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Clinicopathological Spectrum of Interface Dermatitis

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Abstract

Introduction: *Interface dermatitis, which is characterized by variable dermoepidermal interface inflammation and the presence of vacuolar damage to the basal layer, which causes keratinocyte death or necrosis, is a broad term that encompasses many distinct illnesses. . Aim: The present study was planned to correlate the significance of Interface dermatitis with their clinico-pathological aspects.*

Methodology: *This was a retrospective study conducted on 170 cases diagnosed as interface dermatitis over a period of two years. Skin biopsy samples received from the outpatient department were analysed.*

Results: *In the present study, a total of 170 cases were analysed which clinically presented as papulosquamous disorders. On histopathological diagnosis, LP and its variants were the most common type disorder. The most consistent histolopathological characteristic feature noted was basal cell vacuolar degeneration in the epidermis along with a band of lymphocytic inflammatory infiltrate. Clinicopathologic concordance was seen in 86.5% cases.*

Conclusion: *Thus, combination of the histologic details, in correlation with the clinical data helps in arriving at a specific diagnosis of various ID.*

Introduction

Interface dermatitis is a broad term for a variety of conditions marked by variable dermoepidermal interface inflammation and an appearance of vacuolar disruption to the basal layer, which causes necrosis or death of keratinocytes.(1) According to the presence of the inflammatory infiltrates creating alterations at the dermo-epidermal junction, interface dermatitis is typically classified into two categories: a) vacuolar (cell-poor) and b) lichenoid (cell-rich).(2) Hydropic or liquefactive basal layer degeneration is present in all disease entities associated with interface dermatoses. Depending on the type of accompanying epidermal abnormalities, interface dermatitis is further subdivided into hyperplastic with irregular epidermal hyperplasia, hyperplastic with psoriasiform epidermal hyperplasia, and atrophic /pokilodermatosus.(2,3)

The terms interface, vacuolar, and lichenoid have been used interchangeably over the years. Weedon called all of the disease categories as "lichenoid."

The word "lichenoid" was also preferred by Calonje et al. to characterize an epidermal condition in which there is a band-like infiltration that obscures the dermoepidermal junction and basal cell vacuolization or the observed hydropic change.[2-4] It is possible to classify all the entities associated with interface dermatoses, basal layer alterations, and the existence of apoptotic keratinocytes using a range of classification schemes that consider the least common factors, namely, clinical, histological, immunological, and etiological factors.[2,5] The Le Boit classification, which was created in 1995, is the most widely used histological categorization based on epidermal alterations.[6]

Etiopathogenesis of interface dermatitis

Various factors have been implicated in the pathogenesis of lichen planus such as mechanical trauma, systemic reactions, drugs and contact sensitivity, infective agents as well as a genetic predisposition caused by the polymorphism in the TNF Alpha gene activity.[7-9] Since T cells make up the majority of the inflammatory infiltrate, cell-mediated cytotoxicity is hypothesized to have a role in the aetiology of lichen planus. Basal keratinocytes' antigen presentation is considered to result in T cell buildup in the superficial lamina propria, rupture of the basement membrane, and intraepithelial T-cell migration, which leads to CD8+ cytotoxic cell-mediated keratinocyte apoptosis in lichen planus.[2,5,10] According to recent research, a signalling cascade activated by interferon-alpha generated by plasmacytoid dendritic cells appears to exacerbate the damage inflicted by cytotoxic T cells to the epidermal basal cell compartment. This pathway is believed to have had a role in the creation of the prototype lichen planus, in addition to other cellular and molecular processes.[2] It has been noted that ischemia is a result of lymphocytic vasculopathies, which also lead to ischemic cell death.[5]

The clinical appearance of ID lesions is determined by the type and degree of epidermal development as well as the dermoepidermal inflammation.[3,5,11] Lesions almost always have a lichenoid appearance, although they can also develop as macular lesions and plaques. However, these lesions along with surface changes may seem violaceous due to inflammation, basal layer vacuolation, and melanin incontinence. Therefore, it is evident that interface dermatitis lesions can progress to hypopigmented or post-inflammatory pigment modification lesions, and vice versa.

Clinically lichenoid lesions may appear: planar (flat topped), purple, polygonal, pruritic and show the presence of papules or plaques.[12] Vacuolar type of interface dermatitis is marked by various (multiform) lesions that include papules, macules, vesicles, bullae, and target lesions. Additionally, some entities might be linked to liver, gastric, and skin involvement. An erythematous maculopapular rash that starts on the palms and soles and spreads to other areas of the body is another possible presentation. After that, it might advance to vesicle and bulla.[1]

The primary diagnostic criteria for interface dermatitis include basal layer degeneration, vacuolar destruction, and a predominant infiltration at the dermoepidermal junction. The superficial infiltration density observed in cell-poor interface dermatitis variations misinterpreted as "perivascular dermatitis missing epidermal changes" determines the degree of error committed in the initial stages of diagnosis Distinguishing early vacuolar degeneration in the basal layer from basilar spongiosis is the second diagnostic challenge faced by pathologists. Atrophy (poikilodermatoses), regular or irregular (hypertrophic) psoriasis, and a range of epidermal abnormalities, including as vacuolar degeneration and epidermal hyperplasia, can be observed in association with each other. Psoarifiorm or spongiotic dermatitis exhibit reaction patterns associated with inflammatory dermatitis, and additional findings such as basement membrane

thickening and inflammatory infiltrate deposition observed in interface dermatitis are helpful in the diagnosis of connective tissue disorders. It is discovered that there are a number of neoplastic mimics of interface dermatitis, such as mycosis fungoides (lichenoid or poikilodermatosus pattern exhibiting disproportionate epidermotropism) and regressing melanocytic neoplasms (malignant melanomas which demonstrate active inflammatory remission).[(1)]

The current study was conducted to study the histomorphological findings of diseases associated with interface dermatitis and to classify interface dermatitis based on epidermal changes (Le Boit classification). The study also attempted to correlate clinical presentation with histological features and diagnosis.

Methodology

A retrospective descriptive study was conducted in the pathology department of a teaching hospital from August 2020 to December 2022 after taking permission from Institutional Ethical Committee. A total of 170 patients diagnosed as inflammatory diseases belonging to interface dermatitis from January 2017-December 2019 were included in the study. Skin biopsy samples were received in the department and fixed in 10% neutral buffered formalin. 3.5 mm thick sections were cut from the paraffin embedded blocks and stained with hematoxylin and eosin. Wherever necessary, PAS staining was performed. All the slides were examined for histopathological changes. Clinical history and results of other investigations of the cases was taken from the patient's individual case sheets from the medical records department. The clinico-pathological correlation for individual cases was analysed. The data was collected and tabulated in Microsoft excel sheet. The percentages were calculated for purpose of comparison.

Inclusion Criteria: All the skin biopsies that showed dermo-epidermal inflammatory infiltrate were included in the study.

Exclusion Criteria

1) All skin biopsies with histopathological findings other than dermo-epidermal inflammatory infiltrate.
2) Skin biopsies whose blocks/ slides could not be retrieved.
3) Neoplastic mimics of interface dermatitis (including regressing malignant melanomas showing active inflammatory regression, mycosis fungoides, and superficial basal cell carcinoma)

Results

Figure 1: Clinical presentation of the cases

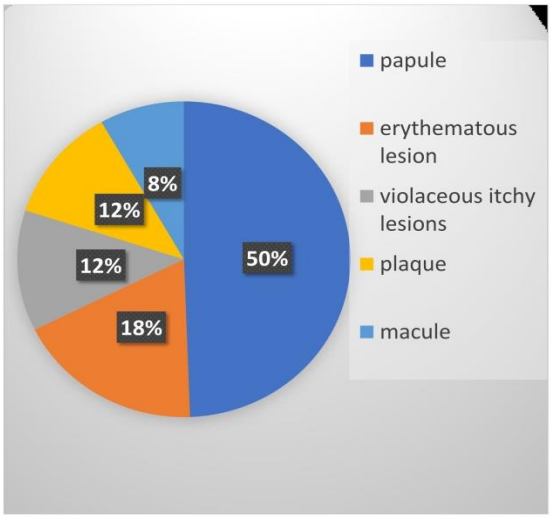

Figure 2: Clinicopathological correlation

Figure 3: 92% (158) out of 170 cases showed presence of spongiosis

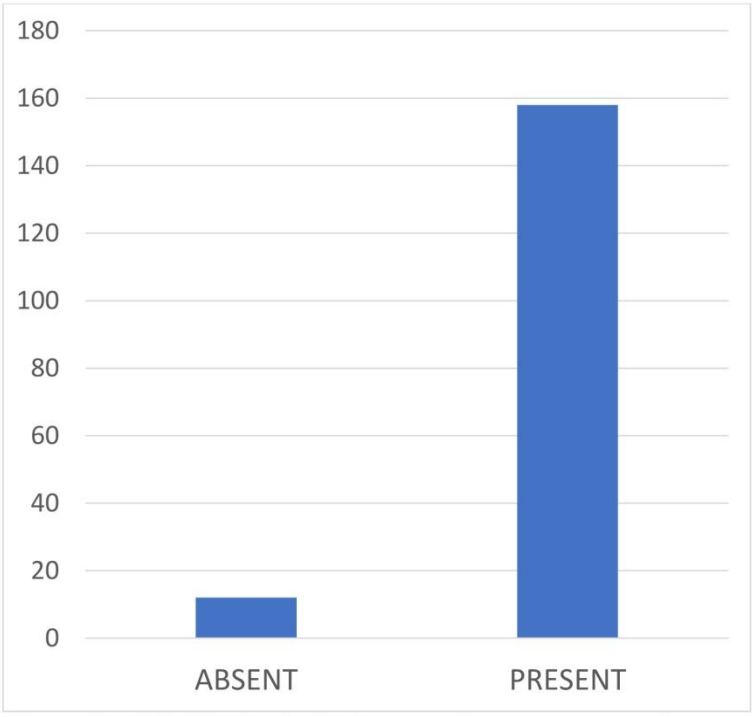

Figure 4- Lichen Planus: Epidermis showing spongiosis, irregular acanthosis, saw toothed rete ridges, lymphocytic exocytosis, and basal cell vacuolation overlying dermis showing dense lymphocytic inflammatory infiltrate obscuring the dermoepidermal junction.(H & E; X400)

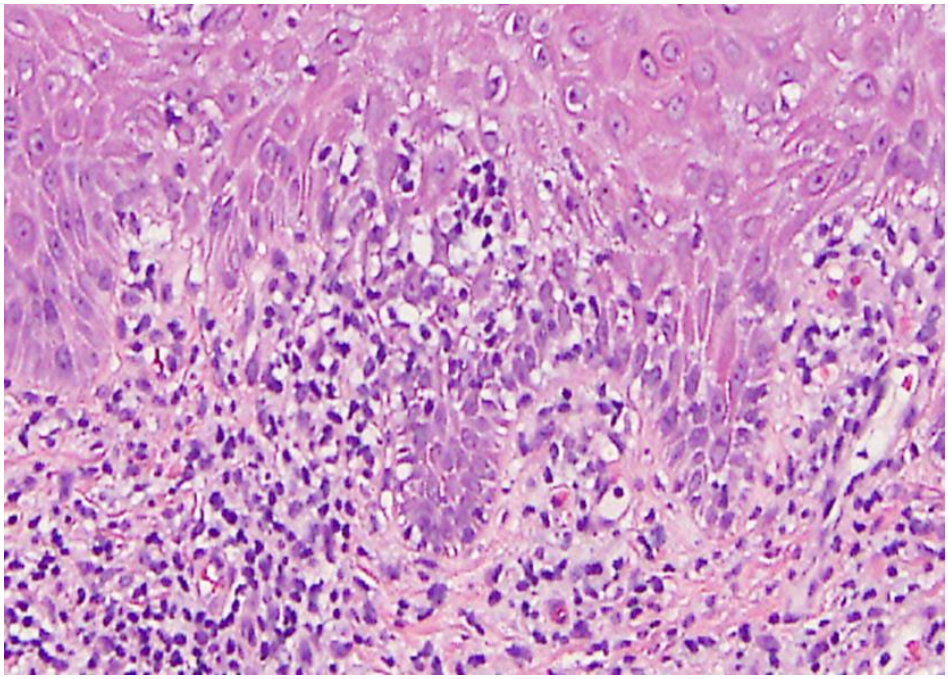

Figure 5– Lichenoid Dermatitis: Epidermis with focal acanthosis, spongiosis, necrotic keratinocytes, and basal cell vacuolation overlying papillary dermis containing dense infiltration by lymphocytes, histiocytes and plasma cells, and melanin incontinence (H&E; X200

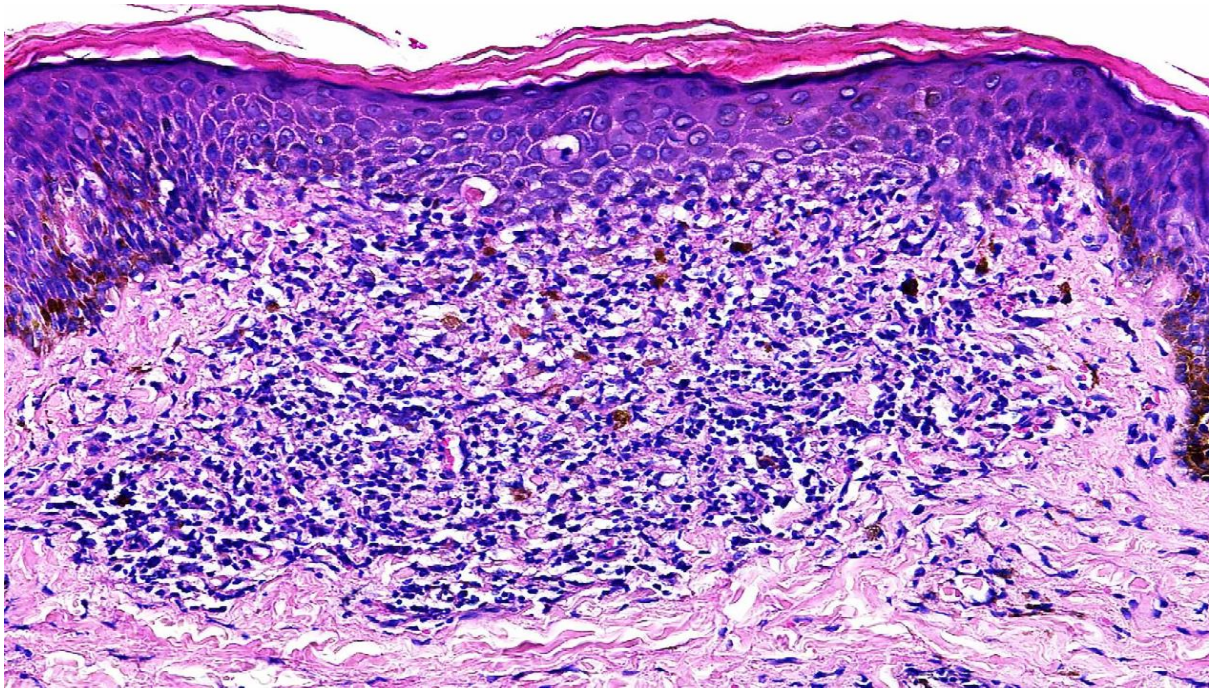

Table 1: Common histological diagnosis

Disease entity	No of cases
Lichen Planus	50
Lichen Planus pigmentosus	36
Hypertrophic Lichen Planus	18
Lichen Actinicus	4
Lichenoid dermatitis	2
Lichen nitidus	6
Lichen sclerosus	14
Lichen Amyloidosis	4
Lichen Planopilaris	2
Lichenoid photodermatitis	3
Lichenoid drug reaction	5
Ashy dermatoses	2
Erythema multiforme	2
Discoid lupus erythematosus	10
PLEVA	1
Pityriasis Lichenoides chronica	9
Poikiloderma atrophicans	2

Table 2: Type of inflammatory infiltrate constituents at the interface

Inflammatory infiltrate	No of cases	Conditions
Lymphocytes only	66	Hypertrophic lichen planus, Lichen planus, lichenoid dermatitis
Lymphocytes + Neutrophils	23	lichen planus pigmentosus , lichen nitidus, PLEVA
Lymphocytes + Histiocytes	45	Lichen nitidus, Hypertrophic lichen planus, Lichen planus, Lichen planus pigmentosus, lichen sclerosis, Pityriasis lichenoides chronica
Lymphocytes + Plasma cells	30	Lupus erythematosus, lichen planus, lichenoid dermatitis, lichenoid dermatitis
Lymphocytes + Eosinophils	6	Lichenoid drug reaction, lichen sclerosus, lupus erythematosus

Table 3: Cases showing the presence and absence of parakeratosis

Parakeratosis		
	No of cases	**Percentage**
Absent	70	41.2
Present	100	58.8
Total	170	100

Table 4: Cases showing the presence and absence of orthokeratosis

Orthokeratosis		
	No of cases	**Percentage**
Absent	17	10
Present	153	90
Total	170	100

Discussion

A comprehensive histological study is necessary to offer an early diagnosis and enable timely and efficient treatment because it might be difficult to differentiate between the several lesions associated with interface dermatitis clinically.[5] Dermatopathologists commonly meet tissue sections that reveal different degrees of dermo-epidermal (interface) inflammatory infiltration, necrotic keratinocytes/ colloid bodies/ civatte bodies, hydropic / vacuolar degeneration of the basal layer of the epidermis.[1,5] The goal of the current study was to categorize interface dermatitis based on epidermal alterations (Le Boit classification) and to examine the histomorphological findings of diseases linked to the condition.

In our study of 170 cases of ID, the lesions clinically presented with papulosquamous lesions included papular lesions (49.4%) were the most common followed by erythematous (18.23%), plaque type (11.7%), violaceous itchy lesions (12.35%) and the least common being the macular lesions (14%). Similar results were obtained in studies by Hegde et al.[5] and Tickoo et al.[13] in which the most common papulosquamous lesions were the erythematous and mixed type.(Figure 1)

In our study the most common histological diagnosis was found to be of lichen planus and its variants which constituted 109 cases (64%).(Table 1) This was in accordance to a study done by Hegde VK and colleague in 2014[5] wherein Lichen planus was noted as the predominant type of ID. Among the lichen planus and variants, classical lichen planus was seen in 50 cases (29.4%), lichen planus pigmentosus in 36 cases (21.2%), hypertrophic lichen planus in 18 cases (11%), lichen actinicus in 4 cases (2.4%). The least number of cases in our study were PLEVA, erythema multiforme, poikiloderma atrophicans, ashy dermatosis, and lichen planopilaris.

A band-like infiltration is a characteristic of interface dermatitis. Differentiating characteristics of interface dermatitis include density, pattern, and composition of the infiltrate, which aid in more precise diagnosis for different types of interface dermatitis[5]. In our study of 170 cases, 90% cases were found to be the cell rich type and 10 % were cell poor type of interface dermatitis. The cell poor variants included entities like 1 case of PLEVA, 10 cases of Lupus erythematosus including 9 cases of DLE and 1 case of SLE, 9 cases of Pityriasis lichenoides chronica, 2 cases of Poikiloderma atrophicans, 4 cases of Lichen amyloidosis, 2 cases of Ashy dermatoses, 2 cases of Erythema multiforme and 14 cases of Lichen sclerosus. The majority of cases (66 cases) in our study showed only lymphocytic infiltration at the interface which is equivalent to the findings of Dhar et.al [14] and Kumar et.al[8]. Other combinations of inflammatory infiltrate identified in our study included 23 cases of lymphocytes and neutrophilic infiltrate, 45 cases of lymphohistiocytic infiltrate, 30 cases showed lymphocytes along with plasma cells, and 6 cases

presented with lymphocytes along with eosinophils. Table 2)

In the current study, out of 170 cases, only 147 were observed to be correlating with the clinical diagnosis. Some discordant cases which were clinically diagnosed as pityriasis lichenoides chronica turned out to be lichen planus when evaluated histologically and vice versa. Few were clinically diagnosed as psoriasis or psoriasiform dermatitis, lupus erythematosus and mycosis fungoides which were the histologically variants of lichen planus. The probable reason for discordance could be the presence of similar clinical picture in those cases, the site of biopsy as well as the age of the lesion. The present study showed a clinicopathological concordance rate of 86.5% (Figure 2) which was similar to that obtained in different studies conducted by Hegde et al[5], Maheshwari GR et al.[3], and Kumar et al.[8] wherein a concordance of 87.2%, 70.9%, and 83.2% respectively was observed. The variation in concordance could be attributed to the variation in sample size and disease prevalence in their region.[9,10]

Le Boit[6] used epidermal changes to classify interface dermatitis because they are easier to interpret and more dependable. This approach is based on the discovery that different diseases exhibit diverse epidermal abnormalities associated with interface dermatitis, which are indicative of pathophysiological differences. Furthermore, the pattern and density of inflammatory cells in the papillary dermis are poorly correlated with the epidermal changes.[5,9]

Table 5: Classification of diseases with ID found in our study according to Le Boit groups.[5,6]

Types	Categories	Disease entities found in our study
Type 1	Acute cytotoxic type (Prototype: Erythema Multiforme)	Erythema multiforme PLEVA
Type 2	ID with premature terminal differentiation (Prototype: Lichen Planus)	Lichen planus (classical) Lichenoid drug reaction Discoid lupus erythematosus Lichenoid photodermatitis Lichen planus pigmentosus Lichen planopilaris Lichen nitidus Lichen actinicus Ashy dermatoses
Type 3	ID with psoarsiform hyperplasia	Pityriasis lichenoides chronica
Type 4	ID with irregular epidermal hyperplasia	Hypertrophic Lichen planus Hypertrophic discoid lupus erythematosus
Type 5	ID with epidermal atrophy	Poikiloderma atrophicans Lichen sclerosus Lichen planus pigmentosus

In the present study, Type 2 was the most predominant type which was in concordance to a study conducted by Kumar S[8] who also obtained a similar result in their study. Parakeratosis was present in 58.8% cases in the current study (Table 3) which was much more than the 8% and 16% as seen in studies by Batchu et al [10] and Hegde et al[5] respectively. Orthokeratosis on the other hand was seen in 90% cases in the current study (Table 4) which was in concordance to that obtained in studies by Batchu et al[10] and Hegde et al[5] who obtained 72% and 74.4% respectively. Spongiosis was present in 92.94% cases in the current study (Figure 3) which was more than that obtained in studies by Kumar et al[8] and Hegde et al[5] (65.42% and 72% respectively)

The current study shows that histological investigation can produce a valid diagnosis; however, since clinical features can lead to clinicopathological incongruence, they should be taken into account. These features include the lesion's location, duration, and type, drug intake history, and any other disease association. The thickness of the blood vessel wall, the presence of fungi, and the thickness of the basement membrane were all assessed using the Periodic Acid Schiff (PAS).(10) Pathologists and dermatologists could cooperate to carry out a more in-depth analysis. Future research can also

look at immunopathogenesis to have a complete understanding of the immune system-related alterations that take place in ID.

Conclusion

Dermatologists and pathologists may find it difficult to discern between different entities in lichenoid eruptions due to slight changes and distinctive features that aid in the diagnosis. A conclusive diagnosis requires clinicopathological correlation. Lichen planus and its variations were the most prevalent kind of interface dermatitis seen in the current investigation. There was an 86.5% clinicopathological concordance rate, with lichen planus having the highest frequency of histological diagnosis. The histologic presentation of various diseases varies significantly depending on the body site sampled, the quality of the sample, and the most important stage of the lesion's evolution sampled. This demonstrates how crucial histology is to establishing a clinical diagnosis.

Strengths and Limitations

An adequate sample size with detailed clinical history provided by our efficient dermatologist colleagues and a good clinicopathological concordance could be considered as strengths if the study. In addition, it also provides us with a thorough understanding of the disease entities presenting as interface dermatitis in our population. However, the current study is not without limitations. Cell rich ID instances were more common than cell poor ID cases in our study. The likely cause is that the majority of cell deficient kinds are acute forms, which are less frequently biopsied because they are clinically identified. The results do not represent the prevalence in a vast population or area because they are based on a specific population or location. Further research is needed which can be accomplished through a nationwide interinstitutional collaboration. There is some bias because the study was retrospective in nature and the histological diagnosis were made beforehand. One possible addition to the study would have been direct immunofluorescence (DIF). Histologically, they showed a mixed image because in some of the instances, the reason was either superadded infection/inflammatory reaction or partial treatment received elsewhere (prior to biopsy).

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