Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies ***NLM ID:*** *101716117*

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr Index Copernicus value = 85.10

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Original Research

Clinico-pathological spectrum of lichen planus in 82 cases

Dr. Ishani Gupta[1], Dr. Reeta Sood[2], Dr. Subhash Bhardwaj[3]

[1]Senior resident, Department of Pathology, Government Medical College Jammu, J & K, India;
[2]Professor, Department of Dermatology, Acharya Shri Chander College of Medical Sciences and Hospital, Jammu, J & K, India;
[3]Professor, Department of Pathology, Government Medical College Jammu, J & K, India

ABSTRACT:
Background: The present study was conducted to assess histopathology of lichen planus. **Materials & Methods:** 82 patients of lichen planus was recorded. Biopsy of skin lesions was done and sent for histopathological examination. Specimens were studied with H & E stains. **Results:** Common type was classical in 35, hypertrophic in 22, linear in 11, follicular in 8, annular in 5 and LP Pigmentosus in 1 patient. The difference was significant (P< 0.05). Common histological findings were hyperkeratosis in 80, basal cell degeneration in 82, focal hypergranulosis in 76, band like inflammatory infiltrate in 56, pigment incontinence in 82, saw-tooth rete ridges in 42 and colloid bodies in 30 patients. The difference was non- significant (P> 0.05). **Conclusion:** Clinico pathological correlation is the key to confirm the diagnosis for further patient care and treatment. Most common clinical form found was classical type. The most consistent histopathological findings were basement membrane degeneration, band like lymphocytic infiltrates and melanin incontinence.
Key words: hyperkeratosis, histopathological, Lichen Planus

Received: October 20, 2020 Accepted: November 27, 2020

Corresponding Author: Dr. Ishani Gupta, Senior resident, Department of Pathology, Government Medical College Jammu, J & K, India

This article may be cited as: Gupta I, Sood R, Bhardwaj S. Clinico-pathological spectrum of lichen planus in 82 cases. J Adv Med Dent Scie Res 2020;8(12):153-156.

INTRODUCTION

Lichen Planus is one of the most itchy dermatoses, worldwide in distribution, without racial, climatic or sex predilection. It is relatively common disease of the skin, also affecting the mucous membranes, nails & hair. Lichen planus produces intolerable itching which may interfere with sleep. The cosmetically unacceptable hyperpigmentation and the hypertrophic lesions produced in the course of the disease, make it a troublesome one.[1]

Various clinical classifications of oral lichen planus have been proposed.[2] These classifications include that suggested by Silverman, who distinguishes three types: reticular, atrophic and erosive. The reticular form usually appears symmetrically on the buccal mucosa and presents few symptoms. It is common to observe the atrophic form at the buccal, lingual and/or gingival level, with the latter appearing in the form of desquamative gingivitis.[3] The erosive form primarily manifests in the buccal mucosa and lingual dorsum, and it along with the atrophic form, presents the most symptoms.[4]

It is evident that immunological mechanisms almost certainly mediate the development of lichen planus. No consistent alterations in immunoglobulins have been shown in lichen planus and humoral immunity is most likely a secondary response in immunopathogenesis. Cell mediated immunity plays a major role in triggering clinical expression of the disease. Both CD4+ & CD8+ T cells are found in lesional skin in dermis while CD8 + T cells infiltrate epidermis.[5] The present study was conducted to assess clinico-pathological spectrum of lichen planus.

MATERIALS & METHODS

The present study was conducted in the department of Pathology. It comprised of 82 patients of lichen planus of both genders. All patients were informed regarding the study.

Data such as name, age, gender etc. was recorded. Symptoms, their duration, history of taking any drugs and nature of occupation of the patients were recorded. Detailed personal history regarding other skin diseases, personal habits was recorded. Biopsy of skin lesions was done and sent for histopathological examination. Specimens were studied with H & E stains. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 82		
Gender	**Males**	**Females**
Number	32	50

Table I shows that out of 82 patients, males were 32 and females were 50.

Table II Clinical types

Types	Number	P value
Classical	35	0.01
Hypertrophic	22	
Linear	11	
Follicular	8	
Annular	5	
LP Pigmentosus	1	

Table II, graph I shows that common type was classical in 35, hypertrophic in 22, linear in 11, follicular in 8, annular in 5 and LP Pigmentosus in 1 patient. The difference was significant (P< 0.05).

Graph I Clinical types

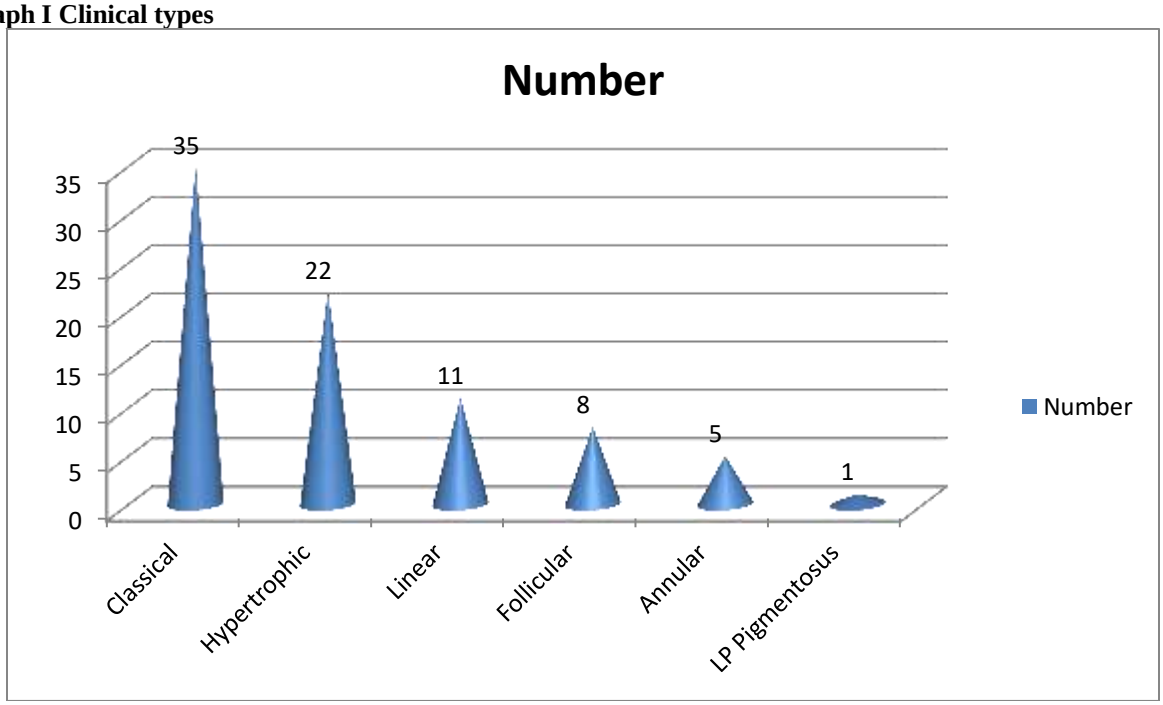
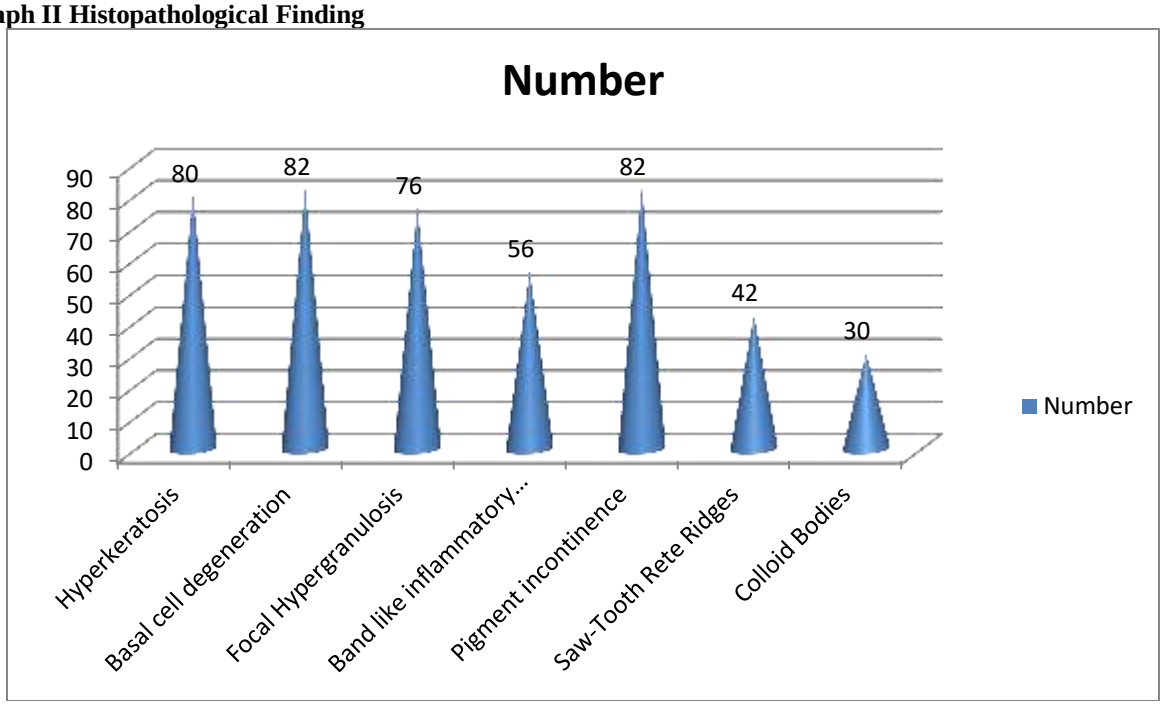


Table III Histopathological Finding

Finding	Number	P value
Hyperkeratosis	80	0.07
Basal cell degeneration	82	
Focal Hypergranulosis	76	
Band like inflammatory Infiltrate	56	
Pigment incontinence	82	
Saw-Tooth Rete Ridges	42	
Colloid Bodies	30	

Table III, graph II shows that common histological findings were hyperkeratosis in 80, basal cell degeneration in 82, focal hypergranulosis in 76, band like inflammatory infiltrate in 56, pigment incontinence in 82, saw-tooth rete ridges in 42 and colloid bodies in 30 patients. The difference was non- significant (P> 0.05).

Graph II Histopathological Finding



DISCUSSION

Lichen planus is characterized by shiny, violaceous, flat topped polygonal papules which retain the skin lines and which vary in size from pinpoint to a centimeter or more across, they may be closely aggregated or widely dispersed.[6] Fine whitish puncta or reticulate networks is considered to be highly characteristic, more easily observed after applying oil, xylene or water and visualising the lesions with a magnifying lens or a handheld dermatoscope. It is due to localized thickening of stratum granulosum although a focal increase in the activity of lichen planus may account for it. In the acute, evolving stage of the disease, scratching, injury or trauma may induce an isomorphic response. It is evident that immunological mechanisms almost certainly mediate the development of lichen planus.[7] No consistent alterations in immunoglobulins have been shown in lichen planus and humoral immunity is most likely a secondary response in immunopathogenesis. Cell mediated immunity plays a major role in triggering clinical expression of the disease. Both CD4+ & CD8+ T cells are found in lesional skin in dermis while CD8 + T cells infiltrate epidermis. Progression of disease leads to preferential accumulation of CD8 + T cells. The CD8+ cells & CD45 + RO cells are responsible for the development of apoptosis in LP.[8] The present study was conducted to assess histopathology of lichen planus.

In present study, out of 82 patients, males were 32 and females were 50. The common type was classical in 35, hypertrophic in 22, linear in 11, follicular in 8, annular in 5 and LP Pigmentosus in 1 patient. De Sousa et al[9] in their study found that seventy eight percent of the patients are female and 22% are male, with an average age of 56.06 years for both sexes. The most frequent

clinical form is reticular, present in 78% of the cases, and the most common location is the buccal mucosa, present in 70% of the patients. Hydropic degeneration of the basal layer and lymphocytic infiltration in the subepithelial layer are observed in the entire sample. Signs of atypia were identified in 4% of the cases, but without dysplasic features. Other common histological findings were the presence of necrotic keratinocytes (92%), hyperplasia (54%), hyperkeratosis (66%), acanthosis (48%), and less frequently, serrated ridges (30%) and the presence plasma cells (26%).

The diagnosis of oral lichen planus is first obtained based on the clinical appearance of the lesions, and is subsequently confirmed by a biopsy and a histopathological study. The majority of authors agree that a biopsy is necessary, given that it allows us to confirm the clinical diagnosis and make the differential diagnosis with other lesions.[10] There are a number of lesions that are similar to oral lichen planus, both from a clinical as well as a histological standpoint. They are called "lichenoid reactions", which have a known cause and include lichenoid contact lesions, drug-induced lichenoid reactions and lichenoid reactions in graft versus host reaction. The difference between a lichen planus and a lichenoid reaction is determined by a series of clinical and histological criteria of the lichen planus itself, which the lichenoid reaction does not meet in its entirely.[11]

LP can affect any part of the body, but the extremities are involved usually symmetrically. The volar aspect of wrist, flexural areas of arms, legs, lumbar region, around the ankles is often involved. Lower limbs have been found to be most commonly involved in many Indian studies. The ankles and shins are the commonest sites for hypertrophic lesions. Face is usually spared and palmoplantar involvement is unusual. Inverse lichen planus affects axillae, groin and inflammatory areas. All the mucosal sites may be affected.[12]

Maheshwari GR et al, aimed to get clinico pathological correlation in lichenoid interface dermatitis which will help in accurate diagnosis by analyzing history, clinical examination as well as histological details of nature and extent of epidermal, interface and dermal changes and the distribution of various inflammatory cell infiltrates. They observed that out of total 117 cases, 108 were of lichen planus, five were of lichen striatus, two of lichenoid drug eruptions and two of lichen nitidus. Clinico-pathological correlation was present in 70.94% of cases of lichenoid interface dermatitis. Correlation was seen in 100% cases of lichen striatus, and 78% cases of lichen planus.[13]

CONCLUSION

Clinico pathological correlation is the key for a conclusive diagnosis for further patient care and treatment. Most common clinical form found was classical type. The most consistent histopathological findings were basement membrane degeneration, band like lymphocytic infiltrates and melanin incontinence.

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.Int J Dermatol. 1976 Dec;15:752-6.
4. Kachhawa D, Kachhawa V, Kalla G, Gupta L. A clinico-aetiological profile of 375 cases of lichen planus. Indian J Dermatol Venereol Leprol. 1995;61:276-9..
5. Van der Meij EH, van der Waal I. Lack of clinicopathologic correlation in the diagnosis of oral lichen planus based on the presently available diagnostic criteria and suggestions for modifications. J Oral Pathol Med. 2003; 32:507-12.
6. S. Desai, A. Badwe, B. Nikam, S. Shetty. Histopathological study of interface dermatitis with its clinical correlation Int. J. Health. Biomed. Res., 2 (3) (2014), pp. 24-32.
7. Pakfetrat A, Javadzadeh-Bolouri A, Javadzadeh A, Shabestari S, Falaki F. Oral lichen planus: a retrospective study of 420 Iranian patients. Med Oral Patol Oral Cir Bucal. 2009; 20.
8. Scully C, de Almeida OP, Welbury R. Oral lichen planus in childhood. Br J Dermatol. 1994; 130:131-3.
9. De Sousa FA, Rosa LE. Oral lichen planus: clinical and histopathological considerations. Brazilian journal of otorhinolaryngology. 2008 Mar 1;74(2):284-92.
10. Bagán-Sebastián JV, Aguirre-Urizar JM, Milián-Masanet A, Peñarrocha-Diago M, García-Pola-Vallejo MJ. A morphometric study of 74 cases of oral lichen planus. Rev Stomatol Chir Maxillofac. 1991; 92:265-8.
11. Batra P, Wang N, Kamino H, Possick P. Linear lichen planus. Dermatol Online J. 2008; 15;14:16.
12. Hall R, Wartman D, Jellinek N, Robinson-Bostom L, Telang G. Lichen planus of the nail matrix with predominant plasma cell infiltrate. J Cutan Pathol. 2008; 35 1:14-6.
13. Maheshwari GR, Mehta HH, Nikam V. Clinico-histopathological correlation for diagnosis of lichenoid interface dermatoses. Journal of Dermatology & Dermatologic Surgery 2016;20:115-124