferating inflammatory cells present in diseases like psoriasis and oral lichen planus, there by reversing the hyper proliferation and inflammation of oral lichen planus.

Conventional Surgical treatment can be used with more ease in the cases where there is a plaque like lesions, but in cases of erosive and atrophic it is not recommended. Apart from Conventional Surgical treatment carbon dioxide laser and cryosurgery also have been recommended.[16]

Conclusion

Oral Lichen planus (OLP) is a common chronic mucocutaneous disorder with an immune mediated pathogenesis. Its appearance may vary from presence of keratotic to erythematous areas. Etiology of OLP is unknown, but it is thought to be the result of an autoimmune process with an unknown predisposing factor. Management of lichen planus can be challenging and discouraging for both the patient and physician. Treatment options should be assessed for any risks and benefits and analyse the severity of the disease.

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