

**Clinicopathological Study of Lichen Planus in a Tertiary Care Center of North India**Shailza<sup>1</sup>, Amarjeet Singh Verma<sup>2</sup>, Jyoti Singh Rajput<sup>3</sup><sup>1</sup>Associate Professor, Department of Pathology, NCR Institute of Medical Sciences (UP)<sup>2</sup>Associate Professor, Department of Dermatology, Venereology & Leprosy, LLRM Medical College, Meerut (UP)<sup>3</sup>Associate Professor, Department of Pathology, NCR Institute of Medical Sciences (UP)

Received: 15-11-2023 / Revised: 19-12-2023 / Accepted: 13-01-2024

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Conflict of interest: Nil

**Abstract:**

**Background:** Lichen planus (LP) is an idiopathic, chronic inflammatory disease of the skin, mucous membrane, and nails. Worldwide distribution is seen, with 0.38% of the lesion load present in India. The present study was conducted to study the demographic profiles (such as age and gender) and histopathology of LP and their clinical presentation.

**Materials and Methods:** The present study was conducted in the Department of Pathology at NCR Institute of Medical Sciences. All cases presenting with LP and undergoing skin biopsy from December 2022 to May 2023 were included in the study. Detailed clinical history was taken, and histomorphological evaluation was performed for all the included cases.

**Results:** A total of 50 cases of LP were analyzed. The mean age of presentation was  $34.89 \pm 16.02$  years, with slight male predominance. The mean duration of the disease was  $7.8 \pm 5.4$  months. Clinically, most cases presented with mild to moderate pruritis (64.51%), followed by intense pruritis (25.16%). Violaceous papules and plaques were the predominant findings in 80.64% of patients, followed by hyperpigmented papules and macules (13.54%). Histomorphologically, hyperkeratosis was observed in almost all cases (99.35%), followed by wedge-shaped hypergranulosis (65.80%). The most common variant found was classic LP (30.96%), followed by hypertrophic LP (29.6%) and LP pigmentosus (26.4%).

**Conclusion:** Detailed history, clinical examination, and careful histological assessment help diagnose LP and differentiate its variants.

**Keywords:** Hyperkeratosis, hypertrophic, lichen planus, violaceous papules.

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**Introduction**

Lichen planus (LP) is a cutaneous disorder with prototypic lichenoid papules that exhibit distinct color, morphology, and microscopic features, develop in typical locations, and manifest characteristic patterns of evolution. [1,2] An autoimmune reaction in which CD8+ T lymphocytes attack basal keratinocytes, leading to apoptosis of the cells, has been favored as a etiopathology that still is unclear [3]. Lichenoid, polygonal papules often show fine white lines called Wickham's striae. Lesions most commonly occur on the limbs and the dorsal aspect of the trunk. [4,5] LP can manifest at any age, preferentially between 30 and 60. [6] It is uncommon in childhood, as only 1–3% of patients are children. [7]

The characteristic histopathological findings of LP include orthokeratosis, hyperkeratosis, circumscribed wedge-shaped hypergranulosis representing the histopathologic substrate of

Wickham's striae, and sawtooth-like acanthosis. The upper dermis shows a band-like infiltrate consisting mainly of lymphocytes. At the dermo-epidermal junction, vacuolar degeneration with Civatte body's small clefts (Max Joseph spaces) may be seen along with band-like lymphocytic infiltration. [8,9]

We studied demographic profiles (such as age and gender), clinical and histopathological features in LP patients, and the association between clinical and histopathological diagnosis of LP.

**Materials and Methods**

The study was conducted after approval from the institutional ethical and research committee in the Department of Pathology at the NCR Institute of Medical Sciences (UP). It was a cross-sectional study of cases of LP over 6 months (December 2022 to May 2023), which included a retrospective study

from January 2022 to November 2022 and a prospective study from December 2022 to May 2023.

### Inclusion Criteria

All cases presented with LP and underwent skin biopsy for histopathology during the study period.

### Exclusion Criteria

- Patients presenting with LP but not undergoing skin biopsy.
- Patients are undergoing skin biopsy for skin lesions other than LP.
- Inconclusive skin biopsies, which were suspected to be LP clinically.

### Data Collection and Methods

For the retrospective study, paraffin blocks were retrieved from the Department of Pathology, and clinical details were obtained from the hospital's medical records department.

### For Prospective Study

All the cases presenting with LP of skin in the Department of Dermatology during the study period were included.

Detailed clinical histories, including symptoms, duration, site, and type of lesion concerning age, gender, and distribution pattern of the lesions for all the cases, were recorded. Informed written consent was obtained from patients before the biopsy. Under local anesthesia, skin biopsies were taken from the ideal lesion site using punch biopsy needles. Skin biopsies received were fixed in 10% neutral buffered formalin. Tissue processing, paraffin embedding, hematoxylin, and eosin staining were done using histological techniques.

Hematoxylin and eosin-stained sections were evaluated microscopically, along with clinical data. Histopathological diagnosis was made based on findings, such as orthokeratosis, hypergranulosis, acanthosis, atrophy, basal vacuolar alteration, ulcer, a band-like dermal lymphocytic infiltrate, perivascular and peri-adnexal lymphocytic infiltrate, basal layer pigmentation, pigment incontinence, melanophages, vacuolar degeneration, and Civatte bodies. Based on histopathological and clinical findings, LP was classified into nine categories, such as classic, hypertrophic, LP pigments, bullous, actinic, atrophic, ulcerative, follicular, and oral. Statistical analysis included a descriptive statistics in percentage was used to show the characteristics of collected data, association was established using the Chi-square test at 95 % confidence interval.

### Results

In the index study, 50 cases of LP diagnosed on histopathological examination were studied.

Overall, slight male predominance was seen, with 28 cases being males (56 %) and 22 females (43 %). Male:female ratio was 1.3:1. Both classic LP and hypertrophic LP showed that males more commonly affected than females. However, female predominance  $n=35$  (70% compared to 30 % males) was seen in LP pigments. Pearson's Chi-square test determined the association between the types of LP and gender to be statistically significant ( $P < 0.05$ ).

The age range of the patients observed was from 4 to 79 years in males and 10 to 70 years in females. The mean age of presentation was  $34.89 \pm 16.02$  years. Overall, the maximum patients were 30–39 years (24 %) and 20–29 years (22 %). The most minor cases were seen in the 0–9 years of age group (4 %). The most common age group affected in classic LP and LP pigmentosus (LPP) was 20–29 years, whereas it was a decade later for hypertrophic LP (30–39 years). The association between variants of LP and age groups using the Pearson's Chi-square test was statistically significant ( $P < 0.05$ ). The most common site of involvement was lower limb (54 %), followed by upper limb (24 %), face and neck (10%), genitalia (2.4%), scalp (2 %), trunk (2 %), and palms and soles (0.6%).

The most common variant of LP was classic LP (30%), followed by hypertrophic LP (28 %) and LPP (26 %) types. Atrophic LP constituted 4 % of cases, and 2% belonged to follicular planus..

Violaceous papules and plaques were a predominant finding in 80 % of patients, followed by hyperpigmented papules and macules in 14 %. White papules were found in 4 % of patients, and violaceous papules with ulceration and vesicle formation in 0.01% . Most cases presented with mild to moderate pruritis (64 %), followed by intense pruritis (26%). Pruritis associated with pain was seen in only 8 % of cases , 0.4 % of cases presented with burning on food intake, while 4 % were asymptomatic. In classic LP and LPP, most patients (80 % and 84 %, respectively) presented with mild to moderate pruritis. In contrast, in hypertrophic LP, intense pruritis was the most typical presentation (76 %). The association between LP types and symptoms was found to be statistically significant ( $P < 0.05$ ).

Overall, the mean duration of the disease was  $7.82 \pm 5.34$  months. In maximum cases of classic LP (86 %), the time of illness was <6 months, while in most cases of hypertrophic LP (56 %) and LPP (40 %), the duration of illness was 6 months to 1 year.

Sections examined were sufficiently characteristic of LP with some degree of variability in histologic features. Epidermal changes included hyperkeratosis in 49/50 (98 %) cases. Wedge-shaped hypergranulosis was seen in 33/50 (66 %) cases.

Histopathological findings observed in hypertrophic LP were hyperkeratosis (100%), hypergranulosis (96%), acanthosis (96%), and lymphocytic infiltrate (100%). LP pigments predominantly showed hyperkeratosis (100%), lymphocytic infiltrate (100%), pigment incontinence (94%), and melanophages (96%).

The Pearson's Chi-square test showed that hyperkeratosis was nonsignificant ( $P > 0.05$ ), as it was observed in 100% of hypertrophic LP and LPP cases and 96% of patients in classic LP. However, the presence of hypergranulosis, acanthosis, a band-like dermal lymphocytic infiltrate, atrophy, pigment incontinence, melanophages, and vacuolar degeneration with Civatte bodies was all found to be statistically significant ( $P < 0.05$ ). The presence of acanthosis was substantial ( $P < 0.05$ ), as it was seen in 90% of cases in classic LP and 96% in hypertrophic LP, while only 28% of cases of LPP showed acanthosis.

Out of 50 cases studied, 45 had a clinical diagnosis of LP, while only 6 had other clinical diagnoses. Of these 6 cases, 4 were diagnosed as hypertrophic LP on histopathology, and 2 were misdiagnosed as psoriasis clinically. Thus, clinical-histopathological concordance was seen in 90% of cases.

## Discussion

A total of 50 cases were analyzed in the present study. LP was found to be widely distributed in different age groups ranging from 4 to 79 years in males and 10 to 70 years in females. Most patients presented in the age group 30–39 years with a mean age of presentation of  $34.89 \pm 16.02$  years. This is in concordance with other Indian studies conducted by Kachhawa *et al.* [10] (20–39 years), Nasreem and Ahmed [11] (20–40 years), Parihar *et al.* [12] (21–40 years), and Durgaraju *et al.* [13] (35–44 years). However, a study by Bhattacharya *et al.* [14] showed the most typical age group to be 40–49 years. It has been suggested that a younger age group is affected in tropical countries and that familial LP, though rare, also occurs at a younger age. [9,15,16] However, a review of western literature reveals younger age, particularly children, to be less commonly affected. [17,18] This difference in age distribution can be attributed to unidentifiable geographical and genetic factors.

The present study showed a slight male preponderance with a male:female ratio of 1.3:1. This is in concordance with the studies done by Kachhawa *et al.* [10] (1.4:1), Nasreem and Ahmed [11] (1.6:1), Durgaraju *et al.* [13] (1.5:1), and Singh and Tosti [19] (1.5:1). Our findings are, however, in contrast to a study done by Parihar *et al.*, [12] where female preponderance was seen with a male:female ratio of 0.8:1. Studies by Srivani *et al.* [20] and Rajalakshmi *et al.* [21] showed that both sexes were equally affected.

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We found the most common variant of LP to be classic LP, followed by hypertrophic LP and LPP. These findings concord with the various other studies done by Bangaru and Karibasappa, Palakurthi *et al.* and Maisnam *et al.* [22–24] This difference in the distribution of cases can be attributed to differences in geographical distribution, climate, and genetic factors.

The most common site of involvement was the lower limb in classic and hypertrophic LP. Studies done by Palakurthi *et al.*, Tickoo *et al.*, Abdallat and Maaita, and Bhutani *et al.* [23, 25–27] also documented the lower limbs to be the most standard site. Venous stasis has been implicated as a likely pathogenic mechanism for the joint involvement of the legs. However, in contrast to predominant lower limb involvement in most cases of LP, upper extremities were involved in a higher proportion of patients. Clinically, pruritus was a significant complaint in majority of the patients. [23] Similarly Ireddy and Udbalkar [28] reported pruritus in 74%, 79.3%, and 82.6% of their cases, respectively. Thus, pruritus is a hallmark feature of LP. These findings were also observed by Rambhia *et al.* [29] and Kumar *et al.* [30]

Studies done by Arora *et al.* [31] and Rajalakshmi *et al.* [21] also supported similar results. Histopathological examination is the gold standard for in LPP. These observations were also found by Nasreem and Ahmed [11] and Parihar *et al.* [12]

The present study showed clinical-histopathological concordance in 90% of cases. This finding was consistent with other studies by Arora *et al.*, [31] Srivani *et al.*, [20] and Maisnam *et al.*, [24] with concordance rates of 92%, 81.6%, and 81%, respectively.

## Conclusion

LP is an inflammatory papulosquamous disease affecting the skin, mucous membranes, and nails, which may present with several morphological variants that can pose a diagnostic dilemma for dermatologists and dermato-pathologists. Clinical features of LP, such as violaceous polygonal papules with Wickham's striae, give important clues to diagnosis. However, histopathological examination is considered the gold standard for definitive diagnosis of LP. Most of the characteristic histopathologic features of LP, such as hyperkeratosis, irregular acanthosis, and band-like lymphocytic infiltrate, were regularly encountered in our study. It can, therefore, be emphasized that combining clinical and histopathological findings can help accurately categorize and adequately manage these cases.

## References

1. Arndt KA. Lichen planus. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM, Austen KF,

- editors. *Dermatology in General Medicine*. New York: McGraw-Hill; 1993. p. 1134-43.
2. Wilson E. On lichen planus. *J Cutan Med Dis Skin* 1869;3:117-32.
  3. Lehman JS, Tollefson MM, Gibson LE. Lichen planus. *Int J Dermatol* 2009;48:682-94.
  4. Rivers JK, Jackson R, Orizaga M. Who was Wickham, and what are his striae? *Int J Dermatol* 1986;25:611-3.
  5. Breathnach SM, Cox NH, Griffiths CEM. Lichen planus and lichenoid disorders. In: Burns T, Breathnach SM, editors. *Cox Neil. Rooks Textbook of Dermatology*. 8th ed. West Sussex: Blackwell Publishing; 2010. p. 307-24.
  6. Puri N, Puri A. A study on lichen planus in children. *Our Dermatol Online* 2013;4:303-5.
  7. Brice SL, Barr RJ, Ratter JP. Childhood lichen planus—A question of therapy. *J Am Acad Dermatol* 1980;3:370-6.
  8. Pinkus H. Lichenoid tissue reactions. A speculative review of the clinical spectrum of epidermal basal cell damage with special reference to erythema dyschromicum perstans. *Arch Dermatol* 1973;107:840-6.
  9. Black MM. Lichenplanus and lichenoid disorders. In: Champion RH, Burton JL, Burns BA, Breathnach SM, editors. *Textbook of Dermatology*. Vol. 3, 6th ed. Oxford: Blackwell Scientific; 1998. p. 1675-98.
  10. Kachhawa D, Kachhawa V, Kalla G, Gupta LP. A clinical-aetiological profile of 375 cases of lichen planus. *Indian J Dermatol Venereol Leprol* 1995;61:276-9.
  11. Nasreem S, Ahmed I. Associations of lichen planus: A study of 63 cases. *J Pak Assoc Dermatol* 2007;17:17-20.
  12. Parihar A, Sharma S, Bhattacharya SN, Singh UR. A clinicopathological study of cutaneous lichen planus. *J Dermatol Dermatol Surg* 2015; 19:21-6.
  13. Durgaraju S, Katakam N. A clinical-histopathological study of lichen planus. *Int J Health Clin Res* 2020;3:165-8.
  14. Bhattacharya M, Kaur I, Kumar B. Lichen planus: A clinical and epidemiological study. *J Dermatol* 2000;27:576-82.
  15. Mahood JM. Familial lichen planus: A report of nine cases from four families with a brief literature review. *Arch Dermatol* 1983;119:29 2-4.
  16. Saffron MH. Familial lichen planus: Report of four cases of lichen planus in one. *Arch Derm Syphilol* 1940;42:653-5.
  17. Scully C, el-Kom M. Lichen planus: Review and update on pathogenesis. *J Oral Pathol* 19 85;14:431-58.
  18. Andreassen JO. Oral lichen planus. I. A clinical evaluation of 115 cases. *Oral Surg Oral Med Oral Pathol* 1968;25:31-42.
  19. Singh OP, Tosti AJ. Lichen planus in India: An appraisal of 441 cases. *Int J Dermatol* 19 76;15:752-6.
  20. Srivani N, Sravani BVN, Srujana S, Kumar OS. A study of clinical and histopathological correlation of lichen planus. *Int Arch Integr Med* 2017;4:136-44.
  21. Rajalakshmi V, Anbukkarasi K, Raga Priya D. Clinicopathological profile of cutaneous lichen planus: An experience from a tertiary care centre. *Indian J Pathol Oncol* 2020;7:295-8.
  22. Bangaru H, Karibasappa N. Clinical and histopathological study of 50 cases of lichen planus. *IP Indian J Clin Exp Dermatol* 2016;2:36-9.
  23. Palakurthi SS, Seetharamanjanyulu K, Ramana GV, Saya S. A cross-sectional study of lichen planus: Its epidemiological, clinic-histopathological and serological perspective. *IP Indian J Clin Exp Dermatol* 2020;6:57-61.
  24. Maisnam J, Kumar N. Lichen planus—A clinical and histopathological correlation. *Trop J Pathol Microbiol* 2018;30:408-14.
  25. Tickoo U, Bubna AK, Subramanyam S, Veeraraghavan M, Rangarajan S, Sankarasubramanian A. A clinicopathologic study of lichen planus at a tertiary health care centre in south India. *Pigment Int* 2016;3: 96-101.
  26. Abdallat SA, Maaita TJ. Epidemiological and clinical features of lichen planus in Jordanian patients. *Pak J Med Sci* 2007;23:92-6.
  27. Bhutani LK, Bedi TR, Pandhi RK, Nayak NC. Lichen planuspigmentosus. *Dermatologica* 197 4;149:43-50.
  28. Ireddy SG, Udbalkar SG. Epidemiological study of lichen planus. *BMR Med* 2014;1:1-9.
  29. Rambhia KD, Kharkar V, Pradhan V, Patwardhan M, Ghosh K, Khopkar US. A study of the prevalence of autoantibodies in patients with lichen planus from Mumbai, India. *Indian J Dermatol Venereol Leprol* 2018;84:667-71.
  30. Kumar SA, Krishnam Raju PV, Gopal K, Rao TN. Comorbidities in lichen planus: A case-control study in Indian patients. *Indian Dermatol Online J* 2019;10:34-7.
  31. Arora SK, Chhabra S, Saikia UN, Dogra S, Minz RW. Lichen planus: A clinical and immuno-histological analysis. *Indian J Dermatol* 2014;59:257-61.