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Research Article

LICHENOID TISSUE REACTIONS- A STUDY OF VARIOUS HISTOMORPHOLOGICAL PATTERNS WITH CLINICAL CORRELATION AND REVIEW OF LITERATURE

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ABSTRACT

Lichenoid tissue reactions (LTRs) represent a diverse group of conditions similar to lichen planus clinically and histopathologically. Lichen planus is a common lesion that may involve the skin, mucus membranes, hair follicles and nails. A retrospective study of two years was conducted on skin biopsies with pathological diagnosis of lichen planus and other dermatological lesions resembling lichen planus clinically. The clinical findings and pathological parameters were noted for analysis and correlation. Histopathological parameters such as epidermal changes, changes at the dermoepidermal junction, dermal changes and dermal reaction pattern were studied. The lesions were divided into three distinct and well defined categories- Lichenoid dermatitis, Interface dermatitis and miscellaneous category. Lichen planus was the classical prototype of lichenoid dermatitis. Erythema multiforme was the classical prototype of interface dermatitis. The criteria used to differentiate and categorize the group of cutaneous lesions help in making a more definitive diagnosis and narrow the differential diagnosis amongst this vast group of chronic superficial dermatoses. Therefore an emphasis on the clinical correlation is crucial for the precise diagnosis of interface dermatitis and histopathological examination remains an indispensable tool that helps in rendering an accurate diagnosis.

INTRODUCTION

Lichenoid tissue reactions (LTRs) represent a diverse group of conditions similar to lichen planus clinically and histopathologically.[1] They can be subdivided into two categories- cell rich and cell poor, depending upon the intensity of interface inflammation. Cell rich LTRs include lichen planus with its variants and cell poor LTRs include conditions such as autoimmune connective tissue disorders and erythema multiforme.[2]

Lichen planus is a common lesion presenting as substance or chronic dermatoses that may involve the skin, mucus membranes, hair follicles and nails.[3] The nails are involved in about 10% of cases. Cutaneous lichen planus has worldwide distribution with its incidence varying from 0.22% to 1% depending upon geographical location.[4] Lichen planus represents approximately 0.38% of all dermatological outpatients in India.[4] It presents as pruritic, purple, polygonal, planar papules and plaques. It is usually self limited most commonly resolving spontaneously one to two years after onset.

Clinical subtypes of lichen planus include hypertrophic, atrophic, annular, vesicular, eruptive, linear, erosive/ulcerative, lichen planus actinicus, lichen planus pigmentosus, lichen planopilaris, lichen planus pemphigoides and overlap syndrome with lupus erythematosus.

Typical lesions of lichen planus histomorphologically show compact orthokeratosis, wedge shaped pattern at the dermoepidermal junction consisting of a dense band like infiltrate of lymphocytes. In addition, there is a basal cell layer vacuolar degeneration.[5,6]

A morphological pattern akin to that seen in lichen planus designated as lichenoid dermatitis can be seen in a variety of cutaneous lesions. This can make the diagnosis of lichen planus very challenging and in some cases difficult. Commonly encountered cutaneous lesions clinically resembling lichen planus are drug eruptions, and lichen planus like keratosis.

The dermatological lesions with lichenoid dermatitis consist of band like chronic inflammatory infiltrate in close proximation to dermoepidermal junction associated with damage at the

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dermoepidermal junction. Lichen planus is the classical prototype of lichenoid dermatitis.

Interface dermatitis is associated with prominent vacuolar change of the basal keratinocytes and apoptotic keratinocytes in the epidermis as seen in erythema multiforme and fixed drug eruption indicating a more severe immunological damage to the epidermis.

Classification based on inflammatory reaction pattern is helpful because there is correlation with the clinical presentation. The study was conducted to analyze and correlate the clinical presentation, anatomic distribution and histopathological features including the different inflammatory reaction patterns present in lichen planus, various subtypes of lichen planus and other cutaneous lesions resembling lichen planus.

MATERIALS AND METHODS

A retrospective study was conducted on skin biopsies received in the Department of Pathology of Hindu Rao Hospital, New Delhi, India with pathological diagnosis of lichen planus and other dermatological lesions resembling lichen planus clinically were included in the study. The study was for a period of two years from January 2016 and December 2017.

The skin biopsies with clinical diagnosis or differential diagnosis of lichen planus and dermatological lesions resembling lichen planus clinically or histopathologically were retrospectively reviewed. Skin biopsies from relevant sites stained with hematoxylin and eosin were reviewed.

Review of the skin biopsies included recording and analysis of the clinical presentation of the cutaneous lesions including gross description of the lesion, site of the biopsy and other sites involved including clinical history and the duration of the lesion, the clinical diagnosis and the histopathological diagnosis.

The clinical findings and pathological parameters were recorded in the case record form for analysis and correlation. Histopathological parameters studied included epidermal changes, changes at the dermoepidermal junction, dermal changes and dermal reaction pattern.

RESULTS

The present study of 50 cutaneous lesions revealed that majority of these lesions which consist of lichen planus, variants of lichen planus and other cutaneous lesions clinically resembling lichen planus involve the cutaneous reactive unit of skin which includes epidermis, papillary dermis and superficial capillary venous plexus at the junction of papillary and reticular dermis. These structures of the skin react together in majority of these cutaneous lesions.

In lichen planus, the histopathological changes are present at the dermoepidermal junction and also involve the overlying epidermis. In the present study the histological changes at the dermoepidermal junction were also found in some of the cutaneous lesions which clinically resemble lichen planus like lupus erythematosus and erythema multiforme. However, the histomorphological analysis revealed that the inflammatory reaction causing damage at the dermoepidermal junction in lichen planus and variants of lichen planus is different from that causing damage to dermoepidermal junction in lupus erythematosus and erythema multiforme which is of greater severity and causes reaction in the epidermis due to chronic irritation and hence affects the clinical presentation. In addition these cutaneous lesions have different inflammatory reaction patterns in the dermis.

On basis of this observation, the cutaneous lesions in the present study were divided into three distinct and well defined categories- Lichenoid dermatitis, Interface dermatitis and Miscellaneous category (table 1).

Table 1 Three main categories

S.no	Category of dermatitis	No. of cases	Percentage %
1.	Lichenoid dermatitis	26	52
2.	Interface dermatitis	14	28
3.	Miscellaneous	10	20

The inflammatory reaction patterns in these cutaneous lesions were analyzed and correlated with the clinical presentation. Lichen planus was the classical prototype of lichenoid dermatitis. Erythema multiforme was the classical prototype of interface dermatitis.

Clinically these cutaneous lesions come in the category of non infectious papulosquamous lesions and majority (25 of the 50 cases), 50% cases present as macules or papules and plaques (table 2).

Table 2 Clinical presentation of the cutaneous lesions

S.no	Clinical presentation	No. of cases	Percentage % (total 50 cases)
1.	Papules	15	30
2.	Papules and plaques	15	30
3.	Erythematous lesions	3	6
4.	Alopecia	9	18
5.	Scaly lesions	3	6
6.	Target lesions	1	2
7.	Macules	1	2
8.	Vesicles	1	2
9.	Pigmented lesions	1	2
10.	Nail involvement	1	2

Most common anatomical sites involved by these lesions were head and neck, upper and lower limbs and back (table 3). The age range of the patients was from 8 years to 50 years. 27 of the patients were males and 23 were females. The duration of the lesions varied between 7 days to 15 years.

Table 3 Anatomical sites involved in cutaneous lesions

S.no	Anatomical sites	No. of cases	Percentage % (total 50 cases)
1.	Head and neck	13	26
2.	Upper limbs	10	20
3.	Lower limbs	7	14
4.	Back and extremities	6	12
5.	Abdomen	4	8
6.	Back	2	4
7.	Thorax	1	2
8.	Buccal mucosa	1	2
9.	Nail	1	2
10.	Unspecified sites	5	10

Clinicopathological correlation revealed that all these cutaneous lesions were characterized histologically by superficial predominantly lymphocytic inflammatory infiltrate with variable effects on other structures of the superficial

integument consisting of epidermis, venules of the superficial capillary venous plexus and papillary dermis. In the present study, lichenoid dermatitis was found in 26 of the 50 cases and lichen planus was the classical prototype of this group of lesions (table 4, fig 1).

Table 4 Cutaneous lesions with lichenoid dermatitis

S. no	Lesions with lichenoid dermatitis	No. of cases	Percentage %
1.	Lichen planus and subtypes of lichen planus	24	92
2	Pityriasis lichenoides chronic	1	4
3.	Pityriasis lichenoides et varioliformis acuta	1	4

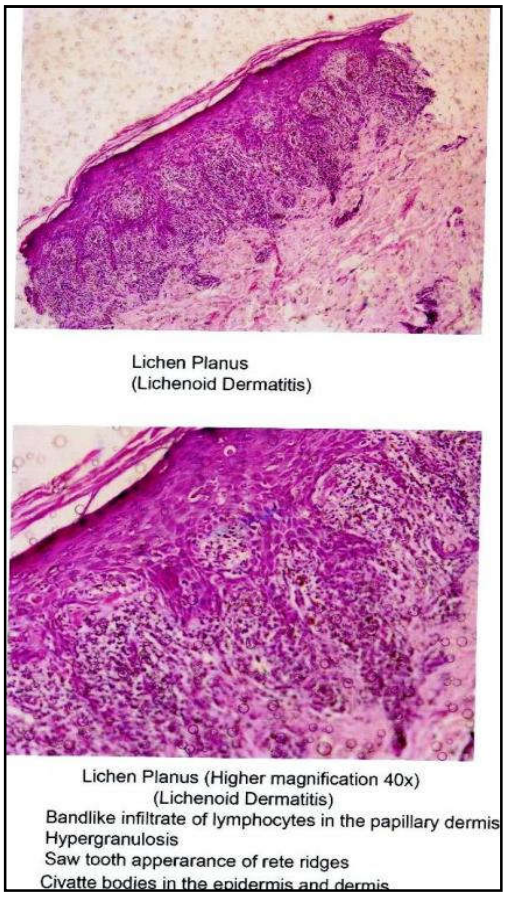

Fig 1 Lichen Planus

10 of the 50 cases were placed in the miscellaneous category (table 5) of superficial cutaneous dermatitis without any histological changes at the dermoepidermal junction. These lesions were clinicopathologically analyzed and were found to have different inflammatory reaction patterns which affected the other structures present in the superficial cutaneous reactive unit.

Table 5 Cutaneous lesions in miscellaneous category

S.no	Miscellaneous lesions	No. of cases	Percentage %
1.	Tuberculosis verruciformis acuta	1	10
2.	Lichen scrofulosorum	1	10
3.	Morphea	2	20
4.	Psoriasis	1	10
5.	Bullous pemphigoides	1	10
6.	Pigmented purpuric dermatoses	1	10
7	Lichen simplex chronicus	1	10
8.	Beckers naevus	1	10
9.	Naevus lipomatosus	1	10

Histological analysis revealed that this group of lesions was heterogeneous in pathogenesis and could be differentiated histologically on basis of inflammatory reaction pattern in the dermis and the composition of psoriasiform hyperplasia noted in psoriasis and granulomatous reaction pattern in dermis seen in lichen scrofulosorum (fig 5).

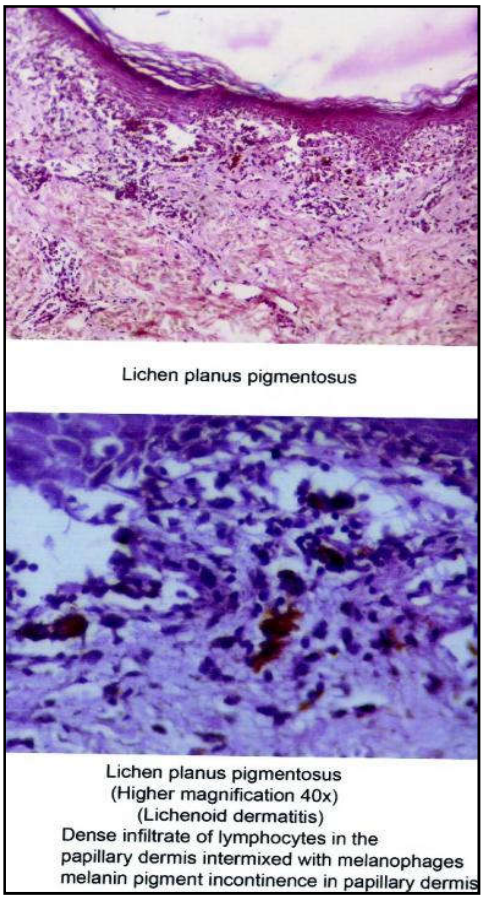

Fig 2 Lichen Planus Pigmentosus

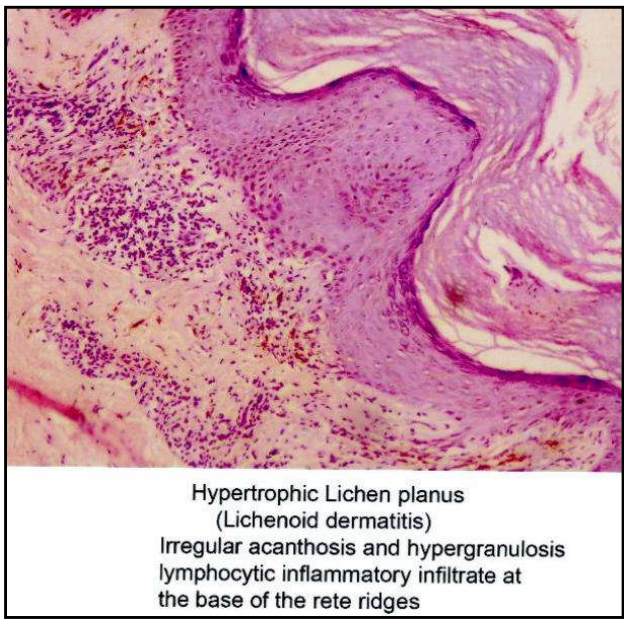

Fig 3 Hypertrophic Lichen Planus (H & E stain, 40x)

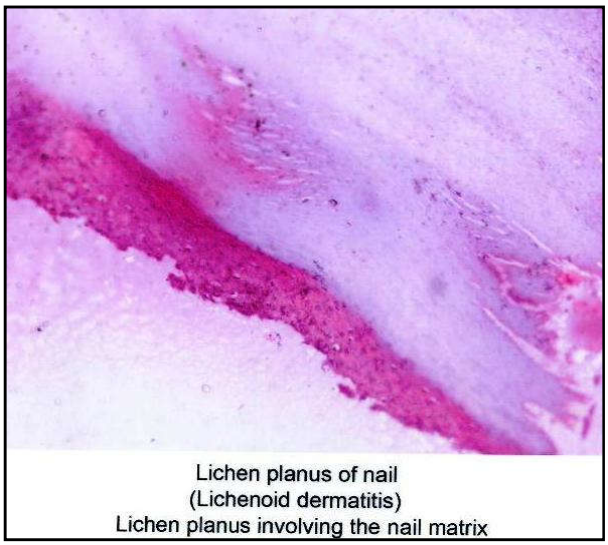

Fig 4 Lichen Planus of nail (H & E stain, 40x)

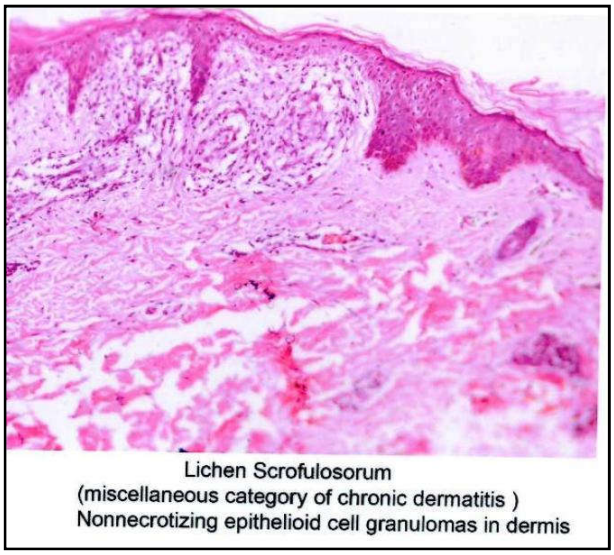

Fig 5 Lichen Scrofulosorum (H & E stain, 10x)

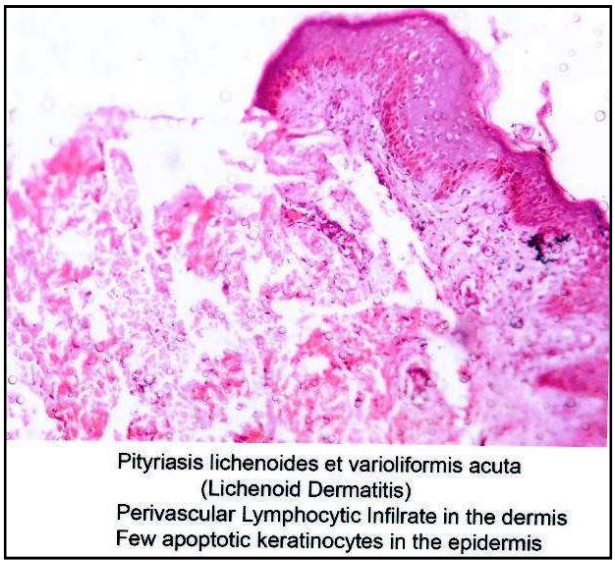

Fig 6 Pityriasis Lichenoids Et Varioliformis Acuta (H & E stain, 10x)

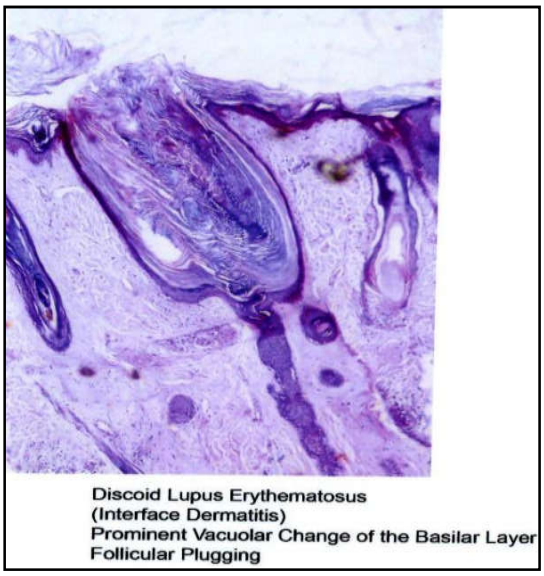

Fig 7 Discoid Lupus Erythematosus (H & E stain, 10x)

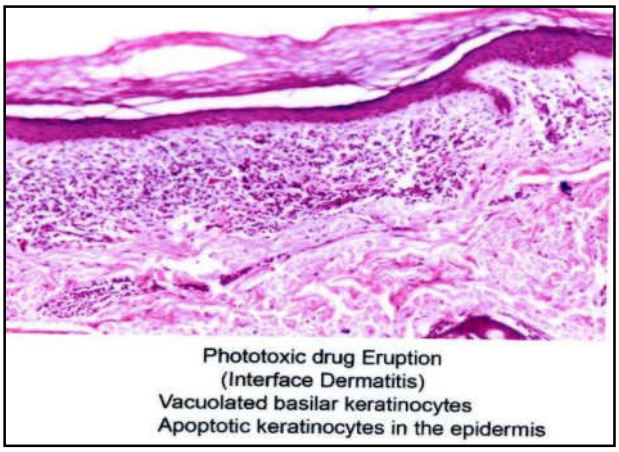

Fig 8 Phototoxic drug reaction (H & E stain, 10x)

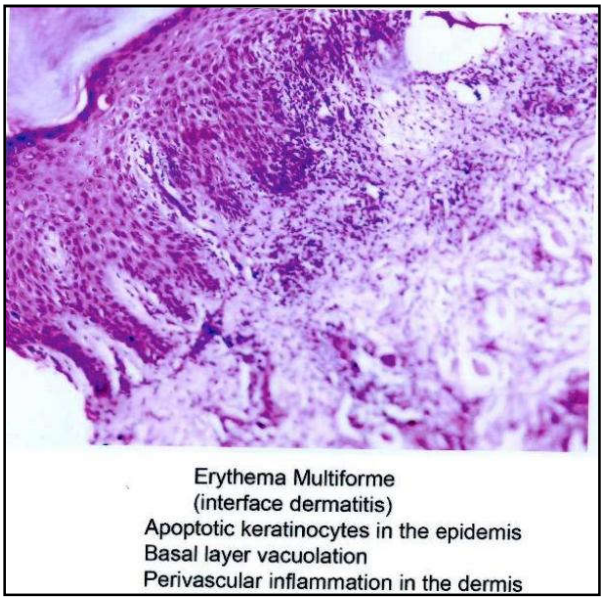

Fig 9 Erythema Multiforme (H & E stain, 40x)

In the present study there were 24 cases of lichen planus and subtypes of lichen planus (table 6) with characteristic lichenoid inflammatory infiltrate at the dermoepidermal junction. Amongst the various subtypes of lichen planus despite the presence of characteristic features that permit there segregation into distinct categories these variants show a similar clinical course and prognosis as classical lichen planus because of their clinical and histological similarity to classical lichen planus. Diagnosis of these variants can be challenging and familiarity with their clinical and histological manifestation is necessary.

Table 6 Cutaneous subtypes of lichen planus

S.no	Subtypes of lichen planus	No. of cases	Percentage %
1.	Classical lichen planus	13	54
2.	Lichen planus hypertrophicus	3	13
3.	Lichen planus pigmentosus	1	4
4.	Lichen planopilaris	6	25
5.	Lichen planus pemphigoides	1	4

Our study also highlights the cutaneous lesions that were commonly encountered in the differential diagnosis of lichen planus (table 7).

Table 7 Cutaneous lesions in differential diagnosis

S.no	Cases in differential diagnosis	No. of cases	Percentage % (total cases 50)
1.	Discoid lupus erythematosus	5	10
2.	Psoriasis	4	8
3.	Lichenoid drug eruption	1	2
4.	Eczema	1	2
5.	Pseudopalade of broq	1	2
6.	Lichen simplex chronicus	1	2
7.	Prurigo nodularis	1	2
8	Macular amyloidosis	1	2
9.	Verrucous epidermal naevus	1	2
10.	Beckers naevus	1	2
11.	Traumatic nail	1	2

A summary of the clinicopathological parameters used to differentiate between the three categories of cutaneous lesions has been enlisted (table 8).

It is possible to segregate the diseases and improve diagnostic accuracy based on the site of involvement in the skin, type of inflammatory reaction pattern and constitution of inflammatory infiltrate. This is helpful in better understanding of pathogenesis of the cutaneous lesions and in correlation of clinical findings with microscopic findings.

DISCUSSION

Lichen planus comes in the category of noninfectious papulosquamous lesions presenting as pruritic polygonal, flat topped, violaceous papules.

In the histological classification lichen planus is included in the general category of disorders of the superficial cutaneous reactive unit of skin which consists of epidermis, papillary dermis, and superficial capillary venous plexus at the junction of papillary and reticular dermis.[7]

Lichen planus is the classical prototype in the category of cutaneous lesions causing damage and histomorphological changes at the dermo epidermal junctions with involvement and changes in the overlying epidermis.

In the present study there was a slight male predilection (54%) as compared to females (46%). 27 of the patients were males and 23 were females. Various studies quoted in the literature showed a predilection of females.[8,9,10,11,12,13] In our study most of the cases were in the age group of 8-50 years of age, similar to a study done by Sehgal *et al* (11- 40 years).[14]

In our study of 50 cases, the commonest clinical presentation was papules noted in 30% (15 of 50) of cases (table 2). In a study of 125 cases, the authors also found a preponderance of papulosquamous lesions (97.6%).[9]

The most common anatomical sites involved by all these lesions were upper and lower limbs mainly extensor surfaces in 30% (15 of 50) cases followed by head and neck, abdomen and thorax (table 3) as compared to other studies in which the lower

Table 8 A summary of the clinicopathological parameters.

Clinical parameter	Lichenoid dermatitis	Interface dermatitis	Miscellaneous
Definition	Band like chronic inflammatory Infiltrate at the dermoepidermal junction	Vacuolar alteration of the basal layer along with chronic inflammatory infiltrate at dermoepidermal junction with or without apoptotic keratinocytes in epidermis	No damage at dermoepidermal junction
Number of cases	26	14	10
Clinical presentation	Papules (12 cases)	Erythematous papules (4 cases)	Hyperpigmented plaques (4 cases)
Site of involvement	Flexor and extensor Surfaces of upper and lower limbs (6 cases)	Targetoid lesions (2 cases) Extensor surfaces of upper and lower limbs (4 cases)	Extensor surfaces of upper and lower limbs (5 cases)
Epidermal changes	Hyperkeratosis without parakeratosis (20 cases) Hyperkeratosis with parakeratosis	Mild keratosis (7 cases) Parakeratosis (3cases)	Mild hyperkeratosis (4 cases)
Epidermal changes	(5 cases) Saw tooth rete ridges (2 cases)	Civatte bodies (6cases)	Focal or confluent parakeratosis (4 cases) Variable granular layer (4 cases)
Dermoepidermal junction	Dense band like chronic inflammatory infiltrate, lichenoid infiltrate (25 cases)	Vacuolar alteration of the basal layer, patchy inflammation (14cases) Apoptotic keratinocytes (4cases) Interface dermatitis (14 cases)	No damage at dermoepidermal junction
Papillary dermis	Lichenoid inflammation (25 cases) Melanin pigment incontinence (9 cases) Civatte bodies (3 cases) Scarring (4 cases)	Melanin pigment incontinence (6 cases) Extravasation of red blood cells (6 cases) Perivascular inflammation (7 cases)	Epitheloid granulomas (2 cases) Collagen (2 cases)
Reticular dermis	Uninvolved	Patchy involvement	Variable involvement by inflammation, collagen
Constitution of inflammatory infiltrate	Entirely of lymphocytes	Lymphocytes, eosinophils, plasma cells	Lymphocytes, neutrophils
Pathogenesis	Cell mediated immunity	Cell mediated immunity, cytotoxic damage to keratinocytes	Variable pathogenesis
Prototype lesions	Lichen planus	Erythema multiforme, drug eruption	Psoriasis

limbs were the most affected site.[15,16] Nail involvement was noted in 2% of the cases in our study (fig 4) as compared to 3.5% in the study conducted by Zaias et al.[17] Review of literature reveals that nails are involved in about 10% cases of lichen planus. Clinicopathological analysis conducted in our study revealed that majority of these cutaneous lesions comes in the category of chronic inflammatory disorders involving superficial cutaneous reactive unit of the skin.[7]

Lichen planus and lichen planus like lesions involve superficial dermis with variable degree of reaction and damage at the dermal epidermal junction such as lichenoid reaction pattern or vacuolar alteration of the basal layer. The primary histologic event leading to the degeneration of basal epidermis appears to be T-cell mediated preceding a sequence of degenerative changes at the dermal epidermal junction, involving the epidermis and leading to variable epidermal reactions.[18] The type of damage occurring at the dermal epidermal junction and the inflammatory reaction pattern in the dermis influence the clinical presentation of the cutaneous lesions.

On the basis of the type of damage occurring at the dermal-epidermal junction and composition and distribution of the inflammatory infiltrate we were able to group the 50 cutaneous lesions into three broad categories- 1) Chronic dermatitis with lichenoid inflammatory infiltrate at dermal epidermal junction (Lichenoid dermatitis), 2) Chronic dermatitis with vacuolar alteration of the basal pigment layer associated with variable infiltrate of lymphocytes at the dermal epidermal junction and presence or absence of apoptotic keratinocytes in the epidermis (interface dermatitis) and 3) Miscellaneous group of chronic dermatitis with no damage at the dermal- epidermal junction. These cutaneous lesions were associated with changes involving other components of superficial cutaneous reactive unit of skin.

In our series we found 52% (26 of 50) cases with lichenoid dermatitis, 28% (14 of 50) cases with interface dermatitis and 20% (10 of 50) cases were grouped in miscellaneous category. Our study revealed that lichen planus with its various subtypes is the classical prototype of lichenoid dermatitis with lichenoid tissue reaction pattern at dermoepidermal junction constituting 48% (24 of 50) cases included in the study (table 4). These findings were comparable to studies done by various authors in which classical lichen planus was the most common lesion encountered.[10,11,16]

In all the subtypes of lichen planus in the study the characteristic histopathological features of lichen planus were present with some variations. Most frequently observed epidermal changes seen were (table 8) hyperkeratosis without parakeratosis in 40% (20 of 50) cases as were seen in studies done by other authors.[10,11,16] Hypergranulosis in 50% (25 of 50) cases almost similar to a study done by Ravikant chauhan *et al* (65.15%).[16] Lichenoid inflammatory infiltrate at the dermal epidermal junction in 50% (25 of 50) cases, which was also one of the most common findings noted in other studies.[10,11,19] Melanin pigment incontinence as a result of damage to the basal pigment layer resulting in clinically hyperpigmented cutaneous lesions was seen in 35% (9 of 26) cases of lichenoid dermatitis as opposed to other studies done by Bhanushree *et al* (93%)[10] , Kumar *et al* (93.33%)[11] and Ravikant *et al* (63.63%).[12]

In our study we found that 28% (14 of 50) cases of interface dermatitis were associated with prominent basal layer vacuolation at the dermal epidermal junction as opposed to other studies quoted in the literature in which vacuolar basal degeneration was present in almost 74-100% cases in studies done by various authors.[10,11,16,19] Perivascular inflammatory infiltrate was noted in 12% (6 of 50) cases (table 8) along with extravasation of RBCs clinically presenting as erythematous lesions as in lupus erythematosus and erythema multiforme.

Cutaneous lesions with prominent vacuolar alteration of the basal layer were lichenoid drug eruptions 7% (1 case), fixed drug eruptions 7% (1 case, fig 8), erythema multiforme 14% (2 cases, fig 9).

Clinicopathological analysis of discoid lupus erythematosus and erythema multiforme which are the prototype lesions of interface dermatitis revealed that these lesions present as well defined erythematous plaques, varying degrees of scaling and perilesional pigmentation. As a result of vacuolar alteration to the basal layer in interface dermatitis there is collateral damage to melanocytes in the basal pigment layer leading to pigment alterations in the skin causing hyperpigmentation and hypopigmentation due to presence of melanophages in the dermis along with variably dense inflammatory infiltrate of lymphocytes and leading to post inflammatory hyper pigmentation as in discoid lupus erythematous.[20]

Presence of apoptotic keratinocytes and civatte bodies in the epidermis indicates more severe immunological damage to the dermal epidermal junction and the epidermis.[21]

Interpretation of pathological findings requires a clear understanding of the reaction patterns associated with the cutaneous lesions.

The study shows that correlation of the clinical findings with histomorphological reaction patterns produced by these cutaneous lesions is helpful in differentiating these lesions and in making a more precise diagnosis by categorizing this vast group of lesions which is very heterogeneous in its pathogenesis into well defined groups.

CONCLUSION

The criteria used to differentiate and categorize the group of cutaneous lesions help in making a more definitive diagnosis and narrow the differential diagnosis amongst this vast group of chronic superficial dermatoses.

The inflammatory changes at the dermoepidermal junction determined to some extent the clinical presentation of these lesions. Therefore, an emphasis on the clinical correlation is crucial for the precise diagnosis of interface dermatitis and histopathological examination remains an indispensable tool for identifying the underlying cause in an order to approach to an early diagnosis and thereby aids in the accurate treatment.

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