

# Lichen Planus – A clinical and histopathological correlation

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## Abstract

**Introduction:** History and characteristic examination findings are often sufficient to diagnose cutaneous lichen planus. Although lichen planus has distinctive clinical features, the diagnosis may present a problem due to variations in clinical pattern. Skin biopsy may be useful to confirm the diagnosis and is of ten required in a typical presentation. In all cases, it is important to consider the possibility of the eruption being drug induced. Lichen planus is not an infectious disease. **Aim:** Main aim of this study was to correlate clinical features with histopathologic study in all clinically diagnosed and suspected cases of lichen planus and to know its clinical and histopathological variants and assess the clinical versus the pathological agreement in diagnosis. **Materials and methods:** A prospective cross-sectional study was conducted with clinico pathological examination of skin biopsy specimens in the Department of Pathology, Vydehi institute of medical sciences and research centre over a period of 2 years between 2010–2012. **Statistical analysis:** This study demonstrated no significant association between variants of LP and sex of the patient ( $p > 0.05$ ) with  $\chi^2 = 5.92$ ,  $0.05 < p < 0.10$  using the probability level (alpha) and degree of freedom (df=1). **Results:** 60 cases of lichen planus were studied. 49 cases were confirmed on histological examination. 11 cases were diagnosed only on histology. Maximum number of cases occurred in the age group of 18 - 50 years. Males were affected more commonly than females. **Conclusion:** The possibility of this lesion to turn malignant justifies the importance of long term follow up for patients with such disease. Clinico pathological correlation is the key to confirm the diagnosis for further patient care and treatment.

**Keywords:** Lichen planus, Basal cell damage, Civatte bodies, Wickham's striae, Dermo epidermal junction

## Introduction

The classical histological picture was first clearly described by Darier in 1909. Later Pinkus (1973) defined lichenoid tissue reactions as those exhibiting epidermal basal cell damage as the primary event which then initiates the cascade of changes which are seen and recognized in the fully developed LP histopathology. A viral etiology has always been an attractive theory, however numerous electron microscopic or virus isolation studies have failed to provide convincing proof that lichen planus is induced by a virus infection [1].

Lichen Planus is subacute or chronic dermatoses that may involve skin, mucous membrane, hair follicles and nails. Pruritic, Polygonal, Planar (flat topped), Purple Papules and Plaques are the six 'Ps' of lichen planus. The disease has a predilection for the flexor surface of the forearms, legs and glans penis. Eruption maybe localized or extensive and Koebner's phenomenon is commonly seen.

Lichen Planus is the prototype of lichenoid interface dermatitis in which the infiltrate comprises mainly lymphocytic population. Lichenoid interface dermatitis is one of the 2 major inflammatory patterns that primarily involve the epidermal basal zone, hence the use of the term interface. The other pattern is vacuolar interface dermatitis. These two patterns can be difficult to separate at times and both changes may be present in same lesion.

In most of these diseases, T lymphocytes infiltrate the basal layer of the epidermis and cause cytotoxic damage to or kill keratinocytes by the induction of a form of cell death known as apoptosis. Apoptotic keratinocytes become detached from their neighbors find their way into the papillary dermis – known as colloid, cystoids, or civatte bodies. The expression of Fas R/ FasL by the basal keratinocytes suggests that apoptosis is an important mode of cell death in LP [2]. The etiology of Lichen planus is unknown. Theories of infections including viral, bacterial, autoimmune, metabolic, psychosomatic and genetic causes have all had their

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proponents. Current evidence suggests that lichen planus is an immunological disease which is thought to represent an abnormal delayed hypersensitivity reaction to an undetermined epidermal neoantigen. Cytotoxic CD8+ cells in the lesional epidermis recognize a unique antigen called lichen planus specific antigen (LPSA). It remains unknown, however, how such auto aggressive T cells could be activated *in vivo* to cause epidermal damage.

## Materials and Methods

This is a prospective cross-sectional study, undertaken in the department of Pathology, Vydehi Institute of Medical Sciences and Research Center (VIMS & RC), White field, Bangalore for duration of 2 years from May 2010 to May 2012. The study population included both male and female patients aged between 18- 65yrs who were clinically diagnosed of Lichen Planus and lichen planus like eruptions from the department of Dermatology, VIMS and RC. Relevant clinical history including age, socio economic status, duration of the lesion, site of the lesion, significant personal and family history, history of any drug intake, history of associated diseases was taken and entered in the proforma.

### Inclusion criteria

Both male and female patients aged between 18- 65 years clinically diagnosed of Lichen Planus and lichen planus like eruptions.

## Results

I. The duration of study was 2 years from May 2010 to May 2012. Total numbers of biopsy inclusive of LP and LP like lesions were 85. Of the total 60 cases, 33 were males and 27 were females.

Out of the 60 cases, 37 (61.6%) cases were of classical lichen planus, 8(13.3%) cases were of hypertrophic lichen planus, 5(8.3%) cases were of lichen planus pigmentosus, 4(6.6%) cases were of actinic lichen planus, 3(5%) cases were of eruptive lichen planus, 1(1.6%) case each of atrophic lichen planus, annular lichen planus and lichen planus of buccal mucosa [Table: 1]. None of the patients had family history of similar lesions. There were no nail, hair and genital involvement. Of the total 60 cases o 49 cases were confirmed on histological examination. 11 cases were diagnosed only on histology [Table: 2]. Their ages ranged from 18 to 65 years (mean 37.1yrs  $\pm$  SD 12.8yrs) [Table: 3].

**Table-1: Distribution of the cases.**

Sl. No.	Lesions	No. of cases	Percentage
01	Classical LP	37	61.6%
02	Hypertrophic LP	8	13.3%
03	Lichen Planus Pigmentosus	5	8.3%
04	Actinic LP	4	6.6%
05	Eruptive LP	3	5%
06	Atrophic LP	1	1.6%
07	Annular LP	1	1.6%
08	Lichen Planus of buccal mucosa	1	1.6%

### Exclusion criteria

1. Patients who are showing clinical features similar to Lichen Planus but not proven histologically.
2. Patients giving history of skin eruptions that occur after ingestion, contact or inhalation of certain chemicals like gold salts, beta blockers, antimalarials, thiazide diuretics, furosemide, spironolactone and penicillamine.
3. Pregnant women.

A detailed general and local examination was done and the site for biopsy was selected. The selected patients were explained about the procedure of the biopsy and informed written consent was taken.

### Statistical Methods Used for Data Analysis

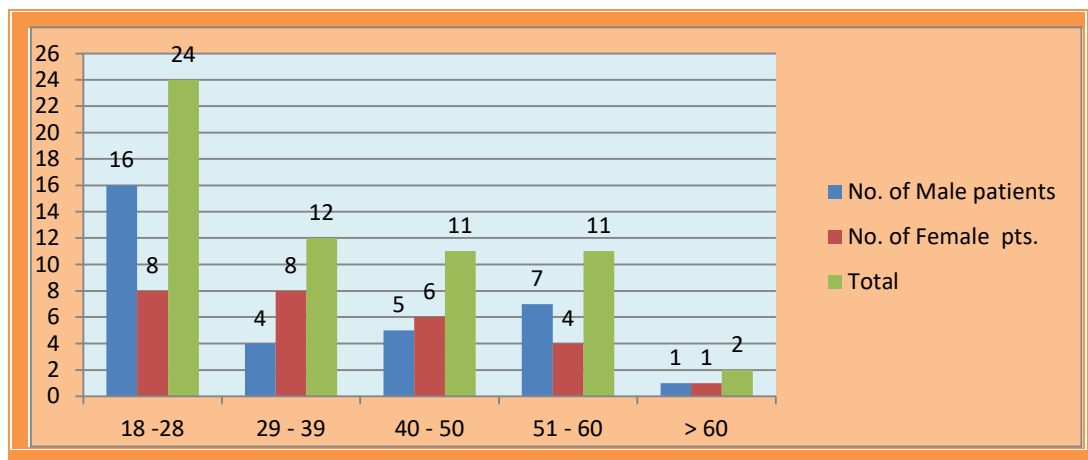
1. The statistical data were studied using the percentage (%) and proportion.
2. Association of the histopathological patterns across the LP and sex was compared using Chi square test.
3. Sensitivity and specificity tests were used in assessing the clinical diagnostic accuracy with lesions resembling LP and correlate histopathologically for accurately diagnosing LP.

**Table-2: Clinical and histological correlation.**

Correlation	No. of cases	Percentage (%)
Histological confirmation	49	81.6
Diagnosed only on histology	11	18.4

**Table-3: Age Distribution.**

Age group	No. of Case	Percentage
18-28 yrs	24	48%
29-39 yrs	12	20%
40-50 yrs	11	18.3%
51-60 yrs	11	18.3%
Above 60 yrs	02	3.4%
Age group	No. of Case	Percentage
18-28 yrs	24	48%
29-39 yrs	12	20%
40-50 yrs	11	18.3%
51-60 yrs	11	18.3%
Above 60 yrs	02	3.4%



**Figure-1: Age and Sex distribution**

The mean duration of the lesion was 12.02 months (approx 1yr). Of the total 60 cases, 33 were males and 27 were females.

**Discussion**

In this study, the maximum number of cases 37 (61.6%) were those of classical lichen planus followed by hypertrophic lichen planus, 8 cases (13.3%). The other variants found are lichen planus pigmentosus, 5 cases (8.3%), Actinic lichen planus, 4 cases (6.6%), eruptive lichen planus, 3 cases (5%). One cases each of atrophic, buccal, and annular lichen planus [Fig: 1]. Similar observation were found in other studies [3, 4].

Kachhawa et al (1995) found the incidence of lichen planus in western India as 0.8% [5].

LP actinicus (LPA) is a distinct variant of LP also called subtropical LP and elanodermatite lichenoides. It occurs mainly in the Middle-East and predominantly on sun exposed areas of the skin. Its reported incidence in India is between 0.4 to 19.2%. In the present study, LPA comprised 6.6% of all cases. Lichen planus affects both sexes. In the present study males were affected slightly more than the females. Few other studies have suggested a male predominance.

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The difficulty in the diagnosis of the lesions on clinical levels resulted from their non-specific features, and/ or similar clinical appearance and/ or lack of criteria of correct clinical diagnosis in some lesions.

Typical papule of LP show –

Compact orthokeratosis

Irregular acanthosis

Wedge shaped hypergranulosis

Vacuolar alteration of the basal layer

A band like dermal lymphocytic infiltrate in close approximation to the epidermis

This constellation of findings is sufficiently diagnostic that a histologic diagnosis can be rendered in more than 90% of the cases. Dermal changes are characterized by a band-like inflammatory infiltrate predominantly of lymphocytes with a few macrophages hugging the dermo-epidermal junction. The inflammatory infiltrate is predominantly perivascular. The differing clinical and histopathological diagnoses may be partly due to the insufficient information given by the patients and partly due to non-availability of the histological slides.

There are many variants of lichen planus. Whether these represent separate diseases or part of lichen planus spectrum is unknown. They all demonstrate typical lichen planus histologically and are described separately since their clinical features are distinct from classical lichen planus.

The present study showed a male preponderance (55%) as compared to females (45%). OP Singh and Kanwar AJ also found male preponderance (3:2) in 1976. In a similar study done by Kachhawa et al also found 58.7% cases of male in a total of 375 patients of lichen planus in 1995. However, in a study by Bhattacharya M et al in 2000, both sexes were equally affected [Table: 5]. The present study has shown that most of the patients were in the age group 18-39 years. Singh and Kanwar found most patients having the disease in the 3rd decade of life.

Kachhawa et al found maximum number of cases in 20-39 years age group [5].

**Table-4: Comparison of distribution of disease in male and female patients**

Study	Year	Male/Female ratio
Singh and Kanwar	1976	0.5
Kachhawa et al	1995	1.42
Bhattacharya M et al	2000	1.0
Present study	2012	0.8

**Table-5: Showing comparison of age distribution of disease**

Study	Year	Common age group
Sehgal VN, Gege VL	1973	31-40yrs
Singh and Kanwar	1976	30-39 yrs
Kachhawa	1995	20-39 yrs
Bhattacharya M	2000	20-49yrs
Present study	2012	18-39 yrs

Pediatric cases were not included in the study as the study material was available from adult patients only. This study found no association of the disease with occupation, educational background of the patients, and degree of physical activity. There was no seasonal variation in the incidence or disease progress of lichen planus which is similar to observations found by other workers [6].

Kachhawa et al found the maximum number of cases registered during February to September. Seasonal variation does occur in tropical countries with peak being during the rainy season, April to September (65%)[7].

## Original Research Article

Majority of the patients presented with moderate to severe degree of itching. Bhattacharya M et al also observed similar views in which they observed 79.3% [8] had predominant symptoms of itching as compared to 75% in the present study. In the present study none of the patients gave any family history of similar lesion. An eruption of lichen planus may abate completely within a few months may remit partially and at irregular intervals over months to years or may progress into chronic lichen planus. Persistence of the disease seems related to the presence of mucous membrane lesions. Those patients with only cutaneous involvement clear more promptly. Familial LP differs from the classical form clinically, with earlier age at onset, more generalized involvement, and more common mucosal involvement. The prevalence of familial LP in a large series has been reported to be 1.5% [9].

Kachhawa et al reported familial LP in 8 families (1995) in western Rajasthan. However, none of the patients in the present study gave history of any precipitating cause of the disease similar to other studies done by other workers [10,11]. The role of emotional factors in the development of lichen planus is controversial. Psychogenic factors appear to be important in many patients and psychic stress has been related to the onset of the disease, the precipitation of additional attacks and resolution once the stressful factors are removed. Such events are most probably circumstantial. Lichen planus has also been reported in association with diabetes mellitus, myasthenia gravis (MG) and thymoma [12].

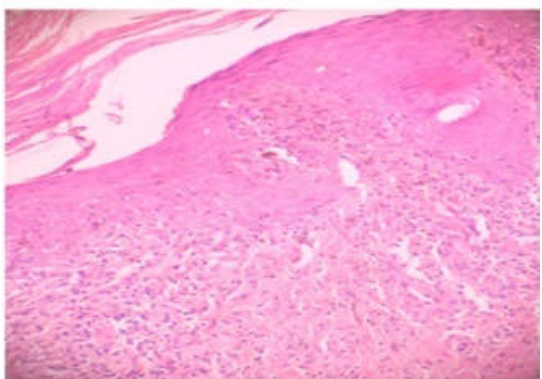
Rachael Mathai et al in 1983 reported a case of linear lichen planus in a child for an unusual histological feature, namely the presence of multiple foreign body giant cells in the inflammatory infiltrate [13]. Lichen planus is considered to be rare in children. However, it does not appear to be uncommon in Indian subcontinent. Most of the large studies have been reported from India, the largest one by the authors in 2009 involving 100 children below 18 years of age seen over 6.5 years [14]. Lichen planus affects one or more nails in 10% of cases, sometimes without involving the skin surface. If all nails are abnormal and nowhere else is affected it is called twenty nail dystrophy. It is a rare acquired idiopathic nail dystrophy characterized by excess longitudinal ridging, distal notching, splitting, and loss of nail luster and thinning of nail plates that may affect 1 to 20 nails [15].



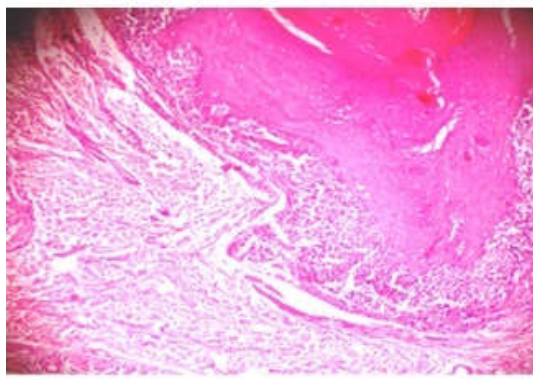
**Figure 2: Lichen Planus of the upper arm**



**Fig 3: Hypertrophic Lichen Planus– raised, warty violaceous plaques**

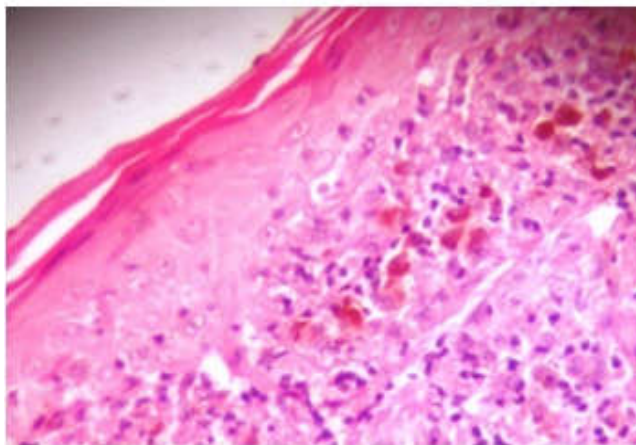


**Fig 4: Lichen planus–10x showing basal layer degeneration and civatte bodies**



**Fig 5: Hypertrophic Lichen Planus – 10x showing marked hyperkeratosis, acanthosis and band shaped inflammatory infiltrate**





**Fig 6: Lichen Planus pigmentosus showing epidermal thinning, basal cell degeneration, melanin pigment incontinence and lymphohistiocytic infiltrate in the dermis (10x H and E)**

## Conclusion

Lichen planus and Lichenoid tissue reaction are clinically and histo-pathologically very similar but have different treatment and prognosis. Lichenoid tissue reaction is characterized by epidermal basal cell damage which takes the form of liquefaction degeneration or cell death, either by apoptosis or necrosis with an associated cascade of histological events in epidermis and dermis.

The term "lichenoid" refers to papular lesion of certain skin disorders of which lichen planus is the prototype. However, this type of reaction can also be seen in skin disorders associated with systemic illnesses like lupus erythematosus and the skin changes of potentially fatal disorders such as graft versus host disease, Stevens Johnson syndrome and toxic epidermal necrolysis.

The histopathologic findings are a crucial clue to help clarify LP.

Early diagnosis and treatment are key to prevent wide spread involvement and differentiate from other skin lesions. Treatment options depend on disease severity in terms of symptoms and extent of involvement. Topical, intralesional and systemic therapies singly or in combination may be necessary. Although there are new definitive curative modalities, new discoveries and conceptual advances continue to broaden our treatment options of this rather complex condition.

Recognition and diagnosis of the atypical variants require clinicopathologic correlation and the reviewing pathologist should be aware of the clinical presentation of the lesions. Lack of clinico pathologic correlation may lead to inconclusive diagnosis which may alleviate the patient's anxiety and suboptimal treatment. Every

specimen submitted should be accompanied by patient clinical information including the differential diagnosis.

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**Permission from IRB:** Yes

## References

1. Black MM. What is going on in lichen planus? Clin Exp Dermatol. 1977 Dec;2(4):303-10.
2. Sherry M Farley, Lisa J Wood. An Epidermotypic Model of Interface Dermatitis Reveals Individual Functions of Fas Ligand and Gamma Interferon in Hypergranulosis, Cytoid Body Formation, and Gene Expression, Am J Dermatopathol, 2011;33(3): 244-250.
3. Singh OP, Kanwar AJ. Lichen planus in India: an appraisal of 441 cases. IntJ Dermatol. 1976 Dec;15:752-6
4. Garg VK, Nangia A, Logani K, Sharma RC. Lichen Planus-a Clinico-histopathological. Indian J Dermatol VenereolLeprol. 2000 Jul-Aug;66(4):193-5.
5. Dilip Kachhawa, Vandana Kachhawa, G Kalla. A clinico-etiological profile of 375 cases of lichen planus Indian J Dermatol VenereolLeprol. 1995, 61; 5:276-279.
6. Sehgal VN, Rege VL Lichen planus: an appraisal of 147 cases Indian J Dermatol VenereolLeprol. 1974; 40:104
7. Alabi GO, Akinsanya JB. Lichen planus in tropical Africa. Trop Geogr Med. 1981 Jun;33(2):143-7.
8. Bhattacharya M, Kaur I, Kumar B. Lichen planus: a clinical and epidemiological study. J Dermatol. 2000 Sep; 27 (9):576-82.

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9. ALTMAN J, PERRY HO. The variations and course of lichen planus. Arch Dermatol. 1961 Aug;84:179-91.

10. Kanwar AJ, Handa S, Ghosh S, Kaur S. Lichen planus in childhood: a report of 17 patients. Pediatr Dermatol. 1991 Dec;8(4):288-91.

11. Handa S, Sahoo B. Childhood lichen planus: a study of 87 cases. Int J Dermatol. 2002 Jul;41(7):423-7.

12. Sarwat Nasreen, Ijaz Ahmed, Zarnaz Wahid. Associations of lichen planus: A study of 63 Cases Journal of Pakistan Association of Dermatologists. 2007; 17(1): 17-20.

13. Rachel Mathai, Susan Ebenezer, Adolf Walter. Unusual Histological Feature in a Case of Lichen Planus. Indian J Dermatol Venereol Leprol. Vol 49, 1983; 4: 168-171.

14. Kanwar AJ, De D. Lichen planus in childhood: report of 100 cases. Clin Exp Dermatol. 2010 Apr; 35 (3): 257-62. doi: 10.1111/j. 1365-2230.2009.03613.x. Epub 2009 Oct 23.

15. Lee YB, Cheon MS, Park HJ, Cho BK. Clinical study of twenty-nail dystrophy in Korea. Int J Dermatol. 2012 Jun;51(6):677-81. doi: 10.1111/j.1365-4632. 2011.05118.x. Epub 2011 Sep 14.

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