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## Oral lichen planus: An overview

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### Abstract

Lichen planus is an immunologically mediated mucocutaneous disease that is triggered by varied etiological agents. The oral lichenoid reaction is considered a variant of the disease that needs to be clearly diagnosed as a separate entity from oral lichen planus and treated. They follow a strict cause-effector relationship, protocols that suggest the differentiation. Lichen planus has varied clinical forms in the oral mucosa and cutaneously that has different prognosis. This condition also arises in association with various other systemic conditions such as hypertension, diabetes mellitus. There have been cases reported in the esophagus, larynx, scalp, nail, cutaneous areas, especially arms and wrists, trunk. There is reported malignant transformation that necessitates careful examination, treatment protocol and regular follow-up sessions. This article throws light on the disease condition of oral lichen planus and oral lichenoid reaction that is essential for the differentiation and treatment.

**Keywords:** Betel quid lichenoid lesion, corticosteroids, Grinspan syndrome, malignant transformation, oral lichen planus, oral lichenoid reaction, photochemotherapy, retinoids, vulvovaginal-gingival syndrome

### Introduction

Lichen planus is a comparatively common, mucocutaneous disorder that is mediated immunologically. It can also be autoimmune in pathogenesis. It is chronic in occurrence, with periods of exacerbations and remission. It was first described by British Physician Wilson Erasmus in 1869. Lichens are primitive plant that consists of symbiotic algae and fungi and the word planus in Latin means flat <sup>[1]</sup>. Lichen planus has varied clinical manifestations affecting the skin, oral mucosa, nail, genital mucosa, nail, and scalp <sup>[2]</sup>. This lesion has well-established clinical features and histological features that aid in the diagnosis.

### Etiology

#### Stress

The main etiological factor of lichen planus is stress. There are reported exacerbations of the lesion associated with anxiety and psychological stress <sup>[3-5]</sup>. Psychosomatization arising from prolonged emotional stress contributes greatly to initiation and clinical expression of the lesion <sup>[3, 6]</sup>.

### Systemic medications

Systemic medications such as beta blockers <sup>[7]</sup>, nonsteroidal anti-inflammatory drugs <sup>[8]</sup>, anti malarials <sup>[9]</sup>, diuretics, oral hypoglycemics <sup>[10]</sup>, penicillamine <sup>[11]</sup>, oral retroviral medications <sup>[3, 12]</sup>, are reported to initiate or exacerbate oral lichen planus and oral lichenoid reaction.

### Chronic liver disease and hepatitis C virus

The association between chronic liver disease was first proposed by Mokni et al. <sup>[13]</sup> 1941. Controversy persists in identification of the cause in the association between these two conditions. However, geographical heterogeneity, presence of human leucocyte antigen-DR 6 (HLA-DR) allele is postulated to be associated <sup>[14]</sup>. This association of hepatitis C virus and oral lichen planus is most prevalent in the Mediterranean regions and Japan. The interferon therapy and ribavirin therapy used in the treatment of hepatitis C viral infection are also put forward to aggravate the condition.

### Genetics

Genetic basis of this disease condition plays an important role <sup>[15]</sup>. Higher frequency of HLA-A3 <sup>[16]</sup> is reported to cause lichen planus.

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### Tobacco chewing

Zain et al. proposed the term “betel quid lichenoid lesion”<sup>[17]</sup> for the lesion developed at the area of tobacco chewing and placement in the oral mucosa. It is described as oral lichen planus like lesion. Clinically the lesion was white in color, nonelevated streaks that were nonscrapable. It had a linear, wavy or parallel presentation<sup>[18]</sup>. Graft versus host disease Immune system involvement is seen where the immunological effector mechanism that results in T-cell infiltration leading to the rupture of epithelial basement membrane and basal keratinocyte apoptosis.



**Fig 1:** Presence of ulcerated mucosa in the right cheek.

### Clinical features

Occurs predominantly in females with a female: male ratio of 1.4:1<sup>[2]</sup> and in the age group of third to seventh decade in life. It is seen frequently in all regions of the oral mucosa, mostly noticed in buccal mucosa, gingiva and tongue. They are present bilaterally in most cases. Classically present as six types clinically: Reticular (fine white striae cross each other in the lesion), Atrophic (areas of erythematous lesion surrounded by reticular components), papular type, bullous type, plaque type, erosive or ulcerative type. The reticular type of oral lichen planus is often asymptomatic<sup>[19]</sup>, only can be seen clinically. Ulcerative type in which erythematous areas are seen surrounded by reticular elements.

Periods of remission are seen in oral lichen planus where the symptoms and lesion appear and regress at intervals. The characteristic feature is the Wickham's striae<sup>[2]</sup>, which is found on the surface of the lesion, formed by very fine grayish white lines. They are bilaterally symmetrical in presentation.

Cutaneous presentation involves even the wrist, flexor surfaces of forearms, knees, thighs involving mainly the sacral area. The areas of shin present this condition mostly as hypertrophic plaques.

The vulvovaginal-gingival syndrome<sup>[3, 20]</sup> is considered as a variant lichen planus. This is typically characterized by erosion and desquamation of gingiva, vagina, and vulva. When the scalp is involved, it is called the planopilaris<sup>[2]</sup>.

Nail involvement of oral lichen planus has been reported where the finger nails showed scarring at the matrix of the nails, with subsequent obliteration of the nail plate<sup>[21]</sup>.

- Formation of Pterygium
- Irregular and longitudinal grooving with ridging of the nail plate
- Atrophy and shedding of the nail bed
- Keratosis
- Hyperpigmentation subungually.

Severe pruritis that becomes intolerable is the primary symptom. The association and occurrence of oral lichen planus along with diabetes mellitus and hypertension is termed Grinspan syndrome<sup>[2]</sup>.

### Oral Manifestation

Oral lichen planus is classically present as lesion with radiating whitish gray lines thread like papules, velvety appearance. Bilateral in presentation. They can be lacy or reticular, annular, patches or strings.

The occurrence and distribution of lesion in the oral mucosa is 80% in the buccal mucosa, 65% in the tongue, 20% lips, <10% seen in floor of mouth and palate.

Vesicle and bulla are seen in the oral lesions of lichen planus. The disease manifests in the oral cavity several weeks before the skin lesions. About 15% of oral lichen planus patients have concurrent skin lesions<sup>[22]</sup>.



**Fig 2:** Irregular plate with crusted surface on arm.

The reticular form has a better prognosis as 40% of cases has spontaneous remission<sup>[23]</sup>, the erosive type being long standing and with frequent exacerbations and severe pain and complications.

### Etiopathogenes

Lichen planus is a T-cell mediated disease autoimmune in nature where the CD8+ T-cells trigger the apoptosis of oral epithelial cells at the basal layer<sup>[24]</sup>. Keratinocyte antigen expression and antigen unmasking are involved in the disease mechanism. It may be a self-peptide or a heat shock protein. Then subsequently T-cells migrate towards the basal keratinocytes during surveillance or chemokine mediated action. The antigen directly binds to the migrated CD8+ cells by the major histocompatibility complex-1 (MHC) on keratinocyte. Langerhans cells are increased in lesions of lichen planus and there is upregulated MHC-II expression. The CD4+ cells and interleukin-12 activate CD8+ T-cells that are involved in the apoptosis of keratinocytes through FasL mediated and tumor necrosis factor-alpha (TNF-alpha).

The vascular adhesion molecules receive the expression of the reciprocal receptors of infiltrating lymphocytes<sup>[25]</sup>. This is the cytokine-mediated lymphocyte that contributes to the homing mechanism. Matrix metallo protease 9 are majorly involved in the tissue matrix protein degradation.

### The chronic nature of the disease is contributed by the

Regulated on Activation, Normal T-cell expressed and secreted<sup>[26]</sup> that is, a member of “CC chemokine family”. This greatly recruits the mast cells and lymphocytes that results in degranulation, releasing chymase and TNF-alpha.

The heat shock proteins [27] are also upregulated by the lesional keratinocytes of oral lichen planus. Inflammation that exists prior to the disease condition can contribute to the expression of heat shock protein.

### Malignant Transformation

The follow-up studies of oral lichen planus reveals the malignant transformation of this condition to be up to 5.3%. The highest rate of malignant transformation has been noted in erythematous and erosive type. The chance of malignant transformation drops if initial dysplasia is excluded [3]. Cases of oral lichen planus have been reported with a malignant transformation of squamous cell carcinoma.

### Investigation

Clinical examination with a thorough history, followed by tissue biopsy is routinely sufficient for the diagnosis of oral lichen planus. Histopathological examination from the biopsy of the site of lesion reveals the diagnosis of lichen planus. Immunofluorescence is an adjunctive technique in the diagnosis of lichen planus. The direct immunofluorescence of lichen planus shows "Linear pattern" in the basement zone and exhibit positive fluorescence with antifibrinogen [28]. IgA, IgG, complement C3 were seen on colloid bodies. Indirect immunofluorescence aids in the detection of antibodies in the circulating blood of the lichen planus patient. The circulating antibodies that react and bind to the basal cells of the epithelium gives rise to the "annular fluorescence" or the "string of pearls" appearance [29].

### Diagnosis

- Allergic patch test
- Immunofluorescence.

### Management

- Removal of the causative agent
- Medication modification in respect to dosages.

### Discussion

The use of homeopathic treatment in LP has been described in the literature as a possible alternative to corticosteroids. Medications like Antim-crud., Ars-alb., Ars-iod., Jugl-c., Kali-bi., Sul-iod, Ign, Sepia, Sulphur and Thuja, for example, have already been tested and proved effective in controlling this pathology [30, 31]. Mousavi et al., [32] in a single blind randomized control clinical trial with 30 patients with OLP treated with Ignatia noted that there was a statistically significant reduction of pain and size of lesions compared to the control group (placebo). These authors also report that, after the treatment, the patient had an improvement of quality of life and was able to eat better and perform oral hygiene without discomfort. Ignatia amara is suited for nervous temperaments, women in a sensitive and excitable nature and of great contradictions [33]. In the case described in this paper, the initial prescription was also Ignatia Amara, given the emotional elements presented in the first instance. However, a new homeopathic interview revealed other features that culminated in the replacement of the medication by Natrum muriaticum. Among the mental characteristics of Natrum muriaticum, according to *Materia Medica* [34], are: "Psychic causes of disease; ill effects of grief, fright, anger, depressed, particularly in chronic diseases. Consolation aggravates. Irritable; gets into a

passion about trifles. Awkward, hasty, Wants to be alone to cry".

LP is a T-cell-mediated autoimmune disease. Inflammatory cells involved in this process consist of T helper and T cytotoxic lymphocytes, natural killer (NK) cells, and dendritic cells. T-cell activation is central to the pathogenesis of the pathology. Cytotoxic T-cell infiltration into the epithelium results in apoptotic basal keratinocytes [35]. LP is a complex disease and can be caused or triggered by genetic malfunction and/or environmental factors. The existence of familial cases of LP may suggest a possible genetic predisposition [36]. Associated factors and disease conditions seen in LP include but are not limited to stress/anxiety, hepatitis C virus (HCV), autoimmune diseases, internal malignancies, dyslipidemia, and viral infections [37].

The use of homeopathy for the treatment of OLP is based primarily in its correlation with stress, as previously described in the literature. It has been documented that if the adrenal medullary sympathetic system is activated excessively, persistently and too often, illness and disease may occur [38]. Chronic stress may cause a reduction in mitogenesis, alterations in lymphocytes, reductions in the ratio of T-helper cells and T-suppressor cells and an elevation in the number of natural killer cells [39].

Nanoy et al. [40], in a study with 148 patients with OLP reported that 51% of them revealed having experienced stressful life events prior to the appearance of the lesions. Furthermore, over 37% of patients believed that the pathology was caused by stress. The main categories of causes of stress were death and illness of beloved ones, work and interpersonal and family relationships. At this point, one can understand that the main purpose of the homeopathic remedy is help the patient to make the effort of facing his conflict, adjusting himself to reality and of integrating himself as a healthier person. This process is what happened to the patient described in this paper who, during the years, learned how to co-exist with her condition and make internal adjustments that made her able to overpass the difficulties that all social relations brings along to people involved.

### Conclusion

Oral lichen planus even though may portray a similar clinical picture, the site of occurrence, detailed history and investigation can draw a clear line of demarcation hence establishing variable treatment protocol. Knowledge of the condition is essential to distinguish. Regular follow-up is very important as this lesion poses a chance of malignant transformation.

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