UDC 616.5
DOI: 10.15587/2519-4798.2022.258496

STUDY OF CLINICAL AND HISTOPATHOLOGICAL FINDINGS OF INTERFACE DERMATITIS AND ITS CORRELATION

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Interface dermatitis is a broad term used for all the lesions having clinical features and histological features of epidermal basal cell damage and extensive mononuclear cell infiltration in the papillary dermis, all these lesions are also known as lichenoid dermatosis or "Lichenoid tissue reaction" (LTR).

***The aim of the study** was to study in detail histopathological findings associated with interface dermatitis.*

***Materials and methods:** a total of 112 cases were studied. Material for this study included patients who were clinically diagnosed as having interface Dermatitis from the Department of Dermatology, Gandhi Medical College, Secunderabad, during the period from 2009-2011.*

***Results**: clinical diagnosis of the 112 cases diagnosed as interface dermatitis in the present study were as follows: The maximum number of cases 44 (39.29 %) were those of Lichen Planus, followed by discoid lupus erythematosus 10 (8.93 %), vitiligo 10 (8.93 %), lichen planus pigmentosus 9 (8.04 %), erythema multiforme 9 (8.04 %), subacute lupus erythematosus 6 (5.36 %), fixed drug eruption 6 (5.36 %), lichen sclerosis et atrophicus 6 (5.36 %), hypertropic lichen planus 6 (5.36 %) and 1 case of linear lichen planus, lichen plano pilaris, lichen nitidus, bullous lichen planus, atrophic lichen planus, lichen amyloidosis, and drug induced lichenoid reaction.*

***Conclusion**: the interface dermatitis encompasses disease in which there is epidermal basal cell damage, apoptosis of the cell with formation of colloid & civatte bodies, hydropic degeneration of the basal cell, basement membrane thickening, band like or patchy inflammatory infiltrate hugging the dermoepidermal junction and melanin incontinence*

***Keywords:** interface dermatitis, lichen planus, dermoepidermal junction, dermatosis, hydropic degeneration, basal cell, basement membrane thickening, lichen amyloidosis, drug induced lichenoid reaction, patchy inflammatory infiltrate*

How to cite:
Neelima, M., Anitha, S., Karre, S., Devojee, M., Kavitha, D. (2022). Study of clinical and histopathological findings of interface dermatitis and its correlation. ScienceRise: Medical Science, 3 (48), 34–38. doi: http://doi.org/10.15587/2519-4798.2022.258496

1. Introduction

The skin is the largest organ of the body, with a surface area of $2m^2$ and accounting for 16 to 20 % of the total body weight. Human skin is of two types, non-hairy (glabrous) skin (as on the palms and soles) and hair bearing skin [1].

Knowledge of the structure and functions of the skin is essential for the diagnosis and the treatment of skin diseases. The dermoepidermal junction is one of the largest epithelial-mesenchymal junctions in the body, which forms an extensive interface between the dermis and epidermis [2]. One of the most challenging aspects in dermatopathology is to try to make specific diagnosis of inflammatory skin disease. Histological study is one of the most valuable means of diagnosis in dermatology. The greatest diagnostic accuracy is obtained by correlating the clinical and histological finding [3].

Interface dermatitis is defined as a dermatosis in which the infiltrate (usually composed mostly of lymphocytes) appears to obscure the dermoepidermal junction when sections are observed at scanning magnification.

Interface reactions are so named because they are cell-mediated immunologic reactions whose targets are basal keratinocytes that reside above the dermoepidermal junction.

Interface dermatitis is histologically classified into two categories:

a) Interface dermatitis with lichenoid inflammation.

b) Interface dermatitis with vacuolar change [4].

This study is oriented towards the recognition of the histological pattern seen in interface dermatitis with clinical correlation. This will help us in arriving at a more specific diagnosis by light microscopy.

The aim of the study was to study in detail histopathological findings associated with interface dermatitis.

2. Material and methods

The patients were followed up for a period of 3 year from January 2009 to December 2011 in the Outpatient Department of Dermatology and Venereology, Gandhi Medical College, Secunderabad. A total of 112 cases clinically diagnosed as having interface Dermatitis from the Department were studied. Patients' relevant clinical history, personal history, history of any drug intake and particulars about the skin lesion noted in the proforma. The most representative lesion biopsied after taking patients consent. The specimen obtained with

4 mm punch (3 mm in case of face) was immediately fixed in 10 % formalin and completely processed. The tissue bits were subjected to routine processing technique. 4 mm thick sections were prepared from paraffin block and stained with haematoxylin and eosin. PAS staining was done wherever necessary.

The age of patient with interface dermatitis ranged from 8 years to 77 years. Majority of the patients were in the age group of the 30–60 years.

Inclusion criteria

– Clinically suspected cases of Interface dermatitis

– Patients of all age group and both genders were included in this study

Exclusion criteria

– Patients unwilling for biopsy or not giving informed valid consent.

– Inadequate biopsy samples (biopsies showing only dermis or epidermis on histopathological examination)

– Skin biopsies done for cases other than Interface dermatitis.

Ethical institute permission was taken from the institute

Written informed consent was taken from all the patients included in the study. Prospective study was done in the department of Dermatology, Gandhi Medical College, Secunderabad, for duration of 3 years – from January 2009 to December 2011. Ethical clearance is IEC/GMC/2008/05/12 dated 12/5/2008 (Name of ethics commission is from Gandhi Medical College).

Histological examination of skin biopsy

Each skin biopsy was subjected to systematic, critical assessment in sequence of epidermal changes like basal cell death or vacuolar change, varying thickness of different layers of epidermis. Dermal changes like dermatitis and composition of different cell types, focal or diffuse nature of the lesion, pigment incontinence along with appendiceal involvement were noted.

Interpretation

Nucleus-Blue

Cytoplasm-Pink

3. Results

Data was entered in Microsoft excel and analysis was done using SPSS version 20. Descriptive statistical analysis was done. Results on continuous measurements are presented as Mean & Standard Deviation. Results on categorical measurements are presented as Percentages. Significance is assessed at 5 % level of significance. Student t test (independent, two tailed) has been used to find out the significance of study parameters on a continuous scale between two groups. Chi square test is used to find out the significance of study parameters on a categorical scale between two groups.

The present study showed **a** female predominance that is 60.71 %.

Presence of associated illness like diabetes mellitus 33.3 % (3/112), hypertension, hypothyroidism and history of drug intake were noted. 3 patients were diabetic, associated hypertension was seen in 3 of the patients, 6 patients were hypertensive, 3 patients gave history of drug intake. One case associated with hypothyroidism was also encountered.

Distribution of lesions with different type of Interface Dermatitis was categorized. 66.96 % (75 cases) presented with generalized lesions. 33.04 % (37) of patients had localized lesions.

Pruritus was seen in 36 cases (40 %), photosensitivity in 8 (8.89 %) and loss of hair is 2 cases (2.22 %).

Table 1

Distribution of Lichen planus and its variants

Lesions	Lichen planus and its variants								LA	EM	LSEA	LE		FDE	Vitiligo
	LP	HLP	LPP[1]	LLP	LN	LPP[2]	BLP	ALP				DLE	SALE		
Macule	–	1(0.8 %)	4(3.5 %)	–	–	–	–	–	–	1(0.8 %)	–	–	2(1.7 %)	2(1.7 %)	–
Papule	20 (17.8 %)	3(2.6 %)	2(1.7 %)	1(0.8 %)	1(0.8 %)	–	1(0.8 %)	–	–	7(6.2 %)	–	1(0.8 %)	–	–	–
Plaque	22(19.6 %)	3(2.6 %)	1(0.8 %)	1(0.8 %)	–	–	–	1(0.8 %)	1(0.8 %)	5(4.4 %)	1(0.8 %)	2(1.7 %)	2(1.7 %)	3(2.6 %)	–
Patch	2(1.7 %)	–	8 (%)	–	–	1(0.8 %)	–	–	–	–	5(4.4 %)	6(5.3 %)	2(1.7 %)	(3.5 %)	–
Vesicle	–	–	–	–	–	–	–	–	–	–	–	1(0.8 %)	–	–	–
Bulla	–	–	–	–	–	–	1(0.8 %)	–	–	–	–	–	–	–	–

*Note: LP – Lichen planus, HLP – Hyperkeratosis lenticularis perstans, LLP – **Lichen** planus pigmentosus, LN – **Lichen** Nitidus., LPP – lichen planus pigmentosus, BLP – bullous lichen planus, ALP – Atrophic lichen planus, LA – Lichen amyloidosis, EM – erythema multiforme, **LSEA –** Lichen sclerosis et atrophicus, LE – Lupus erythematous, DLE- Discoid lupus erythematous, SELA – sub acute lupus erythematosus, FDE– fixed drug eruption*

Plaques and papules were the dominant lesions in Lichne planus. Hyper pigmented macules and patches were common in patients with lichen planus pigmentosus. Waxy papules were seen in Lichen amyloidosis. Erythematous plaques were seen in patients' erythema multiforme. Patients with Discoid lupus erythematous presented mainly with patches, some with plaque and associated with photosensitivity. All the patients with Lichen sclerosis et atrophicus presented with grey-white patches.

All the cases showing interface dermatitis were examined and analyzed with respect to the histological features which differed in different types of interface dermatitis (Table 1).

Table 2

Distribution of Clinical Diagnosis

Clinical Diagnosis		No. of Cases	Percentage
Lichen planus and its variants	LP	44	39.29 %
	HLP	6	5.36 %
	LPP1	9	8.04 %
	LLP	1	0.89 %
	LN	1	0.89 %
	LPP2	1	0.89 %
	BLP	1	0.89 %
	ALP	1	0.89 %
LA		1	0.89 %
EM		9	8.04 %
LSEA		6	5.36 %
LE	DLE	10	8.93 %
	SALE	6	5.36 %
FDE		6	5.36 %
VITLIGO		10	8.93 %

*Note: LP – Lichen planus, HLP– Hyperkeratosis lenticularis perstans, LLP – **Lichen** planus pigmentosus, LN – **Lichen** Nitidus., LPP – lichen planus pigmentosus, BLP – bullous lichen planus, ALP– Atrophic lichen planus, LA – Lichen amyloidosis, EM – erythema multiforme, **LSEA-** Lichen sclerosis et atrophicus, LE – Lupus erythematous, DLE – Discoid lupus erythematous, SELA – sub acute lupus erythematosus, FDE – fixed drug eruption*

Table 3

Distribution of Histopathological Examination

Significative	Lichen planus and its Variants								LA	EM	LSEA	LE		FDE	Vitiligo
	LP	HLP	LPP[1]	LLP	LN	LPP[2]	BLP	ALP				DLE	SALE		
Hyperkeartossi	4(3.5 %)	6(5.3 %)	9	1(0.8 %)	1(0.8 %)	1(0.8 %)	1(0.8 %)	–	1(0.8 %)	7(%)	5(4.4 %)	7(6.2 %)	5(4.4 %)	5(4.4 %)	6(5.3 %)
Parakeratosis	4(3.5 %)	–	–	–	–	–	–	–	–	7(6.2 %)	–	5(4.4 %)	–	–	4(3.5 %)
Hypergranulosis	12(10.7 %)	–	–	–	–	–	–	–	–	–	5(4.4 %)	–	–	–	–
Follicular plugging	1(0.8 %)	–	4(3.5 %)	–	–	–	–	–	–	–	–	–	3(2.6 %)	–	–
Acanthosis	12(10.7 %)	5(4.4 %)	3(2.6 %)	–	1(0.8 %)	1(0.8 %)	–	–	–	6(5.3 %)	6(5.3 %)	4(%)	–	5(4.4 %)	3(2.6 %)
Atrophy	1(0.8 %)	–	6(5.3 %)	–	–	–	–	1(0.8 %)	–	–	4(3.5 %)	3(2.6 %)	3(2.6 %)	–	–
Basal cell vacuolation	16(14.2 %)	6(5.3 %)	7	2(1.7 %)	1(0.8 %)	1(0.8 %)	–	–	1(0.8 %)	6(5.3 %)	6(5.3 %)	10	4(3.5 %)	5(4.4 %)	5(4.4 %)
Apoptosis	4(3.5 %)	1(0.8 %)	2(1.7 %)	0	–	–	–	–	–	–	–	–	1(0.8 %)	–	–

Note: LP – Lichen planus, HLP – Hyperkeratosis lenticularis perstans, LLP– Lichen planus pigmentosus, LN – Lichen Nitidus., LPP– lichen planus pigmentosus, BLP– bullous lichen planus, ALP – Atrophic lichen planus, LA – Lichen amyloidosis, EM – erythema multiforme, LSEA – Lichen sclerosis et atrophicus, LE – Lupus erythematous, DLE – Discoid lupus erythematous, SELA – sub acute lupus erythematosus, FDE– fixed drug eruption

The epidermal changes observed were hyperkeratosis, parakeratosis, hypergranulosis, follicular plugging, atrophy, basal cell vacuolation and apoptosis. All the cases of Lichen Planus showed hyperkeratosis, irregular acanthosis, hypergranulosis and basal cell vacuolation. Civatte bodies were seen in 30 % of cases. The variants of Lichen Planus seen also showed hyperkeratosis, acanthosis and basal cell vacuolation. Lichen Amylodosis, showed hyperkeratosis, irregular acanthosis. Nine cases of erythema multiforme showed hyperkeratosis, acanthosis, focal spongiosis and basal cell vacuolation. Lesions of lichen sclerosis et atrophicus showed thinned out epidermis with hyperkeratosis. Cases of discoid lupus erythematosis and subacute lupus erythematosis showed hyperkeratosis, follicular plugging, and basal cell vacuolar degeneration. Colloid bodies were seen in 30 % of cases of discoid lupus erythematosus (Tables 2, 3)

All the cases of lichen planus showed moderate to severe band like inflammatory infiltrate in the papillary dermis. Melanin incontinence was seen in 72.22 %. Lymphocyte and plasma cells were the predominant cell type. The variants of Lichen Planus showed mild to moderate inflammatory infiltrate in the papillary dermis. Striking melanin incontinence was seen in all the nine cases of lichen planus pigmentosus. Lichen amyloidosus showed globular eosinophilic deposits in the papillary dermis. Moderate to severe inflammatory infiltrate in the dermis was seen in all the cases of Erythema Multiforme. Subepidermal vesiculation was seen in 4 (50 %) of the 9 cases. Perivascular and periappendageal inflammatory infiltrate was seen in discoid lupus erythematosus. Milder inflammation was seen in sub-acute lupus erythematosus. Cases of lichen sclerosis et atrophicus showed homogenisation of the papillary dermis and lymphocytic infiltrate beneath it. Focal areas of basement destruction was seen in all cases of Lichen Planus and its variants, and erythema multiforme. Basement membrane thickening was seen in discoid lupus erythematosus and subacute lupus erythematosus.

All the patients with interface dermatitis were followed up for a period of one year and clinically they were assessed. 87 cases were cured, ten patients were not available for follow up and fifteen patients are still under treatment showing improvement.

The first clinical differential diagnosis had a very good significant P value (Table 4).

Table 4

Correlation of the histological diagnosis with clinical differential diagnosis

Agreement	Percentage	P Value
DD1	50	< 0.0001
DD2	32.89	< 0.0001
DD3	9.3	< 0.0043

4. Discussion

The accurate diagnosis of inflammatory conditions in dermatopathology requires integrating the histopathologic findings with clinical features. Interface reactions are so named because they are cell mediated immunologic reaction, whose targets are basal kertinocytes that reside above the dermoepidermal junction. An attempt has been made in this study to diagnose the various lesions of interface dermatitis by a pattern based histopathologic appearance and correlating with clinical features.

The age incidence of interface dermatitis was found to be maximum between the age group of 20–60 years (78.89 %). This correlate with the observation by Tompkin J. K. et. al. [5] in 1995, who also noted maximum incidence in the age group of 20–60 years.

Present study showed a female preponderance (60.71 %). Tompkin J. K. et al [5] also noted 61 % incidence in females. Study by Singh and Boyd et al [6] have reported a familial incidence of Lichen Planus of 10.7 %. Familial association was not seen in our study. Familial association has also been reported in systemic Lupus Erythematosus. An association with Diabetes mellitus is seen in 3 patients, Hypertension in 6 patients, both diabetes mellitus and Hypertension in 3 patients and Hypothyroidism in one patient. History of drug intake was noted in 3 patients. Krishnendra Varma et al [7] among 100 patients with LP, 33 (33 %) had diabetes with a significant statistical association. Intake of anti-hypertensives for a long time has been associated with lichen planus like skin eruption.

Clinical diagnosis of the 112 cases diagnosed as interface dermatitis in the present study were as follows: The maximum number of cases 44 (39.29 %) were those of Lichen Planus, followed by discoid lupus erythematosus 10 (8.93 %), vitiligo 10 (8.93 %), lichen planus pigmentosus 9 (8.04 %), erythema multiforme 9 (8.04 %), sub-acute lupus erythematosus 6 (5.36 %),fixed drug eruption 6 (5.36 %), lichen sclerosis et atrophicus 6 (5.36 %), hypertropic lichen planus 6 (5.36 %) and 1 case of linear lichen planus, lichen plano pilaris, lichen nitidus, bullous lichen planus, atrophic lichen planus, lichen amyloidosis, and drug induced lichenoid reaction.

4. 1. Comparative studies related to clinical features

44 cases of Lichen Planus constituted 39.29 % of the study. Lesions were mostly seen on the extremities. 2 of the cases also had genital lesions. In Manjunath et al study [8] Pruritus was seen in 36 cases (40 %), photosensitivity in 8 (8.89 %) and loss of hair is 2 cases (2.22 %)

Multiple lesions with papules and plaques with a violaceous hue were seen in all the cases. Similar findings have been reported by Boyd et. al. [6]. All the 9 cases of lichen planus pigmentosus had the disease for 6 months to 3 years. Face and neck were the commonest site affected. This confirms to report by Knawar A. J. et al. [9].

Lichen plano pilaris presented as localized pruritic patches over the scalp in one and hypopigmented patch over the back in other.

Lichen amyloidosis seen in our study was a male patient with waxy papules in generalized manner. Weedon et al mentions extensor aspects of lower extremities as the favoured site.

All the nine patients with Erythema multiforme had localized erythematous plaques over the extremities. Similar findings have been reported by Le Boit PE [10].

Six cases of lichen sclerosis et atrophicus were encountered, all of them were postmenopausal females, presented with grey-white patches in the vulvar region, which concurs with findings of Marfatia et al. [11].

Of the 10 patients with Discoid lupus erythematosus 6 patients (60 %) had localized cutaneous involvement of the head and scalp, and 4 patients (40 %) had generalized form. 60 % of the patients had photosensitivity. 75 % of subacute lupus erythematosus were seen in women on sun exposed areas. A single case of drug induced lichenoid reaction seen in our study gave the history of being on treatment with anti-tuberculosis drugs. This might have initiated the lesion.

4. 2. Comparative studies related to Histopathology

The epidermis in Lichen Planus showed hyperkeratosis, hypergranulosis and irregular acanthosis in almost all cases. This conforms to the findings reported by Boyd et. al. [6]. Civatte bodies were seen in 30 %.

All the cases of hypertrophic lichen planus showed psoriasiform hyperplasia of the epidermis, dermal infiltrate near the tip of the rete ridges and vertically oriented collagen fibers in the papillary dermis. Similar findings have been recorded by Weedon et al. [12].

The histopathological changes with lichen planus pigmentosus consisted of vacuolar degeneration of basal layer of the epidermis, hyperkeratosis, and mild dermal lymphohistiocytic infiltrate and melanin incontinence with melanophages. Manjunath et al [12] observed the epidermal changes observed were hyper keratosis (HK), para keratosis (PK), hyper granulosis (HG), follicular plugging (FP), atrophy, basal cell vacuolation and apoptosis. All the cases of LP showed HK, irregular acanthosis, HG and basal cell vacuolation. Civatte bodies were seen in 30 % of cases. The variants of LP also seen showed HK, acanthosis and basal cell vacuolation. The five cases of lichen amylodosis, showed HK in all, irregular acanthosis in 80 %, and FP in 20 %. Four cases of EM showed HK, acanthosis, focal spongiosis and basal cell vacuolation.

Lichen amyloidosis showed irregular acanthosis of the epidermis. Small globular deposits of eosinophilic hyaline material in the papillary dermis were seen in all the cases. These findings confirmed with those mentioned by Weedon et al. [12].

Erythema multiforme: Histologically all the seven cases showed hyperkeratosis, basal cell vacuolation, civatte bodies, moderate to severe inflammatory infiltrate in the dermis. Subepidermal vesiculation was seen in the 2 of the 4 cases. This conforms well with the observation of Le Boit PE. [10]. The cases of lichen sclerosis et atrophicus showed thinned out epidermis with hyperkeratosis, a wide band of homogenized collagen below the dermoepidermal junction and a lymphocytic infiltrate beneath the homogenized area.

All the cases of discoid lupus Erythematosus histopathologically showed hyperkeratosis, follicular plugging, variable degeneration of basal cells, some with civatte bodies, thickened basement membrane, pigment incontinence

and perivascular, perifollicular mononuclear infiltrate of the dermis. Paolo Fabbi et al [13] have recorded similar findings.

Subacute lupus erythematosus lesions show relative absence of deep dermal and subcutaneous perivascular inflammatory infiltrate. This may account for failure of this lesion to develop the central atrophy that is characteristic of discoid lupus erythematosus.

Research limitations. The presence of interface lichenoid infiltrates cannot be considered as a single criterion for the diagnosis of interface dermatitis and its variants. We should also consider clinical presentation, thus aiding accurate diagnosis and treatment.

Prospects for further research. In the present study, the diagnosis is based on histomorphology and clinical presentation which could have subjective bias these would have been overcomed by ancillary techniques such as immunohistochemistry targeting antigens of these dermatoses. In future research need to be concentrated on elucidating the antigenic targets of interface dermatoses which could help us understand their pathogenesis further

5. Conclusion

The interface dermatitis encompasses disease in which there is epidermal basal cell damage, apoptosis of the cell with formation of colloid & civatte bodies, hydropic degeneration of the basal cell, basement membrane thickening, band like or patchy inflammatory infiltrate hugging the dermoepidermal junction and melanin incontinence. Most of the component of the lichenoid spectrum exhibits this reaction, except for subtle difference that defines the variant. Recognition of this commonly encountered cutaneous problem depends upon the familiarity of clinical presentation and the diagnosis can be confirmed with histopathology.

The pathologist ability to render an accurate diagnosis depends on the available clinical information. Every specimen submitted for histopathology should be accompanied by clinical information & include a differential diagnosis so clinicopathological correlation is the key to the patient care.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Financing

The study was performed without financial support.

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Received date 05.04.2022
Accepted date 17.05.2022
Published date 31.05.2022

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