

CLINICAL PRESENTATION OF ORAL LICHEN PLANUS IN A TERTIARY CARE CENTRE – A RETROSPECTIVE STUDY

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Received : 25/12/2024
Received in revised form : 13/02/2025
Accepted : 01/03/2025

Keywords:

Oral lichen planus, clinical variants, retrospective study, histopathology, tertiary care.

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DOI: 10.47009/jamp.2025.7.2.106

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (2); 520-522



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Abstract

Background: Oral lichen planus (OLP) is a chronic mucocutaneous disorder affecting the oral mucosa and often presents with various clinical manifestations. Understanding its epidemiology and clinical variants is crucial for early diagnosis and appropriate management. This study aimed to assess the demographic profile, clinical presentation, and histopathological characteristics of OLP in patients attending a tertiary care centre. **Materials and Methods:** This retrospective analysis included 30 patients diagnosed with OLP for six months. Demographic details, clinical variants, and histopathological findings were recorded. Statistical analyses were performed to determine the prevalence of the different subtypes. **Result:** Most patients were aged > 30 years (53.3%) and had a male predominance (66.7%). The most frequently observed clinical variant was the reticular type (50%), followed by erosive (33.3%), papular (6.7%), plaque (6.7%), and atrophic types (3.3%). Skin involvement was observed in 50% of the cases, with classical lichen planus in 40% and hypertrophic lesions in 10%. Genital involvement was observed in 10% of patients. Nail changes were observed in 33.3% of patients, with long ridging being the most common abnormality. Histopathological examination confirmed classical lichen planus in 46.7% of the cases. **Conclusion:** Our study found a male predominance, and the reticular type was the most common, followed by the erosive type. Given the malignant potential, ulcerative and erosive OLP cases require follow-up every six months.

INTRODUCTION

Oral lichen planus (OLP) is a chronic mucocutaneous disorder affecting the skin, oral, and genital mucosae.^[1] Oral lichen planus can occur without skin lesions; however, skin lesions may appear, follow, or co-occur. The occurrence of oral lesions without skin lesions was first reported by Andry in 1894.^[2] The worldwide prevalence of oral lichen planus in the adult population is 0.22% to 1.2%.^[3] In the Indian population, the prevalence is 2.6%.^[4] Oral lichen planus occurs between the third and sixth decades of life and is more persistent and resistant to treatment. The aetiopathogenesis of oral lichen planus is complex. Genetic and environmental factors have been associated with this condition. Oral lichen planus is usually asymptomatic; however, pain may be present in erosive and atrophic lesions. It is a chronic disorder with remissions and exacerbations,

leading to varying degrees of morbidity. Histopathologically, the lesion shows hyperkeratosis with parakeratosis and vacuolar degeneration of the basal layer with a band-like inflammatory infiltrate in the papillary dermis.^[5] Accurate identification of OLP variants is crucial for effective treatment and monitoring. However, limited regional data hinder a comprehensive understanding of its prevalence and clinical pattern.

This study aimed to assess the age, sex, incidence, and various clinical types of oral lichen planus in a tertiary health centre.

MATERIALS AND METHODS

This retrospective analytical study included 30 patients from the outpatient department of a tertiary health centre over 6 months. The Institutional Ethics Committee approved the study before its initiation, and informed consent was obtained from all patients.

Inclusion and exclusion criteria

We included patients with oral lichen planus attending our outpatient department. Patients unwilling to participate and those who had already been treated for oral lichen planus were excluded.

Methods: A detailed personal history, including demographic details, lifestyle factors, and possible sources of stress, was recorded. Additionally, a thorough drug history was obtained to identify any potential medication-related aetiologies, and the presence of comorbidities was assessed.

All patients underwent routine blood tests, including serological tests. To rule out fungal infections, oral lesion scrapings were examined for the presence of *Candida* species. A biopsy of the oral lesions was performed for histopathological confirmation of

diagnosis. All data are presented as frequencies and percentages.

RESULTS

Of the 30 patients, the majority were aged >30 years 16 (53.3%), while 14 (46.7%) were aged <30 years. Regarding sex, 20 (66.7%) patients were male and 10 (33.3%) were female.

Regarding disease duration, 27 (90%) patients had symptoms for <1 year, while 3 (10%) had symptoms persisting for more than a year. The most common clinical type was reticular 15 (50%), followed by erosive 10 (33.3%), papular 2 (6.7%), plaque 2 (6.7%), and atrophic 1 (3.3%).

Table 1: Clinical and histopathological characteristics.

		N (%)
Age (years)	<30	14 (46.7%)
	>30	16 (53.3%)
Sex	Male	20 (66.7%)
	Female	10 (33.3%)
Duration (year)	<1	27 (90%)
	>1	3 (10%)
Type	Reticular	15 (50%)
	Atrophic	1 (3.3%)
	Papular	2 (6.7%)
	Plaque	2 (6.7%)
	Erosive	10 (33.3%)
Skin	Nil	15 (50%)
	Classical LP	12 (40%)
	Hypertrophic	3 (10%)
Genital	Absent	27 (90%)
	Present	3 (10%)
Nails	Nil	20 (66.7%)
	Thinning	2 (6.7%)
	Long ridging	7 (23.3%)
	20 nail dystrophies	1 (3.3%)
HPE Skin	Nil	16 (53.3%)
	Classical LP	14 (46.7%)

Skin involvement was absent in 15 (50%) patients, while classical lichen planus was observed in 12 (40.0%) and hypertrophic lesions in three (10%). Genital involvement was observed in three (10%) patients.

Nail changes were observed in 10 (33.3%) patients, with long ridging (7, 23.3%) being the most frequent abnormality, followed by nail thinning 2 (6.7%) and 20-nail dystrophy 1 (3.3%). Histopathological examination of the skin revealed classical lichen planus in 14 (46.7%) patients, while no histopathological changes were noted in 16 (53.3%) patients [Table 1].

DISCUSSION

OLP is a complex condition that can occur independently or in association with other cutaneous lesions. Studies suggest that up to 70% of patients with cutaneous lichen planus may also present with oral manifestations.^[5] The exact cause of OLP remains unknown, but it is strongly linked to T cell-mediated immunity.^[6] Cytotoxic CD8+ T cells play a

key role in triggering basal cell apoptosis.^[7] Following keratinocyte antigen expression, T cells migrate into the epithelium and bind to MHC I on keratinocytes. Activated CD8+ cells, through tumour necrosis factor-alpha (TNF- α) and Fas ligand (FasL), induce apoptosis of basal keratinocytes.^[8]

T cell-secreted matrix metalloproteinase-9 (MMP-9) disrupts the epithelial basement membrane, further promoting keratinocyte apoptosis.^[9] Additionally, RANTES (Regulated on Activation, Normal T Cell Expressed and Secreted) plays a critical role in the recruitment of lymphocytes, natural killer (NK) cells, mast cells, and basophils, leading to mast cell degranulation via G protein-coupled receptors.^[10]

Several drugs have been implicated in the development of OLP, including Arsenic, gold, NSAIDs, naproxen, allopurinol, and antimalarials.^[11,12] Artificial dentures made of silver amalgam, gold, cadmium, cobalt, and non-metals like epoxy resin have also been associated with OLP.^[13] Hepatitis C viral infection has been linked to OLP, particularly in individuals carrying the HLA-DR6 allele.^[14] OLP predominantly affects middle-aged

individuals, with a higher prevalence in females.^[15] It can occur on any oral site, with the buccal mucosa being the most commonly affected, followed by the tongue and gums.^[16]

According to Anderson's classification, there are six types of OLP, with the reticular type being the most common and usually asymptomatic.^[17,18] At initial presentation, the erosive form of the disease was the most common, seen in 40% of patients, with symptoms affecting most patients across all disease types.^[6] Murti et al. found that histological analysis revealed epithelial atrophy in 74% of the 94 biopsies taken from patients with oral lichen planus.^[19] OLP follows a chronic course with periods of remission and exacerbations. The atrophic and erosive types have an increased risk of malignant transformation.^[20] The underlying mechanism for malignancy in OLP is thought to be the accumulation of inducible nitric oxide synthase (iNOS) and 8-nitroguanine, which leads to oxidative DNA damage.^[4]

The chronicity of the disease is attributed to nuclear factor kappa B (NF- κ B) activation and transforming growth factor-beta (TGF- β) inhibition, which contributes to keratinocyte hyperproliferation.^[21] Nail involvement in OLP is uncommon, but when present, pterygium formation is the most frequently reported finding.^[22]

CONCLUSION

Our study observed a male predominance, which may be due to the small sample size of the study. The reticular type was the most common, followed by the erosive type. Given the risk of malignant transformation, patients with ulcerative and erosive OLP should undergo follow-up every six months. Future studies with larger sample sizes and longer symptom durations are needed to better analyse risk factors and clinicopathological correlations, aiding in the early detection and improved management of malignancies.

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