

Clinico - Histopathological features of Lichen Planus-an Appraisal

Raghavendra B N¹, Jaffer Basha S K²

¹Associate Professor, Department of Dermatology, M V J Medical College and Research Hospital, Hoskote, Bengaluru, ²Assistant Professor Department of Dermatology, Shadaan Institute of Medical Sciences Peeranchuvuru, Hyderabad

Address for correspondence: Dr.Raghavendra B N, Associate Professor, Department of Dermatology, M V J Medical College and Research Hospital, Hoskote, Bengaluru.

Email- raghavderm@gmail.com

ABSTRACT

Background: Lichen planus is a common, chronic, papulosquamous disorder of obscure etiology. Though many etiological factors have been incriminated, autoimmunity has been increasingly accepted as the most likely etiology.

Objective: To study the clinical and histopathological features of lichen planus and to assess the extent of correlation.

Materials and Methods : 100 patients of lichen planus who consented were enrolled in this observational study by random selection. A detailed history, general physical examination and dermatological examination was done and recorded in a proforma. A 4mm punch biopsy was obtained in all study subjects and was sent for histopathological examination.

Results: Classical lichen planus was the most common morphological type observed in this study (44%), followed by hypertrophic lichen planus (12%). A clinico-histopathological correlation of 96% was observed in the study.

Conclusion: A high degree of clinico-histopathological correlation increases the sensitivity of diagnosis and helps in effective therapy.

Keywords: Autoimmunity, Lichen planus, Melanin incontinence.

INTRODUCTION

Lichen planus (LP) is a common, pruritic dermatoses that affects the skin, hairs, nails and mucus membranes. It is characterized classically by violaceous, scaly, flat topped, polygonal papules on flexural aspects¹. It runs a chronic course and lesions regress with post inflammatory hyperpigmentation, alopecia and scarring (over scalp)^{1,2}.

The exact prevalence of Lichen planus is unknown. Nevertheless, the estimated prevalence of LP is in the range of 0.22% to 5% worldwide³. LP typically affects middle-aged adults of both genders. No sexual predilection is evident but some reports indicate a slight predominance in women up to a ratio of 2:1⁴.

The etiopathogenesis of lichen planus is largely unknown. The current trend is to consider lichen planus as autoimmune disease. An increased association of LP and other autoimmune diseases such as ulcerative colitis, myasthenia gravis and diabetes mellitus further support this hypothesis⁵. Activated CD8+ T cells trigger basal cell apoptosis through perforin/ granzyme or Fas/ Fas ligand pathways. Keratinocyte damage by CD8+ T cells will lead to basement membrane disruption, paving way for the influx of more CD8+ T cells and further basal cell damage resulting in a vicious cycle leading to chronicity of the disease⁶.

The prognosis for LP is good, as most cases regress within 18 months. Hypertrophic LP and in lesions on the scalp can cause atrophy and scarring. Cutaneous LP does not carry a risk of skin cancer, but ulcerative lesions in the mouth, particularly in men, do have a low rate of malignant transformation. However, the malignant transformation rate of oral LP is low (< 2% in one report)⁷.

The literature search revealed a paucity of Indian studies on clinical and histopathological features of LP. Hence this study was planned to appraise the epidemiological, clinical and histopathological features of LP in Indian population.

MATERIALS AND METHODS

In this prospective observational study undertaken in a tertiary care center in Kolar, a total of 130 cases of LP were diagnosed clinically between September 2013 to March 2015 for a period of 18 months. 100 patients who consented were randomly selected and enrolled in the study after obtaining approval from institutional ethical committee. All clinically diagnosed cases of LP were included in the study with exclusion of patients less than 5 years of age. All the study subjects were subjected to a detailed clinical history and a meticulous clinical examination which was recorded in a proforma. A 4mm punch biopsy was performed in all patients and submitted for histopathological examination. The data was tabulated and analyzed using SPSS 20 version software.

RESULTS

A total of 24,312 patients attended the department of dermatology during the study period, out of which lichen

planus constituted 130 cases, contributing to 0.53% of the total skin disorders. The age and sex distribution of patients is shown in Figure 1. The most common age group observed was between 31-40 years (24%). The youngest patient was 5 years old and the oldest 85 years. Males comprised 54% and females 46%. The duration of LP was up to 3 months in 40% of patients, the shortest duration was for 10 days and the longest 3 years. The severity of itching was moderate in 44%, mild in 28% and 6% patients were asymptomatic.

LP presents with varied morphology and configuration (Figure 2). The commonest isolated morphological type was papular lichen planus (44%), followed by hypertrophic lesions (12%). The papular lesions coexisted with hypertrophic lichen planus in 10% of cases and in 4 cases of follicular and 2 cases of bullous lichen planus (Figure 3&4). A large proportion of patients had lesions distributed on trunks and limbs (36%), scalp was involved in 4%, palms and soles in 14%. The lesions were generalized in 8% of cases. Oral cavity, genitalia and nails were involved in 10%, 4%, and 16% respectively. LP manifested exclusively on skin in 86% patients. Both skin and mucus membrane were affected in 14%. Isolated mucus membrane involvement was not observed in our study. Oral cavity was the most common mucosal site involved in the present series, 6% patients had reticulate lesions, erosive and plaques were seen in 2 patients each. The glans penis had papular lesions in 4 patients.

Nail changes were seen in 16% of patients characterized by longitudinal ridging (6%), longitudinal melanonychia, pterygium and onychodystrophy was seen in 2% each of patients (Table 1). The phenomenon of Koebner's was seen in 60% patients. The present study had coexisting dermatophytosis in 6 patients, atopic dermatitis and vitiligo in 2%. Anemia and diabetes mellitus was observed in 18% and 8% respectively.

Skin biopsies were performed in 100 patients and mucosal biopsy sampling was done in 6 cases of oral lichen planus which were subjected for histopathological examination. Hyperkeratosis (87%), saw toothed rete ridges (85%) and basal cell degeneration (93%) were the major epidermal changes observed histologically (Figure 5). Dermal changes mainly consisted of band like infiltration of lymphocytes (97%), melanin incontinence in 95% and melanophages in 51% (Figure 6). The histopathological examination of mucosal samples from oral cavity revealed, hyperkeratosis, focal hypergranulosis and acanthosis in 33%, parakeratosis and saw toothed ridges was observed in 66%. Basal cell degeneration, band like infiltration and melanin incontinence was observed in all the samples (Figure 7).

DISCUSSION

The incidence of LP in this hospital based study was 0.53%. The incidence of LP in various countries varies between 0.5% to 1% among patients with skin disorders⁸. The incidence of LP in two Indian studies was 0.76%⁹ and 0.8%¹⁰. In the present cohort 42% of patients belonged to age group between 31-40 years. Usually two third of cases occur between ages of 30 and 60 years⁸. Males comprised 54% and females 46%, with a male to female ratio of 1.7:1. The male preponderance was also observed in two Indian studies^{9,10}. On the contrary Garg V study showed female preponderance¹¹.

The duration of the disease in majority of patients was 3 months (40%). The shortest duration was 10 days and the longest being 3 years. Kacchawa D in his study observed that the duration of LP varies between 5 days to 3 years¹⁰. Familial LP was observed in two sisters. Samman PD and Kacchawa D have reported a familial incidence of 1.5% and 2.13% respectively^{2,10}.

The most common morphological type of lesion observed in our study was flat topped papules (44%). The percentage of classical lichen planus in Indian studies is between 40% to 73.3%^{10,11,12}. Isolated hypertrophic lesions were observed in 12% of cases. Singh OP observed 12.7% of hypertrophic lesions in his study. The occurrence of hypertrophic LP reflects chronicity of the disease and also immune response of the affected individual. Hypertrophic lesions admixed with classical lichen planus was observed in 10% of cases. The incidence of actinic LP is highly variable and depends on climate and the nature of work, actinic LP was observed in 8% of cases who were engaged in outdoor work. We observed only 2 cases of annular LP in our study. Annular LP is quite a rare entity and was not observed in studies by Singh OP and Garg V^{9,11}.

The lesions were predominantly distributed on upper and lower limbs (36%), followed by trunk and limbs in 32% cases. Boyd and Neldner¹ observed that the flexural areas of wrist are the classical site with arms and legs being the most common sites of involvement. Involvement of scalp, nails, palms and soles was lower in comparison to other Indian studies. This probably reflects the tendency of LP to affect selectively certain areas of body.

Linear LP accounted for 4% of cases in our study. In general 0.24% to 0.62% of all patients with LP have linear configuration. Hamid A¹⁴ and Aziz MA¹⁵ found linear LP in less than 4% of their study cohort. Lichen planus pigmentosus and bullous lichen planus were observed in 2% each of study subjects. The incidence of various morphological types lichen planus varies in different studies and this can be attributed to

factors like genetics, HLA association, occupation and climate.

The involvement of skin and mucus membrane or in combination is highly variable¹⁶. The affection of both skin and mucus membranes was observed in 14% of cases in our study. The percentage of involvement of both skin and mucus membranes was 10.18% and 24.4% in two Indian studies^{10,12}. In many studies isolated involvement of mucus membranes alone in lichen planus is relatively low and we did not observe mucus membranes being exclusively affected in our study. The most common mucosal area affected was the oral cavity (10%), 4% patients had papules on glans penis. Andreason¹⁷ in his study observed a high percentage of oral mucosal involvement (cheeks in 99.13%, lips in 37.39%, gingival in 61.75%). The mucosal involvement was comparatively very low in our study probably pointing to role of genetics and geographical factors in causation of mucosal lichen planus.

LP of nails manifested in 16% of study subjects. Longitudinal ridging with thinning of nail plate was the most common feature observed (6%). The nails of fingers were affected more than toe nails. In two Indian studies nail involvement was observed in 6.4% and 9.33% cases^{10,11}.

Among the study subjects dermatophytosis was observed in 6%, atopic dermatitis in 4% and vitiligo in 2%. The associated systemic disorders of diabetes mellitus, hypertension and anemia was 8%, 6% and 18% respectively. Boyd and Neldner¹³ have suggested that LP can occur in association with a variety of unrelated disorders. It is difficult to ascertain whether association is causal or fortuitous. The association with other diseases could be due to shared autoimmune etiology in some patients or it could be coincidental.

The histopathological examination of skin biopsy revealed hyperkeratosis in 87% of biopsy specimens, other epidermal features observed were focal hypergranulosis (82%), acanthosis (89%), saw toothed rete ridges (85%) and basal cell degeneration in 93%. Colloid bodies were seen in 8% of specimens and Max Joseph spaces in 2% of samples studied.

The dermal features consisted of band like infiltration in 97% of samples, 82% had exclusive lymphocytic infiltration and a mixed infiltrate of macrophages and lymphocytes in 15%. Melanin incontinence was observed at a much higher percentage of 95% and melanophages in 51%. The histopathological diagnosis was concordant with clinical diagnosis in 96% of skin biopsy specimens, the rest of 4% was diagnosed histopathologically as non-specific dermatitis. This discordance could have resulted due to technical errors while processing the samples, further Garg V¹¹ found a clinico histopathological correlation only to the extent of 90.67% .

The mucosal biopsy of oral lesions was performed in 6 cases (10 patients had oral lesions). Hyperkeratosis, focal hypergranulosis and acanthosis was observed in 33% of samples. Parakeratosis and rete ridges in 66%. The dermis showed band like infiltration and melanin incontinence in all the specimens. Scully¹⁷ observed that features of oral lichen planus are very much similar to those of cutaneous lichen planus, but the appearance is not always classical. Hyperkeratosis is more common in oral than cutaneous lichen planus, but the rete pegs are rarely saw toothed in oral lichen planus.

The histopathological features of different morphological types of lichen planus was barely variable, this variation could be influenced by duration and prior treatment of the disorder before skin biopsy.

CONCLUSION

Lichen planus albeit easily diagnosed with its ubiquitous violaceous, flat topped papules can be easily confused with prurigonodularis, psoriasis and other violaceous lesions. A high level of clinico histopathological concordance can eliminate misdiagnosis and can lead to a better therapeutic outcome.

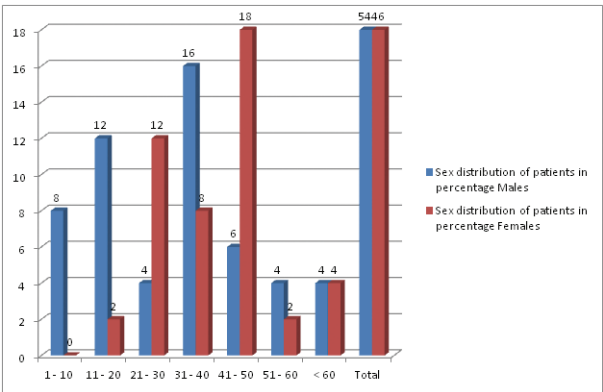


Figure1- Showing age and sex distribution of Lichen planus

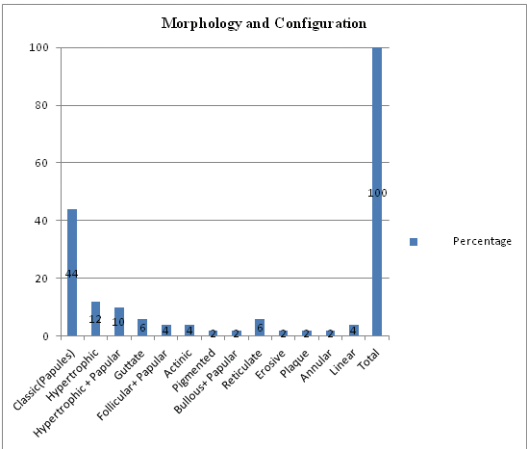


Figure2- Showing percentage of morphological types and configuration in LP



Figure 3: Showing classical lesions present on flexor aspect of forearm with koebnerisation

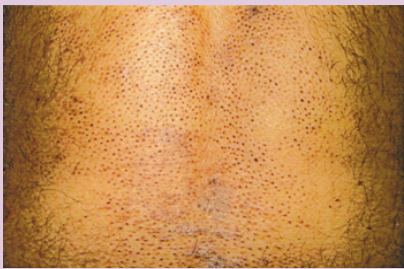


Figure 4: Photograph showing follicular Lichen planus with horny papules and violaceous plaques

Table 1: Showing nail changes in finger and toe nails

Characteristics	Percentage	
	Finger nails	Toe nails
Thinning+ Longitudinal ridging	6	0
Pterygium	2	0
Subungal hyperkeratosis	0	4
Longitudinal melanonychia	2	0
Onychodystrophy	0	2
Total	10	6

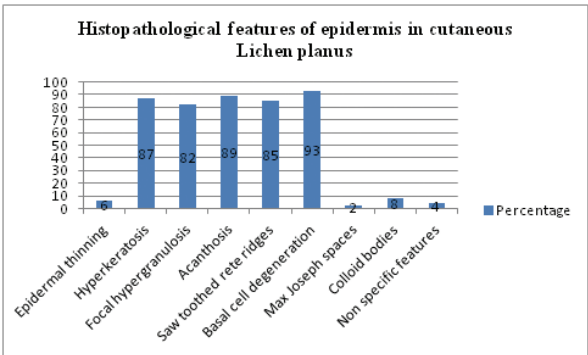


Figure 5: Showing histopathological features of epidermis in cutaneous LP

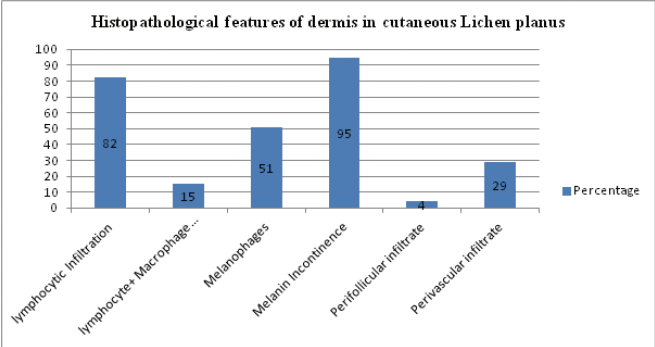


Figure6- showing histopathological features of dermis in cutaneous LP

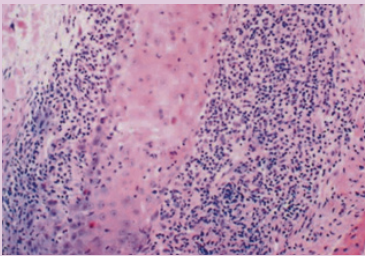


Figure 7: Photomicrograph of follicular lichen planus showing dense perifollicular lymphocytic infiltrate (H&E, 40X)

REFERENCES

1. Oztap P, Onder M, Ylter N, Oztap MO. Childhood lichen planus with nail involvement: A case. The Turkish Journal of Pediatrics. 2003;45:43-5.
2. Samman PD, Lichen planus- A dermatological century(comment).Br J Dermatol 1969;81:306-7.
3. Shiohara T, Kano Y. Lichen Planus and lichenoid dermatoses. In: Bologna JL, Jorizzo J, Rapini RP, editors. Dermatology. New York, NY, USA: Mosby Elsevier; 2008. pp. 159–180.
4. Eisen D. The clinical features, malignant potential, and systemic associations of oral Lichen Planus: a study of 723 patients. Journal of the American Academy of Dermatology. 2002;46(2):207–214.
5. Rao R, Saccchidanand S. Lichen Planus and Lichenoid Reactions. 4thed. In: Sacchidanand S, Oberai C, Inamadar AC, editors, Iadvl Text Book of Dermatology. Mumbai:Bhalani Publishing House. 2014. pp. 1090-116.
6. Rana S, Gupta R, Singh S, Mohanty S, Gupta K, Kudesia M. Localization of T-cell subsets in cutaneous lichen planus: An insight into pathogenetic mechanism. Indian J DermatolVenereolLeprol 2010;76:707-9.

7. Ingafo M, Leao JC, Porter SR, Scully C. Oral lichen planus: a retrospective study of 690 British patients. *Oral Dis.* 2006 Sep. 12(5):463-8.
8. Daoud MS, Pittlekow MR. Lichen planus. 6th ed. In : Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, editors, *Fitzpatrick's Dermatology in general medicine*. New York : McGraw-Hill; 2003.pp. 463-77.
9. Singh OP, Kanwar AJ. Lichen planus in India. An appraisal of 441 cases. *Int J Dermatol* 1976;15: 752-6.
10. Kacchawa D, Kacchawa V, Kalla G, Gupta LP. A clinico-aetiological profile of 375 cases of Lichen planus. *Ind J Dermatol* 1995; 61: 276-9.
11. Garg V, Nangia A, Logani K, Sharma RC. Lichen planus a clinico histopathological study. *Ind J Dermatol* 2006;66:193-5.
12. Sehgal VN, Rege VL. Lichen planus: an appraisal of 147 cases. *Ind J Dermatol Venereol* 1974;40:104.
13. Boyd AS, Neldner KH. Lichen planus. *J Am Acad Dermatol* 1991;25: 593-619.
14. Hamid A, Aziz MA. Mucopolysaccharide changes in lichen planus. *Ind J Dermatol* 1970; 42: 150-3.
15. Hamid A, Aziz MA. Lichen planus: Histopathological study of 57 cases. *Ind J Dermatol Venereol* 1970;36:85-91.
16. Fivenson DP, Mathes B. Treatment of generalized Lichen planus with Alefacept. *Arch Dermatol* 2006;142:151-3.
17. Scully C, Kom EL. Lichen planus: Review and update on pathogenesis. *Journal of Oral Pathology* 1985;14:431-58.

Please cite this article as: Raghavendra, Jaffer Basha. Clinico - Histopathological features of Lichen Planus-an Appraisal. *Perspectives in medical research* 2016;4:2:34-38.

Sources of Support: Nil, Conflict of interest: None declared