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Oral Lichen Planus – A Review

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Abstract:

Lichen Planus is defined as the chronic inflammatory disorder affecting the skin, mucus membrane, nail and scalp. It can be clinically classified as Reticular, atropic, plaque like, bullous and erosive type. Erosive lichen planus has increased chance of malignant transformation.It frequently affects women more than men. The pathogenesis of Oral lichen planusis by unmasking the antigen of heat shock protein or self-antigen. These antigens presented to the T cells causes apoptosis of the basal cells of the epithelium. More CD4 and CD8 T cells migrate to the site. This migration is caused by the antigen binding to the major histocompatibility complex [MCH –I]. MCH- II expression increases the langerhancells in the lesion. Increased antigen presentation activates CD4 and CD8 cells and these cells in turn causes killing of basal keratinocytes and apoptosis. This causes degeneration of the basement membrane and lymphocytic infiltration of the connective tissue adjacent to the basement membrane. The management of oral lichen planus include corticosteroids [triamcinolone, prednisone, clobetasol etc], calciurin inhibitors (tacrolimus, cyclosporine) etc.

Keywords : oral lichen planus, chronic, review, etiology ,disease

INTRODUCTION:

Lichen planus was first described by Erasmus Wilson in 1869.Lichen planus is a chronic inflammatory dermatologic lesion with characteristic oral mucosal changes.[1]These dermal lesions appear as polygonal pruritic papules on the skin which rupture and form whitish lesion. On healing, it produces scar. In skin, the lesion exhibits Koebner's phenomena. Oral lichen planus is associated with burning sensation and the lesion appears in different forms clinically like reticular, papular, atrophic, erythematous and bullous types.[2]Erosive lichen planus seems to have an association with chronic liver disease and erosive lichen planus is considered as a potentially malignant disorder.[3]

Microscopically the disease is characterized by dense subepithelial infiltrate, increased numbers of intraepithelial lymphocytes and degeneration of basal keratinocytes.[4] Degenerating basal keratinocytes form colloid (civatte, hyaline, cytoid) bodies that appear as homogenous eosinophilic globules. Epithelial basement membrane changes are common in oral lichen planusand include breaks, branches and duplications. Degeneration of basement membrane causes weaknesses at the epithelial-connective tissue interface which may result in histological cleft formation and clinical blistering of the oral mucosa (bullous lichen planus) is seen rarely.Parakeratosis, acanthosis and 'saw-tooth' retepeg formation may be seen.[5]

ETIOLOGY:

The etiology of oral lichen planus is unknown.[6]Several factors have been proposed for the etiology including autoimmunity – associated with other autoimmune diseases ,dental materials ,drugs, infectious agents – bacterial and viral infections, , immunodeficiency, food allergies, stress, habits, trauma, diabetes and hypertension , malignant neoplasms and bowel disease.[7]Also the various mechanisms involved in the immunopathogenesis are antigen-specificcell-mediated immune response,non-specific mechanisms,autoimmune response,humoral immunity[8]. In antigen specific cell mediated response , there is migration of T lymphocytes into epithelium .This causes activation of T lymphocytes and Non specific mechanisms like mast cell degranulation and MMP -1 activation further aggravate the T cell accumulation, basement membrane disruption by mast cell proteases and keratinocyte apoptosis (triggered by basement membrane disruption). therefore killing the T lymphocytes.[9]

In non specific mechanism, mast cell degranulation and MMP -1 activation further aggravate the T cell accumulation, basement membrane disruption by mast cell proteases and keratinocyte apoptosis.

In auto immune response , there is deficiency of TGF-β1.This is followed by breakdown of immune privilege , keratinocyte apoptosis and langerhan cell maturation. TGF - β1 deficiency may predispose to autoimmune lymphocytic inflammation.

Humoral immunity is provided by circulating antibodies including autoantibodies against desmoglein 1 and 3.[10]

CLINICAL FEATURES:

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.The most common site affected

are the buccaneers mucosa, dorsum of the tongue, gingiva, labial mucosa and vermillion border of the lower lip.[11]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.

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.[12]

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.[13]

Dental restorative materials such as amalgam, composite resin , cobalt and gold are found to cause oral lichenoid reactions. These reactions may be suspected when oral lichen planus lesions are confined to the mucosa in close contact with the restoration. They are sometimes unilateral.[14]

Drug induced oral lichenoid reactions are most caused by non-steroidal anti-inflammatory agents and angiotensin converting enzyme inhibitors.[15]

DIAGNOSIS:

Since oral lichen planusis a chronic disease, the patient's medical history, psychological state, and treatment compliance, as well as possible drug interaction, must be considered when evaluating the cost effectiveness of any treatment modalities.[16] When oral lichenoid lesions are suspected to be related to the use of a given drug, the medication should be discontinued whenever possible. Plaque and calculus deposits are associated with a significantly higher incidence of erythematous and erosive gingival oral lichen planuslesions, whereas good oral hygiene is essential and can enhance healing.[17]Mechanical trauma of dental procedures, friction from sharp cusps, rough dental restorations, and poorly fitting dental prostheses can be exacerbating factors of symptomatic oral lichen planusand should receive attention.Also dental amalgam restorations can cause oral lichenoid lesions which may improve following replacement of amalgam with other restorative materials.[18]The psychological profile of patient with Oral Lichen planusshould also be taken into account. Studies have reported higher levels of anxiety, greater depression, and increased psychic disorders in oral lichen planus compared with a control group and stress is one of the most frequent causes of acute exacerbations in oral lichen planuspatients.[19]

On clinical examination , there is presence of classic white lesion. However, an oral biopsy with histopathological examination is recommended both to confirm the clinical diagnosis and particularly to exclude dysplasia and malignancy.[20]

Biopsy includes the existence of a band of lymphocytic inflammatory infiltrate in the sub epithelial onnective tissue, hydropic degeneration of the basal layer and the absence of epithelial dysplasia.

TREATMENT:

General considerations:

Mechanical trauma or irritants such as sharp filling margins or rough surfaces or badly fitting dentures should receive attention. A drug history should be obtained to identify reversible causes of lichenoid eruptions as discontinuation of the offending agent is often curative.[21]

Hypersensitivity reactions should be suspected when the lichenoid lesions are confined to oral mucosal sites in close proximity to dental restorations..An optimal oral hygiene program should be instituted in patients with gingival disease.[22] 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.[23]

Drug therapy

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 . [24]

Corticosteroids

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 fluocinoloneacetonide, fluocinonide and superpotenthalogenated corticosteroids such as clobetasol has been reported in 30–100% of treated patients.[25]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.[26] Other forms of corticosteroids, such as dexamethasone and triamcinolone have been used as an oral rinse for patients with diffuse oral involvement or for elderly patient who may find it technically difficult to apply medication to various active locations of the oral cavity. Careful consideration should be given to the vehicle as unlike skin compounds, which have been well-studied, clinical trials that have compared the strength of corticosteroids in various bases in the oral cavity are generally lacking.[27]

Other topical agents

Patients who exhibit desquamative gingivitis, wide- spread oral disease, or diffuse ulcerations, may not respond adequately to topical corticosteroids alone.[28]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.[29,30]

CONCLUSION:

Oral lichen planus (OLP) is among the more common mucosal conditions a clinician is likely to encounter in their practice. The etiology is unknown. Buccal mucosa, tongue and gingiva are more commonly involved. The question of malignant transformation of OLP remains controversial. Management of lichen planus can be

challenging and discouraging for both the patient and physician. Treatment options should be assessed for any risks and benefitsand analyse the severity of the disease.

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