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Lichen Planus-A Mucocutaneous Pigmentary Disorder-Review

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Abstract

Lichen planus is a chronic, inflammatory, autoimmune disease that affects the skin, oral mucosa, genital mucosa, scalp, and nails. Lichen planus lesions are described using the six P's (planar (flat-topped), purple, polygonal, pruritic, papules, plaques). This review highlights the Pathophysiology and management of oral and cutaneous lichen planus.

Keywords: Lichen Planus; Auto immune diseases; Post inflammatory pigmentation

Introduction

Lichen Planus (LP) is a chronic inflammatory and immune mediated disease that affects the skin, nails, hair, and mucous membranes. It was first described by Wilson in 1869 and is thought to affect 0.5–1% of the world's population Cutaneous Lichen Planus (CLP) most commonly involves the flexor surfaces of the extremities and presents as small itchy violaceous papules in middle-aged adults. It affects woman more often than men in a ratio 2:3 "Pruritic, Purple, Polygonal, Planar, Papules, and Plaques" are the traditional 6 "P's" of LP [1]. The lesions are typically bilateral and relatively symmetric. Oral LP (OLP) can be the sole clinical presentation of the disease or accompanied by cutaneous or other mucosal manifestations including the genital area, gastrointestinal tract, and eyes [2]. The malignant transformation rate of oral lichen planus remains unclear, but estimates range from 0.5% to 3% [3] (Figures 1-3).

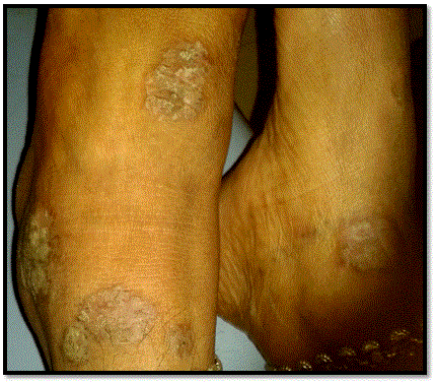

Figure 1: Showing cutaneous lichen planus pigementation on foot.

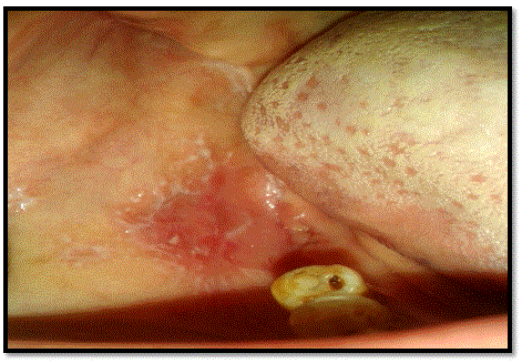

Figure 2: Showing intraoral Lichen planus of reticular pattern depicting striations.

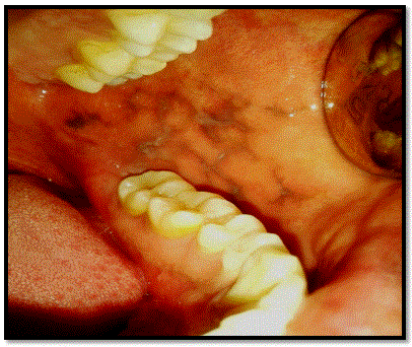

Figure 3: showing post inflammatory melanin pigmentation in oral reticular lichen planus.

Although the aetiology is not fully elucidated, an immunologically induced degeneration of the basal cell layer of the oral mucosa has been suggested.

Basal cells are the prime target of destruction in oral lichen planus. The mechanism of basal cell damage is related to a cell mediated

immune process involving Langerhans cells, T lymphocytes, and macrophages. Langerhans cells and macrophages in the epithelium are the antigen producers that provide the antigenic information for T lymphocytes. Histochemical studies have identified a T-cell origin 6 with CD4 and CD8 subsets in oral lichen planus. There are fewer CD4 helper/inducer cells than CD8 cells, and the CD8 cells, and the CD8 cells are those that are associated with the basal layer [4].

Andreasen [5] divided oral lichen planus into six types:

- Reticular
- Papular
- Plaque-like
- Erosive or ulcerative
- Atrophic or erythematous
- Bullous

Variants of cutaneous lichen planus are distinguished based upon the appearance of the lesions and/or their distribution.

Lesions can affect the:

- Extremities (face, dorsal hands, arms, and nape of neck). This is more common in Middle Eastern countries in spring and summer, where sunlight appears to have a precipitating effect
- Palms and soles
- Intertriginous areas of the skin. This is also known as "Inverse lichen planus".
- Nails characterized by irregular longitudinal grooving and ridging of the nail plate, thinning of the nail plate, pterygium formation, shedding of the nail plate with atrophy of the nail bed, subungual keratosis, longitudinal erthronychia (red streaks), and subungual hyperpigmentation. A sand-papered appearance is present in around 10% of individuals with nail lichen planus [6].
- Scalp: This is also known as lichen planopilaris, acuminatus, follicular lichen planus, and peripilaris, characterised by violaceous, scaly, pruritic papules. Scalp lichen planus can cause scarring alopecia if it is untreated.
- Hair: This variant causes inflammation of hair follicles and gradual replacement with scarring. About 10% of people with lichen planus have the scalp or nail variants of the condition [7].

Characteristics [8-12]

Characteristically, it presents as a series of fine, radiant, white striae known as 'Wickham striae', which may be surrounded by a discrete erythematous border. Koebner phenomena (Skin lesions that responds to local trauma) and Post inflammatory Hyperpigmentation (May occur with lesion resolution (especially in dark skin). Presence of burning sensation with spicy foods is also present often. Oral lesion last much longer than skin lesions, a mean duration of 4.5 years or even more. They are usually bilateral. Nail changes associated with lichen planus include longitudinal ridging and grooving, splitting, nail thinning and nail loss. In severe cases, the nail may be temporarily or permanently destroyed. Lichen Planopilaris is the specific name given to lichen planus on the scalp that causes permanent scarring alopecia with inflammation around affected hair follicles. It mostly affects middle-aged adults as distinct patches of hair loss.

Proposed reasons for the increased risk of oral SCC in patients with OLP include the following:

Compared with healthy mucosa, the oral mucosa affected by OLP may be more sensitive to C albicans and to the exogenous mutagens found in tobacco, alcohol, and betel quid.

In patients with OLP, the chronic inflammatory response and the simultaneous healing response of epithelial wounds may increase the likelihood of cancer-forming gene mutations.

Diagnosis [13-16]

The history, typical oral lesions, and skin involvement are usually sufficient to diagnose OLP, though laboratory studies and biopsy may be required.

Direct immunofluorescence testing can help in distinguishing erosive or the rare bullous OLP from pemphigus vulgaris, benign mucous membrane pemphigoid, dermatitis herpetiformis, and linear immunoglobulin A (IgA) disease. However, OLP has no specific features at direct or indirect immunofluorescence testing.

Skin patch testing is helpful in identifying a contact allergy in some patients with OLP. The current recommendation is to use a standard series; a dental prosthesis series; and a metal salt series that includes gold, mercury, and palladium salts as well as other salts of metals used in dental restorations. The most common allergy is related to mercury contained in amalgam fillings.

Biopsy is required to exclude malignancy or to differentiate between OLP and other white or chronic ulcerative oral lesions, including reactive keratoses, chronic hyperplastic candidosis, epithelial dysplasia, discoid lupus erythematosus, gastrointestinal disease (including oral Crohn's disease), and anemic states [17,18]. 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 and absence of epithelial dysplasia

Management [19-24]

Cutaneous lichen planus may resolve spontaneously within one to two years, although lichen planus affecting mucous membranes may be more persistent and resistant to treatment. Recurrences are common, even with treatment. High-potency topical corticosteroids are first-line therapy for cutaneous lichen planus. Oral antihistamines (e.g., hydroxyzine (Vistaril)) may be used to control pruritus. Hypertrophic lesions are treated with intralesional triamcinolone acetonide (Kenalog), 5 to 10 mg per mL injection (0.5 to 1 mL per 2 cm lesion) Table 1.

Treatment	Dosage	Type of Therapy
High-potency topical corticosteroids	Administered twice daily	First-line therapy
Oral corticosteroids (prednisone)	30 to 60 mg daily for three to six weeks, then dose is tapered over the next four to six weeks	For severe, widespread lichen planus

Phototherapy	30- to 40-minute treatments, two or three times weekly	For severe disease; narrow-band ultraviolet B is preferred over psoralen plus ultraviolet A

Table 1: Treatment of Nongenital Cutaneous Lichen Planus Lesions.

Oral Lichen Planus

Various treatments have been employed to treat symptomatic oral lichen planus, but complete resolution is difficult to achieve. Topical corticosteroids (clobetasol) are first-line therapy. High-potency topical steroids are the most effective, with response rates up to 75 percent compared with placebo. Topical corticosteroids are also first-line therapy for mucosal erosive lichen planus. High-potency corticosteroids applied to the oral mucosa do not appear to cause significant adrenal suppression, even with relatively long-term use. Systemic corticosteroids, such as oral prednisone, should be considered only for severe, widespread oral lichen planus and for lichen planus involving other mucocutaneous sites. A randomized controlled trial revealed that pimecrolimus 1% cream effectively treats erosive oral lichen planus with long-lasting therapeutic effects.

In a randomized controlled trial, aloe vera gel was significantly more effective than placebo in the clinical and symptomatologic improvement of oral lichen planus. If topical corticosteroids are ineffective, carbon-dioxide laser evaporation can lead to long-term remission of symptoms, and may be appropriate as first-line therapy in patients with painful oral lichen planus.

Cutaneous Lichen Planus

Variable treatment responses have been reported with bismuth, grenz rays, arsenic compounds, and topical corticosteroids under occlusion. Hydroxychloroquine, intralesional corticosteroids, and topical sunscreens 5% have been used with a great success. Acitretin, 2% used in combination with topical steroids and sun avoidance, also has resulted in complete resolution of lesions without recurrence. Psoralen-UVA, isotretinoin, systemic corticosteroids, cyclosporine and dapsone have been effectively used to treat classic lichen planus. Some cases may remit spontaneously with sun avoidance and use of sunblock.

Conclusion

It is a self-limiting entity of less known reasons. Being multifactorial, complete cure of the disease is difficult, but with recent advancement in therapeutic medicine, there is more hope for complete cure of this condition. Being oral physicians, it is an important and challenging condition to be diagnosed and managed properly.

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