Original Article

# Patterns of Interface Dermatosis - A Comparative Analysis of Clinical and Histopathological Features



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## ABSTRACT

**Objective:** To analyze the frequency of various histopathological patterns of interface dermatitis and to compare the clinical and histopathological diagnosis.

**Study Design:** Comparative, cross-sectional study

**Place and Duration of Study:** This study was conducted at the Histopathology Department, AMC (Army Medical College Rawalpindi), National University of Sciences & Technology (NUST) Islamabad and Combined Military Hospital (C.M.H) Rawalpindi, Military Hospital (MH) Rawalpindi, from December 2018 to December 2019.

**Materials and Methods:** Skin biopsies of the clinically diagnosed cases of interface dermatitis/lichenoid dermatitis were collected over a period of one year and were categorised on the bases of intensity and type of interface inflammation, prominent histopathological features and then compared with the clinical features.

**Results:** A total of 115 samples were collected from the patients who were clinically diagnosed with interface dermatitis or had a previous history of interface dermatitis and presented with a new skin lesion. Histopathology confirmed interface dermatitis in 97 cases. Lichen planus (58.8%) was found to be the most common cell rich type. Erythema multiforme (50%) was the most common cell poor variant and amid the sub-epidermal blistering disorders, bullous pemphigoid (62.5%) was the most frequent blistering disorder showing interface dermatitis.

**Conclusion**: Interface dermatitis is a clinically diverse entity and clinicopathological correlation helps in differentiating diseases showing interface dermatitis and arriving at an accurate diagnosis.

**Key Words:** Dermo-epidermal junction, interface dermatitis, lichenoid dermatitis, vacuolar alteration

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## INTRODUCTION

Skin is a large organ that not only acts as a barrier to the factors affecting internally and externally but also manifests many of the clinical signs of internal diseases[1]. A number of skin diseases are categorized as superficial inflammatory dermatosis based upon the pattern of tissue reaction and inflammation [2]. One of the categories of the superficial inflammatory dermatosis includes a group of clinically diverse and least known inflammatory skin disease known as interface dermatitis/lichenoid tissue reaction which is characterized by a particular set of histopathological elements [3].

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Diagnosis of interphase dermatitis/lichenoid tissue reaction may present a problem due to variations in clinical and histopathological patterns. In interface dermatitis the principal pathology involves the muddling of dermo-epidermal junction by inflammatory cells comprising mainly of lymphocytes[4]. A number of diseases depending on type, distribution and density of inflammatory cells at the dermoepidermal junction are included in interface dermatitis however they occasionally present the same clinical picture. Thus in order to initiate appropriate therapy the differentiation of one from the other becomes necessary. Clinicopathological correlation is crucial to speculate the sequence and the ideal treatment of the disease. Clinicopathological correlation of lichen planus, a prototypic disease of interphase dermatitis/lichenoid tissue reaction showed that the diagnostic accuracy rate increases by hundred percent when confirmed histopathologically [5,6].

Various histological patterns are seen in interphase dermatitis such as mild or dense inflammatory infiltrate comprising mainly of mononuclear cells, basal cell degeneration causing hydropic change, apoptosis leading to formation of civatte or colloid bodies and pigmentary incontinence due to damage to basal keratinocytes and melanocytes.

**Lymphocytic rich Lichenoid Tissue Reaction/Interface Dermatitis**

1. Discoid lupus erythematosus (LE)
2. Fixed drug eruption
3. Keratosis lichenoides chronica
4. Lichen nitidus
5. Lichen planus
6. Lichen striatus
7. Lichenoid drug reactions
8. Lichenoid and granulomatous dermatitis
9. Lichenoid mycosis fungoides [7]

**Lymphocyte-Poor Tissue Reaction/Interface Dermatitis**

1. Acute graft-versus-host skin disease
2. Autoimmune connective tissue skin diseases
3. Acute cutaneous LE
4. Subacute cutaneous LE
5. Dermatomyositis
6. Mixed connective tissue disease
7. Erythema multiforme
8. Erythema multiforme minor
9. Erythema multiform major (Stevens Johnson Syndrome)
10. Interface dermatitis of HIV infection
11. Morbilliform exanthems
12. Virus-induced
13. Drug-induced
14. Paraneoplastic pemphigus
15. Pityriasis lichenoides Chronica [8]

**Granulocyte subepidermal blistering disorders showing interface dermatitis**

**Cell Rich**

1. Eosinophil predominate
2. Bullous pemphigoid
3. Neutrophil predominate
4. Dermatitis Herpetiformis
5. Epidermolysis bullosa Acqusita
6. Mixed neutophil and eosinophil
7. Linear IgA disease
8. Bullous SLE

**Cell Poor**

1. Epidermolysis bullosa hereditary
2. Diabetic Blister
3. Porphyrias
4. Blister in comatosed patients [2]

The statistics of histopathological patterns of interface dermatitis have not been well documented in our setup. The purpose of designing this study is to address this particular problem precisely.

# MATERIALS AND METHODS

The study was undertaken at the department of histopathology of Army Medical College RWP. The department of dermatology, Combined Military Hospital and Military Hospital Rawalpindi collaborated in executing the study. The study was conducted during 12 months time from December 2018 to December 2019. A detailed medical history was obtained from all the participants of the study. Elliptical full thickness skin biopsy was taken. The prominent histopathological features were included in the diagnostic, intensity of interface inflammation and then clinical features were compared with the results afterwards.

# RESULTS

The present study includes one hundred and sixteen patients and the average age was 38 years (SD $\pm$ 19.68) with minimum age of one year and 85 years as maximum age. Majority of patients were male (74.1%) while 25.9% were female. Out of 115 patients, 81 (69.8%) were diagnosed as interface dermatitis both clinically and histopathologically. Sixteen cases (13.8%) were considered negative clinically for interface dermatitis but on histopathological examination they were confirmed to have interface dermatitis. Eighteen (15.5%) cases were identified with interface dermatitis clinically only while on histopathological examination they were diagnosed with diseases other than interface dermatitis**.** According to histopathological diagnosis, majority of patients; 67 (57.8%) out of 115, were diagnosed as cases of lymphocyte rich IFD. Lymphocytic poor IFD was seen in 21 (18.1%) patients. Granulocyte blistering disorders showing interface dermatitis was confirmed in 9 (7.8%) cases. Nineteen (16.4%) patients were corroborated with diseases which differed from interface dermatitis. The most prevalent disease was found to be lichen planus (figA) rich variant (59.7%).

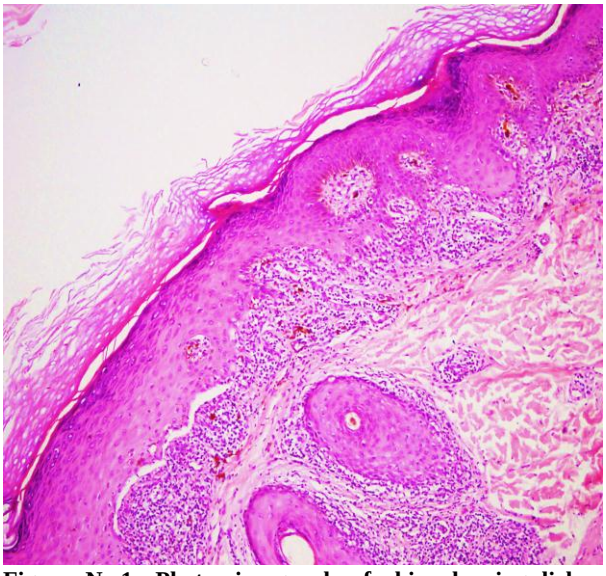

**Figure No.1: Photomicrograph of skin showing lichen planus with hyperkeratosis, prominent granular cell layer, Max Joseph space, band like inflammatory infiltrate and pigmentary incontinence (H&E x 100)**

Erythema multiforme was the most common lymphocytic poor variant (47.6%) and bullous pemphigoid was the most common granulocyte

blistering disorder (55.6%) showing interface dermatitis.

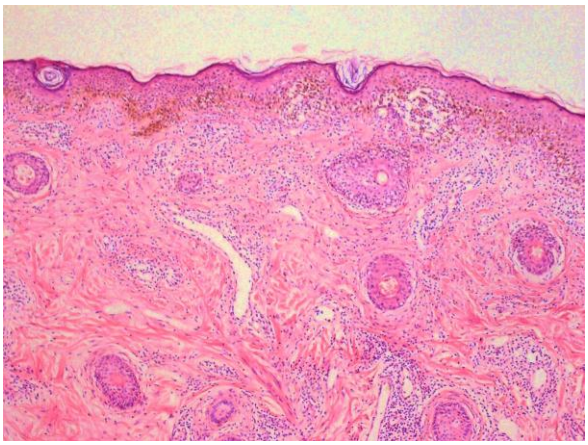

**Figure 2: Photomicrograph of skin showing DLE, presenting with follicular plugging, basal layer vacuolation, pigmentary incontinence, dermoepidermal junctional infiltrate, dermal lymphocytic infiltrate and patchy perivascular infiltrate (H&E x 40)**

**Table No.1: Description of different types of diseases (n = 97)**

| Disease | Frequency | %tage |
|---|---|---|
| **Lymphocyte Rich IFD (n = 67)** | | |
| Lichen Planus | 40 | 59.6 |
| Lichen Striatus | 7 | 10.4 |
| Lichen Nitidus | 6 | 9 |
| DLE | 6 | 9 |
| PLEVA | 4 | 6 |
| Lichenoid Drug Reaction | 3 | 4.5 |
| Lichenoid Graft Vs Host Disease | 1 | 1.5 |
| **Lymphocyte Poor IFD (n = 21)** | | |
| Erythema Multiforme | 10 | 47.6 |
| PLC | 9 | 42.9 |
| Autoimmune Connective Tissue Skin Diseases | 2 | 9.5 |
| **IFD Granulocytic Blistering Disorder (n = 9)** | | |
| Bullous Pemphigoid | 5 | 55.6 |
| Dermatitis Herpetiformis | 1 | 11.1 |
| Epidermolysis Bullosa Acquista | 1 | 11.1 |
| Linear IGA Disease | 1 | 11.1 |
| Porphyrias | 1 | 11.1 |

Hyperkeratosis, hypergranulosis, acanthosis, and basal cell vacuolation were found to be the most common changes in the epidermis whereas pigmentary incontinence, band like infiltrate at the dermoepidermal junction and perivascular inflammatory infiltrate were the most common dermal features (figure No.2)

# DISCUSSION

The range of age of patients in the present study was 1-85 years, however the majority of cases belonged in the range of 31 years to 40 years (20.6%) followed by 21-30 (18.9%) and 41-50 years which are consistent with the results of another study conducted by [9]., 2013. Our results are in conflict with a couple of studies in which majority of the cases belonged to fifth decade of life [10].

With regards to the gender distribution, most of the cases in our present study showed male dominance with 74.1% and female patients being 25.9%, the ratio of male to female was 3: 1. These findings differ from the study conducted by Hedge and Khadikar, 2014 showing female preponderance with (57.6%) among all cases of Interface dermatitis with the male and female ratio was 1:1.3.[11]

In the study under discussion, the most frequent clinical features were itching, papule and plaque formation, hyper and hypopigmentation, scaling, lichenification and bullae formation. These findings are also corroborated by other studies.[12]

The most consistent and uniform histological finding was basal cell vacuolation followed by lymphocytic inflammatory infiltrate, pigmentry incontinence, band-like infiltrate at the dermoepidermal junction. There was a consistent presentation of Civatte bodies. These findings are in harmony with the study carried out by Alsaad[2]. The most persistent secondary epidermal changes seen in addition to the primary histopathological features comprised of hyperkeratosis, acanthosis, parakaratosis, papillomatosis, spongiosis, hypergranulosis and follicular plugging whereas the least observed features were sub-epidermal blisters, Max-Joseph spaces and horn cyst.

This study, subdivides the cases diagnosed with IFD into three categories; cell rich; cell poor IFD and blistering disorders showing IFD. This study is supported by the classification presented by Crowson who classified IFD on the basis of cell poor and cell rich inflammatory process or type of cellular infiltrate [8].

Amongst ninety seven cases that were confirmed histopathologically as IFD, forty were of the Lichen planus type. The most frequent sub group was cell rich IFD (57.8%) followed by cell poor IFD (18.1%) and granulocyte blistering disorders showing interface dermatitis (7.8%). These results were parallel with a study concluding that the most reliable tool for the identification of underlying cause in lichenoid reactions was histopathological examination with the most common prototype being Lichen planus [10].

Dense inflammatory infiltrate characterises cell rich IFD. Basal cell vacuolation, making unclear the dermal/

epidermal junction by a band like inflammatory infiltrate, pigmentary incontinence and dense perivascular and interstitial lymphocytic infiltrate. Lichen planus and its variants are the prototype of this group according to Sehgal [3]. In the present study, amongst the cases labeled as cell rich IFD, lichen planus (59.7%) was found to be the most prevalent type of disease followed by lichen striatus (10.4%), lichen nitidus (9%), DLE (9%), PLEVA (6%), LDR (4.5%) and one case of graft versus host disease.

The prototypic cell-poor interface dermatitis is erythema multiform. Cell poor interface dermatitis shows basal cell vacuolation accompanied by sparse superficial perivascular infiltration of inflammatory cells. In the epidermis Lymphocytic exocytosis is found with civatte bodies [8]. In the present study 47.6% cases of cell poor interface dermatitis, were of erythema multiforme followed by 42.9% cases of pityriasis lichenoid chronica and two cases of connective tissue disease. The common histopathological features (cell poor IFD) seen in epidermis are apoptotic keratinocytes, spongioses, basal cell vacuolation and rarely epidermal necrosis along with subepidermal clefts. In the dermis lymphohistiocytic infiltrate along with scattered eosinophils and few neutrophils are seen.

Sub-epidermal blistering disorders with interface dermatitis are characterised by clefting at the dermoepidermal junction leading to subepidermal blisters in the skin. One of the commonest blistering autoimmune skin disorders is bullous pemphigoid as shown in a study of Bernard and Charneux, 2011.[13] The present study is in agreement with these findings showing bullous pemphigoid as the most frequent blistering disorder (55.6%) followed by each case of dermatitis herpetiformis, epidermolysis bullosa acquista and linear IgA disease exhibiting basal cell degeneration, mixed inflammatory infiltrate comprising of neutrophils and eosinophils in the dermis and basket weave pattern in the epidermis.

# CONCLUSION

Interface dermatitis is a diverse clinicopathological entity affecting the basal cells, the papillary dermis and the dermo-epidermal junction. Lichen planus was found to be the most prevalent variant of interface dermatitis. The clinical correlation should be considered with all the specimens submitted for histopathological examination as the diagnostic accuracy is increased by correlating clinical and histopathological findings enabling accurate diagnosis leading to better prognosis and patient care. The clinical and histopathological disparity was seen maximum in cell rich IFD (lichen planus) and minimally in granulocyte blistering disorders showing IFD. Thus clinicopathological correlation is more required in cases diagnosed with lichen planus.

**Author's Contribution:**

| | |
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| Concept & Design of Study: | Momina Khadija Abbasi |
| Drafting: | Shameela Majeed, Nabeela Naeem |
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| Revisiting Critically: | Momina Khadija Abbasi, Shameela Majeed |
| Final Approval of version: | Momina Khadija Abbasi |

**Conflict of Interest:** The study has no conflict of interest to declare by any author.

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