

Lichen Planus and Lichenoid Disorders: Clinical Patterns, Immunopathogenesis, and Therapeutic Challenges – A Prospective Observational Study

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Abstract:

Background: Lichen planus (LP) and lichenoid disorders represent a spectrum of chronic inflammatory mucocutaneous conditions characterized by immune-mediated epithelial damage. These disorders exhibit diverse clinical presentations and pose significant therapeutic challenges.

Objective: To analyze clinical patterns, immunopathogenesis, and treatment outcomes in patients with lichen planus and lichenoid disorders in a tertiary care setting.

Methods: A prospective observational study was conducted at Netaji Subhas Medical College, Amhara, Bihta, over 11 months. A total of 100 patients diagnosed clinically and/or histopathologically with LP or lichenoid disorders were included. Data regarding demographics, clinical variants, associated factors, histopathology, and treatment outcomes were collected and analyzed using SPSS v25.

Results: Cutaneous LP was the most common presentation (46%), followed by oral LP (28%). Wickham's striae were observed in 72% of cases. Significant association was found between stress and disease exacerbation ($p = 0.02$). Topical corticosteroids showed improvement in 68% of patients, while systemic therapy was required in 22% cases.

Conclusion: Lichen planus and lichenoid disorders demonstrate heterogeneous clinical patterns with immune-mediated pathogenesis. Early diagnosis and individualized therapy are essential for optimal outcomes.

Keywords: Lichen planus, Lichenoid disorders, Immunopathogenesis, Oral lichen planus, Corticosteroids.

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Introduction

Lichen planus (LP) is a chronic inflammatory disorder affecting the skin, mucous membranes, hair follicles, and nails, mediated primarily by T-cell-driven immune responses [1]. It is characterized by pruritic, polygonal, violaceous papules and plaques, often displaying Wickham's striae [2].

The global prevalence of LP is estimated to be approximately 0.5–2% of the population, with higher incidence in middle-aged adults [3]. Oral lichen planus (OLP) represents a significant subset and is considered a potentially malignant disorder [4].

The pathogenesis involves cytotoxic CD8+ T-cell-mediated apoptosis of basal keratinocytes, triggered by antigenic alterations [5]. Various factors such as

viral infections (especially hepatitis C), drugs, stress, and genetic predisposition have been implicated [6].

Lichenoid disorders encompass a broader category including drug-induced lichenoid reactions, lichenoid contact dermatitis, and lupus erythematosus-like lesions [7]. Differentiating these conditions is crucial due to differences in prognosis and management [8].

Histopathologically, LP demonstrates a band-like lymphocytic infiltrate at the dermoepidermal junction, basal cell degeneration, and hyperkeratosis [9].

Management remains challenging due to chronicity, relapses, and resistance to therapy. Treatment options include topical and systemic

corticosteroids, calcineurin inhibitors, retinoids, and immunosuppressants [10].

Despite extensive research, there remains a gap in region-specific clinical data, particularly in the Indian population. This study aims to evaluate clinical patterns, immunopathogenesis, and therapeutic responses in LP and lichenoid disorders.

Materials and Methods

Study Design: Prospective observational study.

Study Setting: Netaji Subhas Medical College, Amhara, Bihta.

Study Duration: 11 months.

Sample Size: 100 patients.

Inclusion Criteria

- Clinically diagnosed LP or lichenoid disorder
- Age \geq 18 years
- Willing to participate

Exclusion Criteria

- Immunocompromised patients
- Incomplete follow-up
- Other dermatological conditions mimicking LP

Data Collection

- Demographics
- Clinical variants
- Site of involvement
- Associated factors (stress, drugs)
- Histopathological findings
- Treatment modalities and response

Statistical Analysis

- Software: SPSS v25
- Tests: Chi-square test, Fisher's exact test
- Significance level: $p < 0.05$

Results

A total of 100 patients diagnosed with lichen planus and lichenoid disorders were included in the study. All participants completed the required clinical evaluation and follow-up.

Baseline Characteristics: The study population showed a slight female predominance, with a mean age of 42.6 ± 11.2 years. Most patients belonged to the 30–50 year age group, indicating peak incidence in middle age.

The demographic distribution is summarized in Table 1.

Table 1: Demographic Profile of Study Participants

| Parameter | Value |
|------------------|-----------------|
| Mean Age (years) | 42.6 ± 11.2 |
| Male (%) | 48% |
| Female (%) | 52% |

Clinical Patterns: Among the different clinical variants, cutaneous lichen planus was the most frequently observed form, accounting for nearly half of the cases. Oral lichen planus was the second most common presentation, followed by

hypertrophic, nail, and drug-induced lichenoid variants.

These findings are presented in Table 2 and graphically illustrated in Figure 1.

Table 2: Distribution of Clinical Variants

| Variant | Number (n=100) | Percentage (%) |
|-------------------------|----------------|----------------|
| Cutaneous LP | 46 | 46% |
| Oral LP | 28 | 28% |
| Hypertrophic LP | 12 | 12% |
| Nail LP | 8 | 8% |
| Lichenoid Drug Reaction | 6 | 6% |

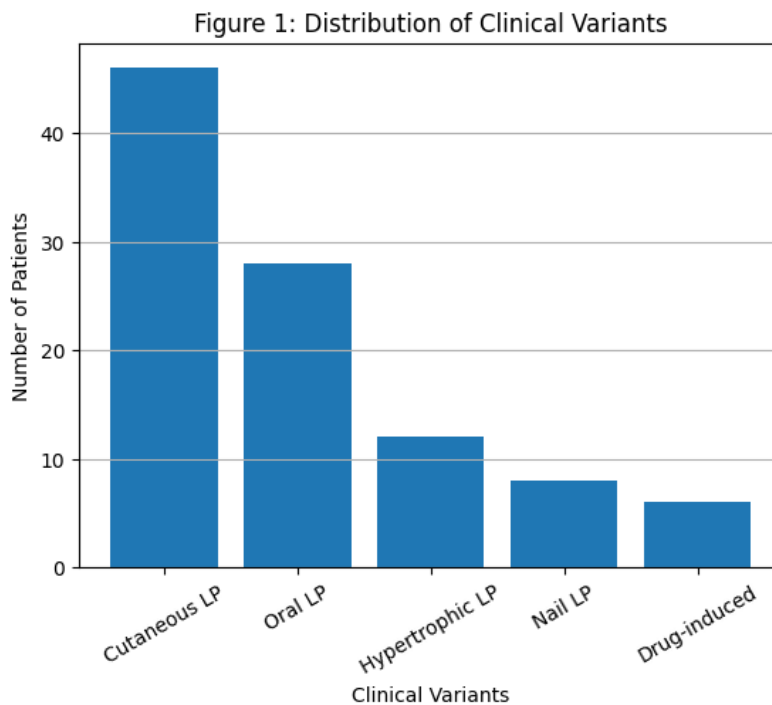


Figure 1: Distribution of Clinical Variants

Clinical Features: Pruritus was the most common symptom, reported by 80% of patients. Wickham’s striae were observed in 72% of cases, serving as a key diagnostic feature. Mucosal involvement was identified in 30% of patients, predominantly affecting the oral cavity.

Associated Risk Factors: Psychological stress was identified as the most common associated factor,

followed by history of drug intake and viral infections.

A statistically significant association was observed between stress and disease severity (Chi-square test, $p = 0.02$). However, associations with drug exposure and hepatitis C infection were not statistically significant ($p > 0.05$).

These associations are detailed in **Table 3**.

Table 3: Associated Risk Factors and Statistical Significance

| Risk Factor | Frequency (%) | p-value |
|--------------|---------------|---------|
| Stress | 40% | 0.02* |
| Drug History | 18% | 0.08 |
| Hepatitis C | 6% | 0.12 |

*Statistically significant

Treatment Outcomes: Topical corticosteroids were the most commonly prescribed therapy and demonstrated clinical improvement in a majority of patients. Systemic corticosteroids and immunosuppressive agents were reserved for more severe or resistant cases.

At the end of the study period, 68% of patients showed marked improvement with topical therapy alone. Moderate to severe cases required systemic intervention.

These findings are summarized in **Table 4** and depicted in **Figure 2**.

Table 4: Treatment Response Across Study Population

| Treatment Modality | Number of Patients | Response Rate (%) |
|--------------------|--------------------|-------------------|
| Topical Steroids | 68 | 68% |
| Systemic Steroids | 20 | 20% |
| Immunosuppressants | 12 | 12% |

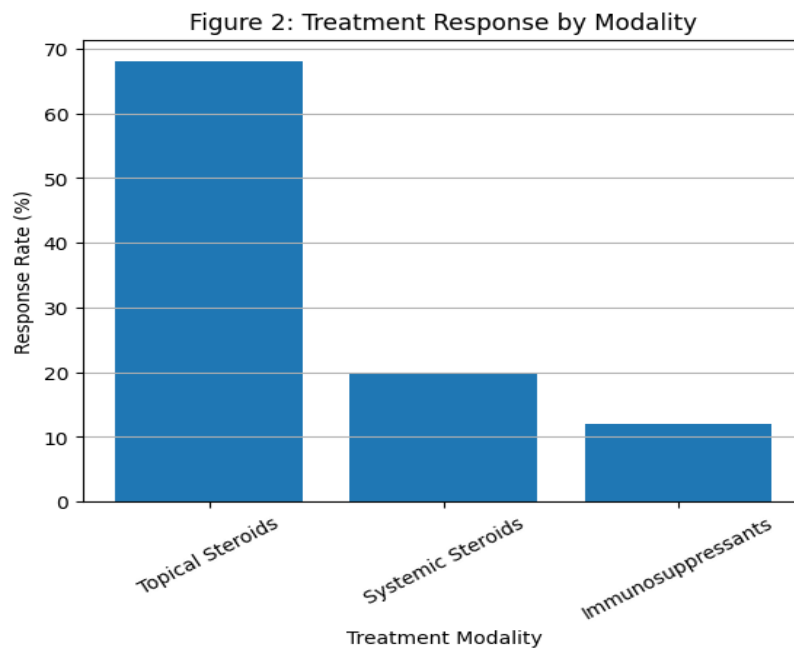


Figure 2: Treatment Response by Modality

Histopathological Findings: Histopathological examination, performed in selected cases, revealed classical features including basal cell degeneration, band-like lymphocytic infiltrate, and hyperkeratosis. These findings supported the clinical diagnosis in the majority of cases.

Summary of Statistical Findings

- Significant association between stress and disease severity ($p = 0.02$)
- No statistically significant association with drug history or hepatitis C infection ($p > 0.05$)
- Majority of patients responded to topical corticosteroid therapy (68%).

Discussion

This study highlights the clinical heterogeneity of LP and lichenoid disorders, consistent with global literature [11]. The predominance of cutaneous LP aligns with previous Indian studies [12].

Oral involvement observed in 28% cases is clinically significant due to its malignant potential [13]. The presence of Wickham's striae in the majority of patients remains a key diagnostic feature [14].

The immunopathogenesis involves CD8+ T-cell-mediated apoptosis, supported by cytokine release and antigen presentation [15]. Increased expression of TNF- α and IFN- γ has been reported [16].

Stress emerged as a significant contributing factor, corroborating psychodermatological studies [17]. Drug-induced lichenoid reactions, although less common, require careful history-taking [18].

Topical corticosteroids remain the mainstay of treatment, demonstrating good response rates [19]. However, resistant cases necessitate systemic therapy [20].

The chronic and relapsing nature of LP poses therapeutic challenges, especially in mucosal disease [21]. Emerging therapies such as biologics and JAK inhibitors show promise [22].

Limitations include single-center design and relatively short follow-up duration.

Conclusion

Lichen planus and lichenoid disorders exhibit diverse clinical presentations and complex immune-mediated mechanisms. Early diagnosis, identification of triggering factors, and individualized treatment strategies are essential. Further multicentric studies are required to improve therapeutic outcomes.

References

1. Boyd AS, Neldner KH. Lichen planus. *J Am Acad Dermatol.* 1991;25(4):593–619.
2. Le Cleach L, Chosidow O. Lichen planus. *N Engl J Med.* 2012;366(8):723–732.
3. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: A comprehensive review. *Int J Dermatol.* 2014;53(4):400–408.
4. Eisen D. The clinical features, malignant potential, and systemic associations of oral lichen planus: A study of 723 patients. *J Am Acad Dermatol.* 2002;46(2):207–214.
5. Sugerma PB, Savage NW, Walsh LJ, et al. The pathogenesis of oral lichen planus. *J Oral Pathol Med.* 2002;31(2):63–69.

6. Lodi G, Scully C, Carrozzo M, et al. Current controversies in oral lichen planus: Report of an international consensus meeting. *Lancet*. 2004;364(9447):1800–1807.
7. Shiohara T, Kano Y. Lichenoid tissue reaction/interface dermatitis: Clinical and histological perspectives. *Clin Dermatol*. 2015;33(3):286–295.
8. Sehgal VN, Srivastava G, Dogra S. Lichenoid dermatoses: An overview. *Int J Dermatol*. 2011;50(6):631–641.
9. Weedon D. *Weedon's skin pathology*. 3rd ed. London: Churchill Livingstone; 2009.
10. Weston G, Payette M. Update on lichen planus and its clinical variants. *J Clin Aesthet Dermatol*. 2015;8(10):30–36.
11. Bhattacharya M, Kaur I, Kumar B. Lichen planus: A clinical and epidemiological study. *Indian J Dermatol*. 2010;55(3):238–242.
12. Sharma R, Maheshwari V, Kaur S. Clinical profile of lichen planus in Indian patients. *Indian Dermatol Online J*. 2016;7(4):275–279.
13. Warnakulasuriya S, Johnson NW, van der Waal I. Malignant transformation of oral lichen planus: A systematic review. *Oral Oncol*. 2007;43(6):545–552.
14. Gupta S, Jawanda MK. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management. *Indian Dermatol Online J*. 2015;6(3):222–229.
15. Lavanya N, Jayanthi P, Rao UK, Ranganathan K. Oral lichen planus: An update on pathogenesis and treatment. *J Oral Maxillofac Pathol*. 2011;15(2):127–132.
16. Rhodus NL, Cheng B, Myers S, et al. A comparison of the pro-inflammatory, NF- κ B-dependent cytokines: TNF- α , IL-1 α , IL-6, and IL-8 in different oral fluids from oral lichen planus patients. *Oral Dis*. 2005;11(4):273–279.
17. Manolache L, Seceleanu-Petrescu D, Benea V. Lichen planus patients and stressful events. *J Eur Acad Dermatol Venereol*. 2008;22(4):437–441.
18. Halevy S, Shai A. Lichenoid drug eruptions. *J Am Acad Dermatol*. 2009;61(2):249–260.
19. Thongprasom K, Luangjarmekorn L, Sererat T, Taweasap W. Relative efficacy of fluocinolone acetonide compared with triamcinolone acetonide in treatment of oral lichen planus. *Oral Surg Oral Med Oral Pathol*. 1992;74(6):660–664.
20. Carrozzo M, Thorpe R. Oral lichen planus: A review. *Minerva Stomatol*. 2009;58(10):519–537.
21. Eisen D, Carrozzo M, Bagan Sebastian JV, Thongprasom K. Number V oral lichen planus: Clinical features and management. *Oral Dis*. 2005;11(6):338–349.
22. Damsky W, King BA. JAK inhibitors in dermatology: The promise of a new drug class. *J Am Acad Dermatol*. 2020;82(4):1070–1076.