


Original Research Article

# A study of clinical and histopathological correlation of lichen planus

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## Abstract

**Introduction:** Lichenoid tissue reaction or interface dermatitis embraces several clinical conditions, the prototype of which is lichen planus and its variants. Others include drug induced lichenoid dermatitis, special forms of lichenoid dermatitis, lichenoid dermatitis in lupus erythematosus, and other disorders all of which have salient clinical and histological features. The aim of this study was to correlate the clinical features with histopathological findings in all clinically suspected cases of lichen planus and assess the clinical diagnostic accuracy.

**Materials and methods:** This study had been carried out over a period of 2 years from July 2015 to June 2017 in the Department of Pathology, Gandhi Hospital. Histopathological sections of skin biopsies from 85 patients were studied after routine H and E staining and morphological changes were noted. The patients' clinical findings were then correlated with the histological changes. The results were then analysed.

**Results:** Total numbers of biopsies inclusive of LP and LP like lesions were 85. Among them, the number of patients having LP was 50. The age ranged from 18 to 65 years (mean 37.1 years). The mean duration of the lesion was 12.02 months (approx 1 year). Males were affected more than females. Out of the 85 cases, 50 were Lichen planus and 35 were lichenoid eruptions. Of the Lichen planus, 31 (61.6%) cases were of classical lichen planus, 6 (13.3%) cases were of hypertrophic lichen planus, 4 (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.

**Conclusion:** In the present study, maximum number of cases occurred in age group of 18-28 years. Though pruritis is a common symptom, it is nonspecific. Therefore, histopathology remains the most important diagnostic tool for proper evaluation of Lichenoid eruptions/ Lichen planus.

## Key words

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Lichen Planus, Lichenoid dermatitis, Skin biopsy, Pruritis.

## Introduction

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Lichenoid interface dermatitis is defined by Destruction of the basal keratinocytes and band like lymphocytic infiltrate of varying density localised to the papillary dermis [1-4].

Careful examination of the BM zone is essential in distinguishing between the 2 patterns:

- Dyskeratosis predominates in the lichenoid pattern whereas vacuolisation of the basal keratinocytes is the hallmark of vacuolar pattern [5-7].
- A combination of both lichenoid and vacuolar interface changes may be present in certain dermatoses, such as Lupus erythematosus [8].

In most of these diseases, T lymphocytes infiltrate the basal layer of the epidermis and cause cytotoxic damage or apoptosis of keratinocytes.

Apoptotic keratinocytes become detached from their neighbours, become round and undergo degradation of their nuclear DNA, lysis of their nuclei and coagulation of proteins in their cytoplasm. Such dyskeratotic cells find their way into the papillary dermis – known as colloid, cystoids, or civatte bodies.

The etiology of Lichen planus (LP) is unknown. Theories of infections including viral, bacterial, autoimmune, metabolic, psychosomatic and genetic causes have all had their 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 [9].

Cytotoxic CD8+ cells in the lesional epidermis recognize a unique antigen called lichen planus specific antigen (LPSA).

In one study, this antigen was demonstrated by indirect immunofluorescence using the patient's serum and autologous lesional skin.

Lichen planus may be found with other diseases of altered immunity like ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, morphea, lichen sclerosis, and SLE. The association of LP with hepatitis C virus has been studied by many authors. LP is associated with a variety of liver cell abnormalities including aberrant liver function tests and serology. A significant association between LP and HLA DR1 and HLA DQ1 has been noted by number of authors. This association pertains to patients with or without mucosal lesions but does not extend to patients with the drug induced variant. Keratinocytes express HLADR1 and antigen presentation to T helper cells results in the development of an autoimmune response [10].

Subsequent migration with resultant CD8+ T cell activation results in basal keratinocytes death due to the combined effects of IFN- $\gamma$ , IL-6, GM-CSF and TNF- $\alpha$ . The expression of Fas R/ FasL by the basal keratinocytes suggests that apoptosis is an important mode of cell death in LP. The dermal infiltrate consists predominantly of CD4+ lymphocytes. CD8+ lymphocytes are also present in close apposition to the dermo-epidermal junction adjacent to foci of basal keratinocytes necrosis and are said to predominate in early lesions [11].

B-lymphocytes are scarce and plasma cells are characteristically absent in cutaneous lesions. Development of the typical papule appears to be due to a combination of continued keratinocytes destruction and regenerative activity, the later depending upon migration of epithelium from the edge of the lesion and from adjacent eccrine ducts rather than from increased mitotic activity.

The typical features of LP depend on a variable interplay between basal cell liquefactive degeneration and irregular epidermal regeneration.

The earliest identifiable change in LP is the presence of Cytoid bodies and associated pigmentary incontinence. They display diastase resistant periodic acid Schiff (PAS) positivity and may be identified within papules, pre lesional skin and even apparently normal skin.

Lichen planus has also been reported in association with diabetes mellitus, myasthenia gravis (MG) and thymoma [12].

### **Histopathology of LP**

Typical papule of LP show—Compact orthokeratosis, Irregular acanthosis, Wedge shaped hypergranulosis, Vacuolar alteration of the basal layer and a band like dermal lymphocytic infiltrate in close approximation to the epidermis.

Epidermal changes are characterized by compact hyperkeratosis (but not parakeratosis), focal hypergranulosis which is uneven and wedge shaped, acanthosis with toothing of rete ridges and basal cell liquefaction. Some authors have revealed ultrastructural investigations in patients with lichen planus. The most consistent findings were (1) multiplication, irregular folding or dislocation of the basal lamina, (2) fragmentation with degenerative changes of basal keratinocytes, (3) formation of numerous fibrillar Civatte bodies and, (4) presence of dyskeratotic elements. In addition, mitotic figures of keratinocytes and Langerhans cells were observed. These findings support the view that

the primary event in LP represents an injury involving epidermal basal cells.

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 [13, 14].

Civatte bodies which are round, eosinophilic bodies in the lower epidermis and papillary dermis may also be observed. They are PAS +ve but diastase resistant [15-18].

WS is an important diagnostic sign of LP and should always be looked for when confused or lesions coexist with similar scaly dermatoses. At present it is agreed that Wickham's striae is due to increase in the granular cell layer in the epidermis.

### **Aim and objectives**

- To correlate clinical features with histopathological study in all clinically suspected cases of lichen planus.
- To know the clinical and histopathological variants of lichen planus and assess the clinical diagnostic accuracy by histopathology.
- To find out age and sex distribution of various types of lichen planus.

### **Materials and methods**

The study included skin biopsies from both male and female patients aged between 18-65 years who were clinically diagnosed of Lichen Planus and lichen planus like eruptions from the department of pathology and Dermatology, Gandhi hospital.

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 were taken and entered in the proforma.

### Selection criteria

#### Inclusion criteria

- Both male and female patients aged between 18-65 years clinically diagnosed of Lichen Planus and lichen planus like eruptions.
- Relevant clinical history including age, socio economic status, duration, site of the lesions, significant personal, family and occupational history were taken.
- Biopsy size  $\geq 4$  mm (punch biopsy) were taken.

#### Exclusion criteria

- Patients aged  $< 18$  years.
- Patients already diagnosed with Lichen Planus and clinically suspected cases of infectious dermatoses like Scabies, Leprosy, Molluscum contagiosum, Acne, Taenia etc.
- Biopsy size  $< 4$  mm

The biopsy specimen was submitted accompanied by detailed clinical information for histopathological examination.

Sections of the skin biopsy were stained with Haematoxylin and eosin stain and examined (Figure – 1 to 7).

**Figure - 1:** Lichen Planus - flat topped, violaceous purplish papules.



### Results

The duration of the study was 2 years from July 2015 to June 2017. Total numbers of biopsies inclusive of LP and LP like lesions were 85. Among them, the number of patients having LP

was 50. The age ranged from 18 to 65 years (mean 37.1 years  $\pm$  SD 12.8 years).

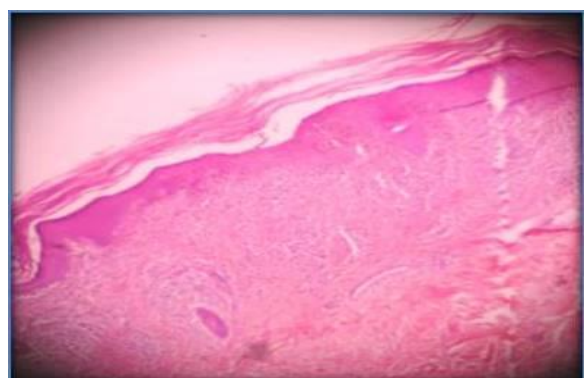
**Figure - 2:** Hypertrophic lichen planus - raised, warty violaceous plaques.



**Figure - 3:** Lichen planus showing Koebnerisation in a linear organization on thigh.



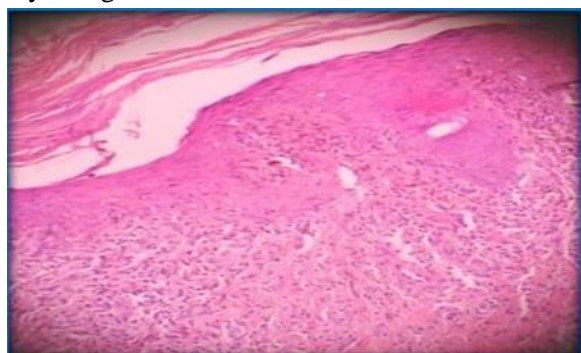
**Figure - 4:** Classical lichen planus: Scanner view showing hyperkeratosis, hypergranulosis, acanthosis and a band like inflammatory infiltrate.



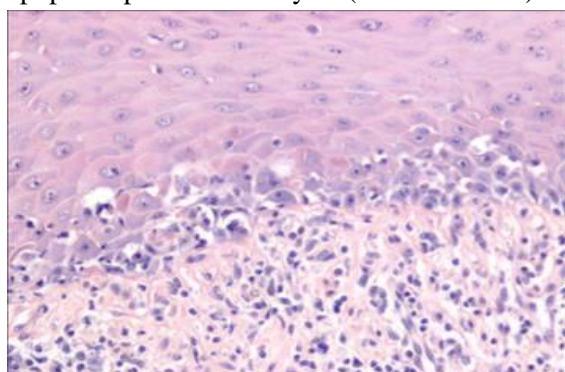
The mean duration of the lesion was 12.02 months (approx 1 year). Of the total 50 cases, 27 were males and 23 were females. Out of the 50 cases, 30 (61.6%) cases were of classical lichen planus, 6 (13.3%) cases were of hypertrophic lichen planus, 4 (8.3%) cases were of lichen

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. None of the patients had family history of similar lesions (Table – 1).

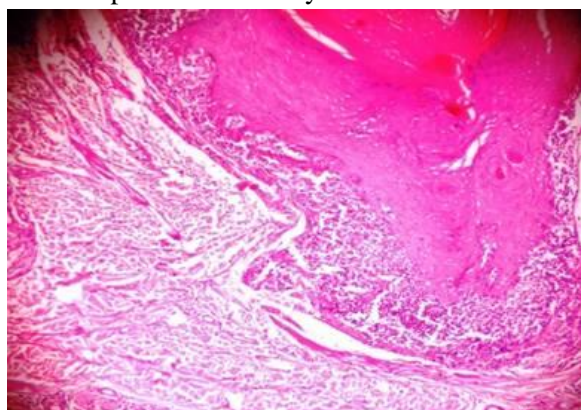
**Figure - 5:** Lichen planus – 10X showing basal layer degeneration and civatte bodies.



**Figure - 6:** 40X showing hydropic degeneration of the basal cell layer is evident with numerous apoptotic pink keratinocytes (Civatte bodies).



**Figure - 7:** Hypertrophic lichen planus – 10X showing marked hyperkeratosis, acanthosis and band shaped inflammatory infiltrate.



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

Lesions	No. of cases	%
Classical LP	30	61.6%
Hypertrophic LP	6	13.3%
Lichen Planus Pigmentosus	4	8.3%
Actinic LP	4	6.6%
Eruptive LP	3	5%
Atrophic LP	1	1.6%
Annular LP	1	1.6%
Lichen Planus of buccal mucosa	1	1.6%

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

Correlation	No. of cases	%
Histological confirmation	41	81.6
Diagnosed only on histology	09	18.4

Out of the 50 cases, 41 were either diagnosed as LP or LP like lesions so that the histopathological and clinical diagnoses were in agreement in 41 out of 50 cases (Table – 2). 09 cases were diagnosed only on histology – Of these Atrophic LP (1), Lichen planus pigmentosus (1), Hypertrophic LP (6 cases), Eruptive LP (1). The other differential diagnosis considered were Psoriasis, Discoid lupus erythematosus, Prurigonodularis, Kryle’s disease, Darier’s disease, Trichome vitiligo, Pityriasis, Granulomaannulare, lichenoid reaction, LSC, Erythema multiforme (EM), pseudopalade of brocq, polymorphic light eruption (PLE).

The present study showed sensitivity of 70% and a specificity of 50%. It detected 70% of the patients with the disease (true positives) but 30% would go undetected (false negative).

In the present study, maximum number of cases occurred in middle aged group of 18 to 50 years in which there were 25 cases of classical lichen planus, 5 cases of hypertrophic lichen planus, 3 cases each of lichen planus pigmentosus and

actinic lichen planus, 1 case of atrophic lichen planus and 2 cases of eruptive lichen planus. In the age group above 50 years, 5 cases were of classical lichen planus, 1 case each of hypertrophic lichen planus and eruptive lichen planus, 1 case each of lichen planus pigmentosus, lichen planus of the buccal mucosa and actinic lichen planus.

**Table - 3:** Age and Sex distribution of Lichen Planus.

Age in years	No. of male patients	No. of female patients	Total	Percentage
18-28	13	7	20	40%
29- 39	3	7	10	20%
40-50	4	5	09	18.3%
51-60	6	3	09	18.3%
Above 60	1	1	2	3.4%
Total	27	23	50	100%

**Table - 4:** Histopathological findings.

Epidermis	50 cases (%)	Dermis	50 cases (%)
Hyperkeratosis	38(75%)	Band like infiltration at dermo epidermal junction	43(86%)
Acanthosis	35(70%)	Predominantly lymphocytes	50(100%)
Atrophy	5(10%)		
Saw tooth rete ridges	27(53%)		
Civatte bodies	38(76%)		
Basal layer degeneration	50(100%)		
Melanin incontinence	45(90%)		

The oldest case was 63 years old man, with classical lichen planus on the abdomen and prurigonodularis in lower leg.

There were 27 males (55%) and 23 females (45%) of the total 50 cases (Table – 3).

#### Changes in the HP variants across males and females groups

Analysis of histopathologic variants within LP group showed maximum number (n=16, 31%) of patients in classical type followed by others (n= 08, 31%), 15(30%), 11(23%).

There was a slightly significant increase in classical variant in male patients (31%), followed by other variants in female patients.

In the age group of 18 -28 years there were 13 males and 7 females, in 29- 39 years age group -

3 males and 7 females, in 40-50 years age group - 4 males and 5 females, 51-60 years age group- 6 males and 3 females and 60 years and above age group – 1 male and 1 female.

#### Itching

Majority of the patients presented with moderate to severe degree of itching.

The itching was more intense, causing loss of sleep, exhaustion, and despair in the generalized form whereas in those varieties which consisted few patches of the lesion, the itching was mild except those with hypertrophic type which was intensely pruritic. It may be almost intolerable in acute cases.

Thirty eight of the sixty patients experienced mild to moderate itching to even severe forms depending upon the type of distribution of the lesions. Eleven patients did not have itching at all. They were cases of lichen planus

pigmentosus, hypertrophic, actinic and eruptive lichen planus.

One case each of atrophic and buccal lichen planus also did not have itching.

### **Family history**

None of the patients gave any family history of similar lesion.

### **Emotional factors**

None of the patients gave any precipitating factor before the onset of lesions.

### **Koebnerisation**

16 out of 50 cases complained of onset of new lesions along the line of scratching. It was a prominent feature in almost all the cases of classical lichen planus cases.

The most significant histopathological findings were basal layer degeneration and predominantly lymphocytic infiltration at the dermo-epidermal junction. These findings were seen in all the 50 cases and were the main diagnostic histological findings. The rest of the features like melanin incontinence, hyperkeratosis, band like infiltration at DEJ were found in varying numbers according to the variant.

### **Discussion**

In this study, the maximum number of cases 30 (61.6%) were those of classical lichen planus followed by hypertrophic lichen planus - 6 cases (13.3%). The other variants found are lichen planus pigmentosus- 4 cases (8.3%), Actinic lichen planus-4 cases (6.6%), eruptive lichen planus- 3 cases (5%), One case each of atrophic, buccal, and annular lichen planus. This is in concordance with other studies.

One study found the incidence of lichen planus in western India as 0.8%. LP actinicus (LPA) is a distinct variant of LP also called subtropical LP and elanodermatite lichenoid. 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 [18].

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

### **Conclusion**

Lichen planus and Lichenoid tissue reaction are clinically and histopathologically 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. Lichen planus and lupus erythematosus are the most common and best studied representatives of the lichenoid tissue reaction.

Lichenoid drug-induced eruptions can clinically and histological resemble idiopathic or classic lichen planus. Integrating drug history, clinical morphology, clinical distribution, and histopathology can aid in the differentiation. Emphasis should be given in taking a detailed history, including drug dosages and interval between drug ingestion and appearance of lesions, in order to make the correct differential diagnosis between lichenoid drug-induced eruptions and lichen planus. In the variants of lichen planus, the classical lichen planus is the most common, although other variants that can occur are hypertrophic lichen planus, lichen

planus pigmentosus, actinic lichen planus, atrophic lichen planus, and follicular lichen planus. Recognition and diagnosis of these 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.

The histopathologic findings are a crucial clue to help clarify LP. Early diagnosis and treatment are key to prevent widespread involvement and differentiate from other skin lesions.

Liason consultations between pathologist and dermatologist may be more helpful in dealing with lichen planus as an autoimmune disorder with no cure but counselling may help in ameliorating symptoms, improving quality of life and enhancing recovery.

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