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Pathology

HISTOPATHOLOGICAL SPECTRUM OF LICHEN PLANUS AND ITS VARIANTS

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ABSTRACT

Background : The Histomorphological spectrum of clinical disease related to Lichen planus and its variants are wider. The most common hyperpigmented skin lesion is lichen planus and its variants. Histopathological examination helps us to derive a concept of the particular tissue reaction and it may cut across boundaries of clinically well established classes of disease.

Aims and Objectives: To know the various histomorphological patterns of lichen planus and its variants.

To assess the accuracy of histopathology in diagnosing various lichenoid skin lesions.

Materials and methods : A prospective study is conducted by histopathological examination of skin biopsies in a tertiary care hospital. Skin biopsies of 60 patients with suspected lichenoid skin lesions in all age groups from dermatology department were subjected for histopathological examination and confirmed lichenoid lesions were considered for the study and special stains were used wherever necessary.

Results : Lichen planus and its variants is the commonest lesion encountered in daily reporting of skin biopsies with features displaying hyperkeratosis, hypergranulosis, acanthosis, hydropic degeneration of the basal cells, melanin

incontinence secondary to basal cell damage and band-like inflammatory

infiltrate hugging the dermo-epidermal junction and perivascular inflammatory infiltrate.

Conclusion: The approach was to study the various histopathological types of lichen planus and its variants lesions, owing to complex features exhibited by skin lesions and histopathological examination is the mainstay for final diagnosis and categorization for treatment

KEYWORDS : Lichen planus, basal cell damage, interface dermatitis

INTRODUCTION

Little more than a century ago, the noted pathologist Rudolph Virchow considered skin as a mere protective covering for more delicate and functionally sophisticated internal viscera Over the past few decades, however, studies have demonstrated the skin to be a complex organ-largest in the body in which precisely regulated cellular and molecular interactions govern many crucial responses to our environment.

The spectrum of clinical disease related to Lichen planus and its variants are wider. The most common hyperpigmented skin lesion is lichen planus and its variants. Histopathological examination helps to derive a concept of the particular tissue reaction and it may cut across boundaries of clinically well established classes of disease.

The pathologist ability to render an accurate diagnosis depends on the available clinical information and categorise lichen planus and its variants.

AIMS AND OBJECTIVES:

To know the various histomorphological patterns of lichen planus and its variants.

To assess the accuracy of histopathology in diagnosing various lichenoid skin lesions

MATERIAL AND METHODS:

This is a prospective study of 60 skin biopsies received from Department of dermatology and histopathological examination done at Pathology Department in a tertiary care hospital . The specimens were fixed in 10% formalin, processed by paraffin embedding, stained with H & E and special stains wherever necessary and examined microscopically

RESULTS AND OBSERVATION

The total number of 60 skin biopsies with histopathologically proven lichen planus and its variants were considered for this study.

60 cases were studied of which 32 were classical lichen and remaining its variants as distributed in Table 1

TABLE 1: DISTRIBUTION OF THE CASES

Sl. No.	Lesions	No. of cases	Percentage
01	Classical lichen planus	32	53.5%
02	Lichen planus hypertrophicus	9	15%
03	Lichen planus pigmentosus	6	10%
04	Lichenoid like keratosis and reaction	7	11.5%
05	Lichen amyloidosis	2	3.5%
06	Lichen simplex chronicus	4	6.5%

Table-2: Comparison of histopathological findings in lichen planus and its variants

Histopathological features of CLP, HLP, LPP, LK, and LR	Present study 60cases	Ellis FA et.al (1967)[1] 100 Cases	Sehgal VN et.al (2011)[2] 375 Cases	Sontheimer RD (2009)[3] 100 Cases
Hyperkeratosis	100%	93%	83%	92%
Parakeratosis	6%	12%	16%	14%
Acanthosis	52%	23%	42%	37%
Hypergranulosis	84%	71%	74%	83%
Civatte bodies	5.20%	37%	23%	14%
Basal cell degeneration	100%	100%	100%	100%
Saw tooth rete ridges	19.50%	11%	18%	13%
Max Joseph spaces	5%	17%	6%	11%
Band like configuration hugging basal layer	98%	100%	91%	100%
Lymphocytic infiltration	100%	100%	100%	100%
Plasma cells	3.30%	3	6%	3.50%

CLP- classical lichen planus, HLP- hypertrophic lichen planus, LPP- lichen planus pigmentosus, LK-lichenoid keratosis and LR-Lichenoid reaction.

HISTOPATOLOGICAL FINDINGS

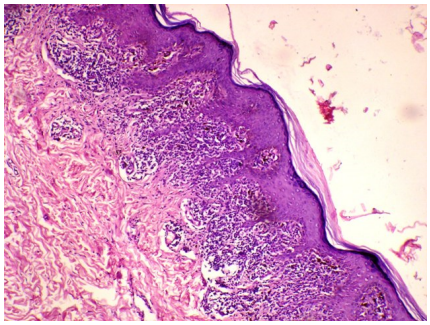

FIG 1: classical lichen planus – H&E stain **10X** showing hyperkeratosis, orthokeratosis, Irregular rete ridges, saw tooth, basal cell degeneration and band like inflammatory infiltrate

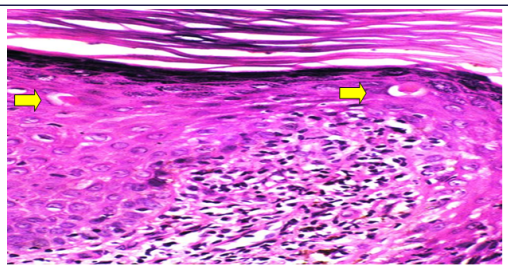

FIG 2: classical lichen planus - H&E stain **40X** showing hyperkeratosis, orthokeratosis, wedge shaped hypergranulosis and arrows showing epidermal civatte bodies

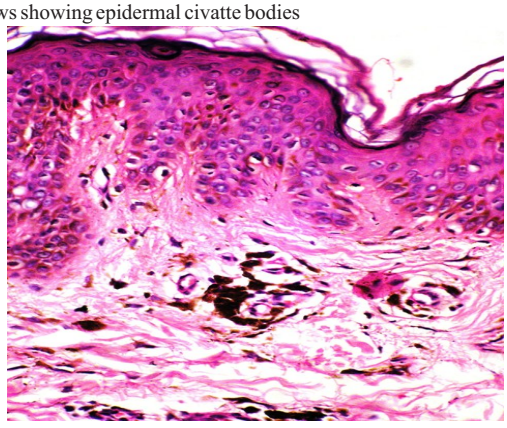

FIG 3: Lichen planus pigmentosus - H&E stain 10 X - Hyperkeratosis, hypergranulosis, irregular acanthosis and dermal pigment incontinence

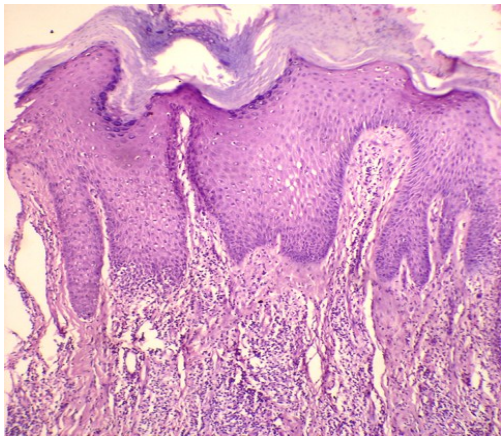

FIG 4: Hypertrophic lichen planus H&E stain 10X showing hyperkeratosis, marked acanthosis, hypergranulosis, papillomatosis, vacuolar basal cell degeneration and interface dermatitis.

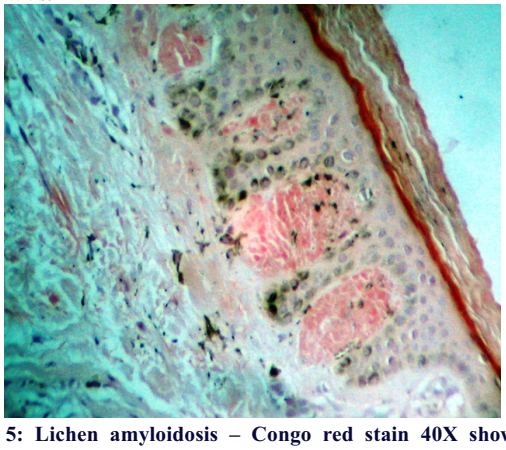

FIG 5: Lichen amyloidosis – Congo red stain 40X showing irregular acanthosis, hyperkeratosis and with the papillary dermis showing eosinophilic amorphous material of amyloid. amyloid in the papillary dermis shows a prominent "salmon pink colour

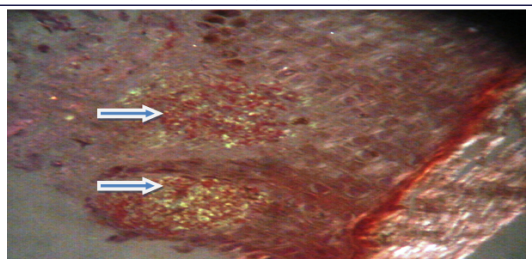

FIG 6: Congo red stain under polarised light 40X shows arrows showing "Apple green birefringence" in papillary dermis which is diagnostic of amyloidosis

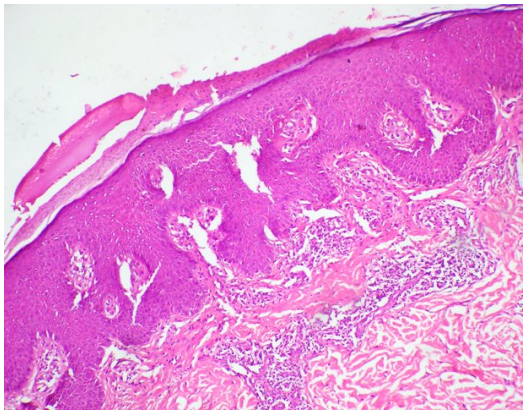

FIG 7: Lichenoid like keratosis - H&E stain 10X showing hyperkeratosis, parakeratosis, hypergranulosis, irregular rete ridges, basal cell degeneration and band like inflammatory infiltrate

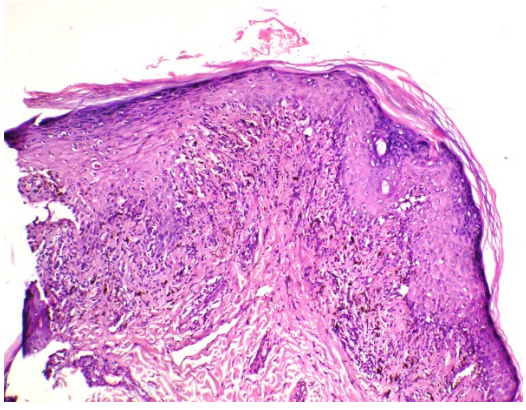

FIG 8: Lichenoid Drug reaction: H&E stain 10X showing hyperkeratosis, parakeratosis, orthokeratosis, and heavy inflammatory infiltrate with prominent interstitial pattern, perivascular inflammation around mid and deep dermal vessels

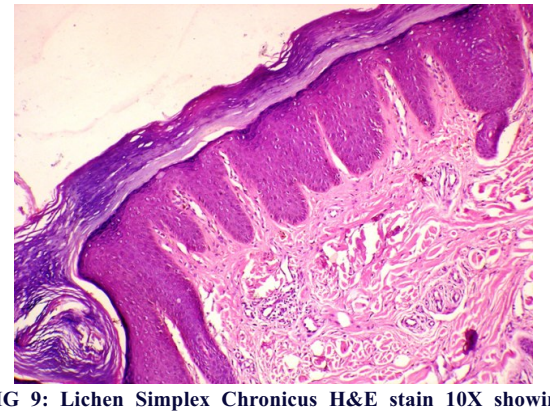

FIG 9: Lichen Simplex Chronicus H&E stain 10X showing hyperkeratosis, parakeratosis, acanthosis, irregular elongation of rete ridges, wedge shaped hypergranulosis, broadening of dermal papillae, superficial perivascular infiltrate

DISCUSSION

Lichen planus : Erasmus Wilson described the word lichen planus in 1869.

Kachhawa D reported the different histopathological features of lichen planus.

the lesin is more common in males and middle age group and the site of involvement is more common in the extremities, upper trunk, back, face oral mucosa and genitalia.

The studies conducted by Costa GD et al on different lichenoid reactions concluded that lichen planus was the most common lesion followed by lichen planus hypertrophicus lichen planus pigmentosus and all other variants.

His study showed that Lichen planus was most frequently seen in middle age groups and showed female preponderance.

Lichen planus lesions are characterized by hyperkeratosis, and irregular acanthosis, epidermis and focal hypergranulosis, basal cell degeneration, pigmentary incontinence, effacement of rete ridge, eosinophilic Civatte bodies and interface dermatitis.

According to Hermann Pinkus, 'Max Joseph' was among the first to stress the subepidermal cleft and referred it as Max Joseph spaces. This is due to more fluid accumulation and there is frayed appearance of lower rete and poor definition of dermo-epidermal junction.

Thyresson and Moberger described colloid bodies or hyaline bodies as eosinophilic degeneration in cells in epidermis or abnormal keratinization. Colloid bodies are PAS +ve but diastase resistant first described by Ellis in 1967.The thickening of the granular layer is uneven and wedge shaped. The granular cells appear increased in size and contain coarse and abundant keratohyaline granules. Acanthosis is variable with irregular elongation of rete ridges which may appear sharply pointed or saw toothed.

Lichen planus in children was studied by Kanwar AJ et al in 2010 and concluded that lichen planus is rare in children. It has been hypothesized that the rarity of associated autoimmune conditions, lack of exposure to drugs and dental restorative materials, infective agents and other environmental triggers that have been known to initiate lichen planus may be responsible for decreased incidence in children

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Francis A et al studied the histopathology of lichen planus based on analysis of 100 biopsy specimens and mentioned that lichen planus has characteristic clinical and histopathological features.

TABLE 3: Histopathological features of lichen planus

EPIDERMIS		DERMIS	
Parakeratosis	12	Band like infiltrate hugging the basal layer	100
Crusting	1	Mostly small mononuclear cells(lymphocytes)	100
Beading of granular layer	93	Sparse or few epithelioid cells	82
Acanthosis with linear lower margin	9	Many epithelioid cells	18
Acanthosis of ridges only	23	Fibrocytes	5
Atrophy with and without ridges	47	Plasma cells	3
Eosinophilic bodies	37	Giant cells	1
Liquefaction degeneration at the basal layer	100	Polymorphonuclears	1
Max Joseph Spaces	17		
Follicular lesions	6		
Lichen nitidus like lesions	8		

Hypertrophic lichen planus

Dhar S reported about "Fountain sign" in lichen planus hypertrophicus that is when corticosteroid injected into the plaques comes out through the follicular openings in a jet mimicking a "fountain". This may be due to degeneration of hair follicular wall; the drug passes through pilosebaceous canal easily and is ejected forcefully through follicular openings.

Dogra D et al in 1997 and Sengupta S et al 2006 did a similar study about malignant transformation of hypertrophic lichen planus into squamous cell carcinoma and also mention that hypertrophic lichen planus is a variant of lichen planus which presents as thickened, hyperkeratotic, erythematous to violaceous intensely pruritic papules and plaques.

Although the incidence of cancer in oral lichen planus is about 1.3% neoplastic transformation in skin is very rare.

Kumari R and her colleagues in 2012 studied hypertrophic lichen planus in HIV infection and showed that lesions are widely distributed, hyperpigmented, pruritic scaly lesions involving face, trunk, upper and lower extremities. Most striking feature was raised extensive hypertrophic hyperpigmented plaques. Histopathological examination of hypertrophic lichen planus usually reveals compact hyperkeratosis, and marked acanthosis of the epidermis with hypergranulosis, vacuolar degeneration of basal keratinocytes and a band like lymphocytic infiltrate at dermo epidermal junction. The papillary dermis contained abundant melanophages and numerous civatte bodies.

Lichen Planus Pigmentosus

Lichen planus pigmentosus is one of the common causes of facial melanoses as described by Khanna N et al in his journal about facial melanoses Indian perspective, mentions that lichen planus pigmentosus is characterized by generally asymptomatic, diffused, mottled, reticulated or perifollicular hyperpigmented dark brown to slate grey to black macules present mostly over exposed areas and flexures.

Histopathology of lichen planus pigmentosus was described in Iranian J in 2010 by Wani A et al as skin biopsy showing atrophic epidermis, basal hydropic degeneration with sparse perivascular lymphohistiocytic infiltrates, with incontinence of pigment and the presence of numerous melanophages.

Lichenoid reaction

Lichenoid skin reaction or Interface dermatitis was recognized, classified and etiology with clinicopathological study was done by Sehgal VN and his colleagues in 2011 and they considered it as a prototype of lichen planus and its variants.

It presents as a shiny, flat topped, polygonal or of different sizes and occur in clusters resembling lichen growing on rock. Histologic examination is characterized by infiltrate of inflammatory cells that fills the papillary dermis in a band like fashion and often obscures the dermo-epidermal junction.

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

Lichenoid keratosis presents as violaceous with thin scaly rusty lesions. The pathognomonic features are lichenoid reaction which is prominent and florid pigment incontinence and colloid bodies. The inflammatory infiltrate is dense composed of lymphocytes, macrophages, few plasma cells and eosinophils are seen.

Lichenoid drug eruptions

Often encountered drug reaction to hetreogenous group if ingested drugs. Histopathologically differentiating features of lichenoid eruptions include focal parakeratosis, mild basal vacuolar changes with a few eosinophils and plasma cells. The degree of melanin incontinence is higher and dermal infiltrate is less dense and less band like than that of lichen planus.

Similar study was done by Sontheimer RD in 2009 and Lakshmi C et al in 2008 about interface dermatitis and perforating lichenoid reaction

and described the same clinical and histological features.
Tan CY et al studied about 212 cases of lichenoid keratosis in 1982 and described that lichenoid keratosis has been applied to solar keratosis that possess some histologic features of lichen planus- particularly basal cell degeneration and a band like dense mononuclear inflammatory infiltrate in the subepidermal zone.

Lichen Amyloidosis
The term "amyloid" was coined in 1854 by Virchow who was convinced by its resemblance to starch or cellulose. Rokitansky gave the first description of amyloidosis in 1842.

Shenoi SD et al in his study about 30 cases of lichen amyloidosis describes that lichen amyloidosis is a form of primary cutaneous amyloidosis. Histopatholgy shows epidermal findings included hyperkeratosis(100%), acanthosis(90%), papillomatosis (30%), hypergranulosis (16.7%) and elongation of rete ridges(5%), amyloid deposits as uniform pink globules occupying dermal papilla in(95%).

Amyloid deposits were visualized using Congo red in all 100% patients.

According to Kobayashi H et al study of the amyloidosis clinical and histopathological features are similar to the study of Shenoi SD except that Congo red stained amyloid shows green birefringence under polarized light.

Bullous lichen planus
Joshi A in 1999 reported that Bullous lichen planus as multiple, scattered, itchy, erythematosus and violaceous papules on the trunk, knees and extremites.
Biopsy shows hypergranulosis, band like infiltrate of lymphocytes in close proximity to the basal layer of epidermis with degeneration of basal cells and subepidermal bulla.
Direct immunofluorescence was negative.

Lichen Simplex Chronicus
According to Lotti T et al lichen simplex chronicus is a skin disorder characterized by central lichenificated plaque thickened and often hyperpigmented skin as a result of primary excessive scratching.

Most common sites are the neck, ankles, scalp, vulva, pubis, scrotum and extensor forearms. The peak incidence is between 35-50 years of age and women are more affected than men.

The lichen simplex chronicus presents histopathologically with epidermal hyperplasia, orthokeratosis and hypergranulosis with regular rete ridges. Perivascular infiltrate of lymphocytes and macrophages occasionally noted.

CONCLUSION
Lichenoid lesions represent clinical and morphological diversity, which is a difficult task for histopathological interpretation essentially based on histopathological aspect, which is important for management.

The pathologist ability to render an accurate diagnosis depends on the available clinical information and categorise lichen planus and its variants.

REFERENCES

1. Kachhawa D, Kachhawa V, Kalla G, Gupta L. A clinico-aetilogical profile of 375 cases of lichen planus. Indian Journal of Dermatol Venereol and Leprol 1995; 61(5): 276-279.
2. Costa GD, Bharambe BM. Spectrum of non-infectious erythematous, papular and squamous lesions of the skin. Indian j Dermatol 2010; 55(3): 225-228.
3. Mehta V, Vasanth V, Balachandran C. Palmar involvement in lichen planus. Dermatology online Journal. 2009 June; 15(6): 12.
4. Ellis FA. Histopathology of lichen planus based on analysis of one hundred biopsy specimens. The Journal of Investigative Dermatology 1967; 48(2): 143-148.
5. Kanwar JA, De D. Lichen planus in children. Indian Journal of Dermatol Venereol and Leprol 2010; 76(4): 366-372.
6. Dhar S "fountain sign" in lichen planus Hypertrophicus. Indian Journal of Dermatol Venereol and Leprol 1997; 63(3): 210.
7. Dogra D, Sharma N, Khanna N. Squamous cell carcinoma arising in Lichen planus Hypertrophicus. Ind J Dermatol 1997; 42(1): 30-31.
8. Senguptha S, Das JK, Gangopadhyay A. Malignant transformation of hypertrophic lichen planus. Indian Journal of Dermatol venereol Leprol 2006; 72(6) : 470.
9. Kumari R, Singh N, Thappa DM. Hypertrophic lichen planus as a presenting feature of HIV infection. Ind J Dermatol 2009; 54:8-10.
10. Wani AA, Jan NJ. Lichen planus pigmentosus with a linear pattern. Iranian Journal of Dermatology 2010; 12(4): 134-135.
11. Sehgal VN, Srivastava G, Sharma S, Sehgal S, Verma P. Lichenoid tissue reaction/interface dermatitis: Recognition, classification, etiology and clinicopathological overtones. Indian Journal of Dermatol venereol Leprol 2011; 77(4) : 418-430.
12. Sontheimer RD. Lichenoid tissue reaction/interface dermatitis: clinical and histological perspectives. Journal of Investigative Dermatology 2009; 129: 1088-1089.
13. Lakshmi C, Srinivas CR, Ramachandran B, Pillai SB, Nirmala V. Perforating lichenoid reaction to amlodipine. Indian J Dermatol 2008; 53(2): 98-99.
14. Tan CY, Marks R. lichenoid solar keratosis – prevalence and immunologic findings. The Journal of Investigative Dermatology 1982; 79: 365-367.
15. Das J, Gogoi RK. Treatment of primary cutaneous amyloidosis with cyclophosphamide. Indian Journal of Dermatol venereol Leprol 2003; 69(2):163-164.
16. Kiboyashi H, Hashimoto K. Amyloidogenesis in organ- limited cutaneous amyloidosis: An antigenic identity between epidermal keratin and skin amyloid. The Journal of Investigative Dermatology 1983; 80: 66-72.
17. Joshi A, Khaitan KB, Verma KK, Sing. Generalised and Bullous lichen planus treated successfully with oral minipulse therapy. Indian Journal of Dermatol venereol Leprol 1999; 65: 303-304.
18. Lotti T, Buggiani G, Prignano F. Prurigo Nodularis and Lichen simplex chronicus. Dermatology Therapy 2008; 21: 42-46.