

CLINICOPATHOLOGICAL STUDY ON INTERFACE DERMATITIS

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Abstract

Background: Diagnosis of skin disorders that exhibit interface dermatitis is challenging in few scenarios. Despite sincere efforts, clinical examination per se can help us reach a handsome of differential diagnosis but not a gunshot specific diagnosis. Dermatopathology acts as a saviour to clinicians in such cases. Though histopathology is the gold standard, still one cannot make a “specific” diagnosis by histopathology alone because many have overlapping features. Therefore, the present study aims at the importance of clinicopathological correlation. The objectives were to study the clinical and histopathological features of various dermatoses, which exhibit interface dermatitis histopathologically and estimate clinicopathological concordance. **Materials and Methods:** This was a cross-sectional study done on 50 patients attending outpatient department (OPD), with the lesions suggestive of dermatoses known to exhibit interface dermatitis histologically. Strobe guidelines were followed. After a thorough clinical examination, punch biopsies were done and observed microscopically to detect interface dermatitis, if present. Secondary pathological features were studied to assess clinicopathological concordance. Microsoft excel and statistical package for the social sciences (SPSS) 21.0 were used for data analysis. **Result:** 76.74% cases were cases of lichen planus and its variants. The most common clinical presentation was papules. Among microscopic features, predominant finding was basal cell vacuolar change in epidermis (97.70% cases). Clinicopathological concordance was seen in 43 cases (83%). 7 cases were diagnosed solely based on histological correlation. **Conclusion:** A myriad of dermatoses exhibit interface dermatitis as a primary pathological feature. Only an apt correlation of clinical features with secondary pathological features can lead to a specific diagnosis from a bunch of differential diagnoses.

INTRODUCTION

Interface dermatitis is the common terminology that we come across in day-to-day practice of both dermatology and pathology. The term “interface” in skin refers to the lowermost layer of epidermis i.e. stratum basalis, the dermo-epidermal junction, the underlying papillary dermis, and adventitial dermis around the adnexae.^[1] Dermatologic disorders, with their pathology revolving around this interface, are many with myriad of clinical presentations. Lichen planus (LP), lichenoid drug eruptions (LDE), fixed drug eruptions (FDE), erythema multiforme (EM), lupus erythematosus (LE), dermatomyositis (DM), graft versus host disease (GVHD), lichen striatus and pityriasis lichenoides are considered major interface dermatoses. Other commonly encountered entities like morbilliform drug reactions and viral eruptions and radiotherapy/chemotherapy induced

dermatitis also exhibit primary interface dermatitis. It is not only limited to inflammatory dermatoses but can also be evident in infective and neoplastic conditions.^[1]

Damage to the basal layer of epidermis is the signature finding to label a particular pathological picture as “interface dermatitis” as it is universally found in all interface dermatoses.^[1] Interface dermatitis can be cell rich (lichenoid) or a cell poor (vacuolar) based upon the intensity of inflammation at interface. Cell rich category includes lichen planus with its variants and cell poor category includes conditions such as erythema multiforme, pityriasis lichenoides, and autoimmune connective tissue disorders.^[2] The incidence of lichen planus (prototype of lichenoid interface dermatoses) is 0.38% in India. It presents as purple, pruritic, polygonal, planar papules and plaques. Commonly encountered clinical mimics of lichen planus are

drug eruptions, prurigo nodularis, lichenoid variant of sarcoidosis, polymorphous light eruption (PMLE), guttate psoriasis, granuloma annulare, lichen simplex chronicus, and porokeratosis.^[3] Likewise, lupus erythematosus, FDE, and EM have many differential diagnoses making specific clinical diagnosis difficult.

From the perspective of the pathologist also, the other histological features of all the lesions under the spectrum of interface dermatitis overlap each other showing very minute difference in each of them. The primary pathological feature that is, basal cell damage is common and universal in all of them. Secondary changes of the epidermis and papillary dermis along with distribution, density and type of inflammatory infiltrate are used for reaching a specific diagnosis of the various diseases that exhibit the same interface changes.^[1]

Therefore, clinicopathological correlation remains an indispensable tool for precise diagnosis of interface dermatitis. Specific diagnosis also helps in predicting the course of the eruption and planning optimal management.

Hence with the aim of focussing the importance of clinicopathological correlation, the objectives of the study were to study the clinical and histopathological features of various dermatoses, which exhibit interface dermatitis histopathologically and also estimate clinicopathological concordance.

MATERIALS AND METHODS

A hospital based observational cross-sectional study was carried out in the department of dermatology, venereology, and leprosy and department of pathology of Government General Hospital and Kurnool Medical College, Kurnool using Strobe guidelines conducted on 50 patients during 2019-2021.

Source of Data: Patients presenting to OPD with the lesions suggestive of dermatoses, known to exhibit interface dermatitis histologically as a primary feature (lichen planus and its variants, lichenoid drug eruptions, EMF, DLE, DM, vitiligo, and trachyonychia).

Sample Size Calculation: As it is a qualitative data, where proportions and percentages are used to finally estimate percentage of correlation between 2 independent variables, the sample size was calculated using the formula given below where N =sample size, p =positive character (correlation % obtained in previous studies), $q=1-p$, l =allowable error (10% of p). $N=4pq/l^2$

Previous studies' average correlation % was 89, hence my sample size is 49.43, so 50 is chosen.

Inclusion Criteria

Patients aged between 10-60 years who are willing for study and are diagnosed with fresh lesions suggestive of interface dermatitis for which treatment is not yet started.

Exclusion Criteria

Patients who are not willing for the procedure and are already on treatment for the existing lesions.

Ethical clearance was obtained from the institutional ethics committee prior to the commencement of study. Patients who fulfilled the selection criteria were briefed about the nature of the study and informed consent was obtained.

Data Collection

First a detailed history was taken from the subjects regarding the onset, progression and duration of lesions, associated symptoms, aggravating or precipitating factors, past history, treatment history, personal and family history. Then they were subjected to a thorough examination from head to toe, mucosae, nails, palms, soles and scalp for inspection and palpation of lesions (appropriate signs also elicited). Then a bunch of differential diagnosis was noted. Then the patient was subjected to punch biopsy from the fresh lesion under local anaesthesia. Then it was sent to Pathology lab for processing, fixation and staining by haematoxylin and eosin for histopathological examination. All the clinical features and histopathological features were noted and entered in the proforma. The frequencies and percentages of all variables were calculated and finally clinicopathological correlation was assessed.

Statistical Analysis

All the clinical features and histopathological features thus obtained were tabulated and subjected for descriptive statistical analysis. Results on continuous measurements were presented as mean±standard deviation (SD) (max – min). Results on categorical measurements were presented as frequencies and percentages for comparison with other studies. Statistical software- SPSS 21.0 was used for the analysis of the data and Microsoft word and excel was used to generate graphs and tables.

RESULTS

Of 50 cases in study population, 43 exhibited interface dermatitis as a primary feature histopathologically and 7 cases which were clinically suspected to fit in dermatoses known to exhibit interface dermatitis did not show signs of same. Hence clinical and histopathological features of 43 cases were studied in detail.

The most common age group affected was 10-40 years with mean age of 33.12 ± 15.15 years [Figure 1].

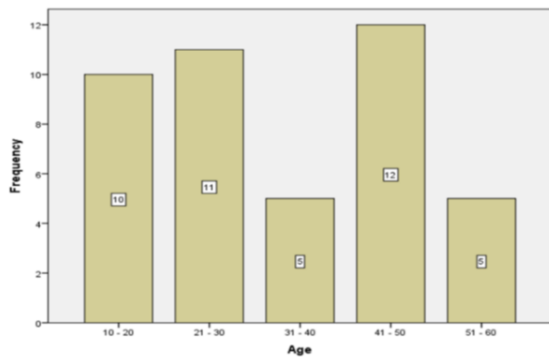


Figure 1: Age distribution of study population.

Male predominance was seen with a ratio of 2.07:1 (67.4% versus 32.6%) as represented in [Figure 2].

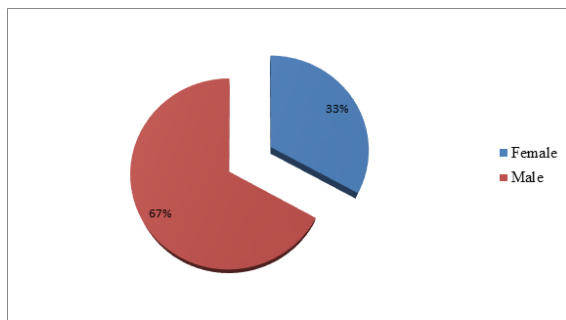


Figure 2: Distribution of patients according to gender.

Most of the patients presented with the pruritic skin lesions (62.80%), followed by asymptomatic skin lesions (18.60%) and hair loss (16%). Mean duration of the lesions was 7.09 ± 9.06 months. 7 cases (14%) gave a history of drug intake prior to the onset of lesions. 6 used NSAIDs and one patient used doxycycline. One case gave a history of herpes labialis prior to the onset of lesions.

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Majority of the cases had violaceous lesions (62.8%) and were distributed over legs (21 of 43 cases- 49%) followed by trunk (37.21%), flexors of upper limbs (30.23%), scalp (18%), face (6%), palms (4.6%). 58.1% cases, the lesions were symmetrical. 14% cases had oral lesions- violaceous hue with reticulate pattern over buccal mucosa and were asymptomatic. 2 cases had nail involvement with longitudinal ridging and 1 case had trachyonychia with pterygium. Genitalia was involved in 2 cases.

Most common primary lesions were papules followed by plaques, patch, vesicle/bulla and target lesions. Wickham striae were seen in 39.50% cases and koebnerisation in 34.90% cases.

Based on clinicopathological correlation, the following diagnoses were made as illustrated in [Table 1].

Histopathological features across the spectrum of interface dermatoses were as mentioned in the [Tables 2 and 3].

The primary lesions in different interface dermatoses were analysed as follows- all classical lichen planus lesions were papules (100%). All hypertrophic lichen planus lesions showed hypertrophic plaques. All LP pigmentosus cases presented as slate grey patches. Two third of the lichen planopilaris cases exhibited atrophic cicatricial plaques over scalp, one third had cicatricial patches with violaceous perifollicular papules. Linear lichen planus cases exhibited papules and plaques. Actinic lichen planus lesions were violaceous plaques over face. Genital lichen planus case had hyperpigmented to violaceous papules. Case of nail lichen planus had no cutaneous lesions, exhibited only nail changes of trachyonychia and dorsal pterygium. A case of lichenoid drug eruption presented as erythematous macules over back, violet papules and plaques over extremities. Lichen nitidus showed tiny shiny discrete papules. A case of inflammatory vitiligo had depigmented patches and 1 atrophic plaque with loss of hair but intact sensations. 2 cases (100%) of fixed drug eruption had hyperpigmented patches, 1 of them had bullous lesions and erosions over trunk and genitalia. All cases of DLE exhibited atrophic hypopigmented cicatricial plaques over scalp. A case of erythema multiforme minor presented with dark red plaque and typical target lesions over palms with central dusky red hue and pale oedematous ring.

Finally, as illustrated in [Table 4], clinicopathological concordance was 86%, with correlation coefficient of +0.984801 as depicted in [Figure 3].

43 cases showed interface dermatitis on histopathology and 7 cases had no significant changes at interface. Of 43 cases, we were able to confirm 7 cases solely on the basis of histopathologic correlation. We were able to diagnose inflammatory vitiligo from its other differentials like lichen sclerosus and indeterminate hansen's, genital lichen planus from bowenoid papulosis and nail LP from other causes of trachyonychia, 1 classical lichen planus from Nekams disease, lichenoid drug eruption from guttate psoriasis, another classical lichen planus from prurigo nodularis, lichen nitidus from lichen spinulosus and follicular eczema.

List of discordant cases

Erythema dyschromicum perstans, actinic lichen planus, lupus panniculitis, verruca/hypertrophic LP, nevus/lichen planus pigmentosus, inflammatory verrucous nevus/hypertrophic LP, bullous pemphigoid/lichen planus pemphigoides.

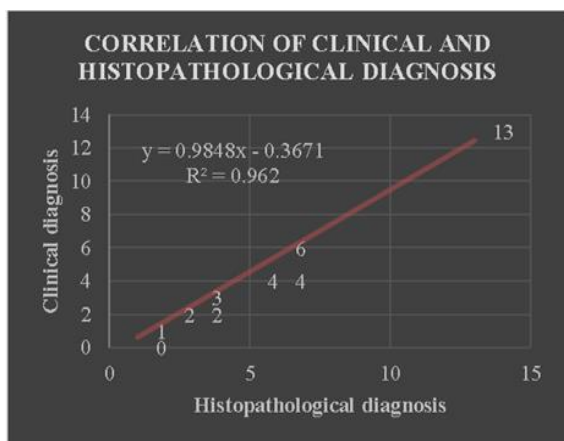


Figure 3: Correlation between clinical and histopathological diagnosis.

Table 1: List of diagnosis based on clinicopathologic correlation.

Diagnosis	Frequency	Percentage
LP and its variants		
Classical lichen planus	13	30.2
Hypertrophic lichen planus	4	9.3
Lichen planus pigmentosus	4	9.3
Lichen planopilaris	6	14.0
Linear lichen planus	2	4.7
Actinic lichen planus	2	4.7
Genital lichen planus	1	2.3
Nail lichen planus	1	2.3
Lichenoid drug eruptions	1	2.3
Lichen nitidus	1	2.3
Vitiligo	1	2.3
Fixed drug eruption	3	7.0
Discoïd lupus erythematosus	3	7.0
Erythema multiforme	1	2.3

Table 2: Epidermal changes across the spectrum of interface dermatoses.

Epidermis	Frequency	Percentage
Hyperkeratosis	31	72.10
Parakeratosis	3	7
Hypergranulosis	29	67.44
Acanthosis	29	67.44
Atrophy	6	14
Basal cell vacuolation	42	97.70
Civatte bodies	19	44.20
Saw toothed rete ridges	18	41.90
Follicular plugging	9	20.93

Table 3: Dermal changes across the spectrum of interface dermatoses.

Dermis	Frequency	Percentage
Inflammation at DEJ	36	83.72
Perifollicular inflammation	9	20.90
Perivascular lymphocyte cuffing	25	58.13
Pigment incontinence	39	90.70
Subepidermal bulla	1	2.30

Table 4: Clinicopathologic correlation.

Concordance with interface dermatitis	Frequency	Percentage
Concordance	43	86
Disconcordance	7	14

Table 5: Comparison of histopathological features.

S. no	Features	Present study (%)	Ravikant et al8 (%)	Banushree et al17 (%)	Kumar et al5 (%)
1	Hyperkeratosis	72.10	71.21	80	93.33
2	Parakeratosis	7	16.66	5	6.66
3	Hypergranulosis	67.44	65.15	-	-
4	Acanthosis	67.44	60.60	73.33	83.33
5	Loss of rete ridges	41.90	6.06	33.33	60
6	Civatte bodies	44.20	25.75	80	21.11
7	Vacuolar basal cell	97.70	74.24	83	96.66

	degeneration				
8	Follicular plugging	23.30	7.57	5	13.33
9	Inflammatory infiltrate over DEJ	83.72	48.48	96.6	93.33
10	Melanin incontinence	90.70	63.63	93	93.33
11	Perivascular inflammatory infiltrate	58.13	60.66	-	-
12	Periadnexal inflammatory infiltrate	20.90	36.36	-	-
13	Subepidermal bulla	2.30	1.51		

Table 6: Frequency of types of interface dermatitis (ID) according to Le Boit groups.

Type of ID and clinical condition	No. of cases	Percentage
I		
Erythema multiforme	1	2.3
Fixed drug eruption	3	7
II		
LP and its variants	33	76.74
Lichenoid drug eruptions	1	2.3
Discoid lupus erythematosus	3	7
III		
Hypertrophic Lichen planus	1	9.3
IV		
	-	-
V		
Discoid lupus erythematosus	3	7
Lichen plano pilaris	1	2.3
Vitiligo	1	2.3

Table 7: Comparison of clinicopathological concordance with other studies.

Study	Total cases	Cases concordant	Cases discordant
Dhar et al10	104	82	22
Sarin et al6	50	40	10
Dixit et al14	166	148	18
Kumar et al5	107	84	23
Manjunatha et al7	90	83	7
Present study	50	43	7

DISCUSSION

In this study, most patients belonged to the age group of 10-40 years (60.5%). This is comparable with the findings of Sehgal et al (11-40 years) and Kumar et al (1-30 years), whereas study done by Sarin et al, reported 8-50 years as the most affected age group and Manjunatha et al as 30- 60 years age group.^[4-7] This wide variation is because of multiple different subentities, which come under the same umbrella term interface dermatoses.

In the present study, males outnumbered females in the ratio of 2.07, 29 (67.44%) were males and 14 (32.56%) were females. In the subcategory of lichen planus also, males are affected predominantly with a ratio 1.54:1; i.e. 60.60% males and 39.40% females. This is comparable to the studies done on interface dermatitis by Sarin et al and Chauhan et al which showed predilection for males by 54% and 53% respectively.^[6,8] Female preponderance was noted in the studies done by Kumar et al (57.78%), Pawar et al (59.9%), Dhar et al (58.60%) and Hegde et al (57.6%).^[5,9-11] Studies on lichen planus by Kumar et al showed a ratio of 60% males and 40% females, Kachhawa et al with 58.6% males and 41.3% females.^[12,13]

The chief complaints in our study group were pruritic skin lesions in majority – 27 of 43 cases (62.80%) followed by alopecia in 7 cases (16.30%).

This is comparable to study done by Manjunatha et al where pruritus was predominant symptom in 40% cases and Dixit et al where itching was seen in 94.59% cases.^[7,14] 22 of 33 cases (66.67%) of lichen planus and its variants had pruritic skin lesions. All cases of classical lichen planus and hypertrophic lichen planus were having itchy skin lesions. This is in concordance with study done by Sehgal and Rage et al with 85.91% cases presented with itchy skin lesions and Kachhawa et al with 72.8% symptomatic cases.^{4,13} Kumar et al reported 92% cases of lichen planus cases with pruritus.^[12]

Of 43 cases in present study, 7 cases (16.28%) gave a history of drug intake (6 took NSAIDs and 1 used doxycycline). Dixit et al reported 4.06% cases of interface dermatitis were using bronchodilators/oral contraceptives/antiepileptics.^[14] Manjunatha et al also reported 3.33% cases with positive history of drug intake.^[7]

In this study, the most common site involved was legs (49%). This is similar to studies done by Dixit et al, Khaled et al, and Parihar et al.^[14-16]

Spectrum of diseases under interface dermatitis

In our study, majority (76.8%) of the cases were of lichen planus and its variants, followed by DLE and FDE, then lichen nitidus, lichenoid drug eruptions, erythema multiforme and inflammatory vitiligo. Among LP and its variants- classical lichen planus was the most common. This is in concordance with

the studies done by Kumar et al, Chauhan et al and Dixit et al.^[5,8,14] Lichen planopilaris was the second most common LP variant in our study followed by lichen planus pigmentosus and hypertrophic LP with equal frequency. But other studies like Chauhan et al and Banushree et al reported lichen planus pigmentosus as the second most common LP variant.^[8,17]

Classical lichen planus

In this study, 30.62% cases were classical lichen planus. All cases presented as violaceous pruritic papules with Wickham striae and koebnerisation in 84.61%. Most common sites involved were flexors of upper limbs, trunk and legs. 1 case had oral lesions in the form of violaceous reticulate pattern [Figure 4 and 5].



Figure 4: Violaceous flat-topped papules (a) over flexor aspects of forearms, and (b) plaques over extensor aspects of legs.

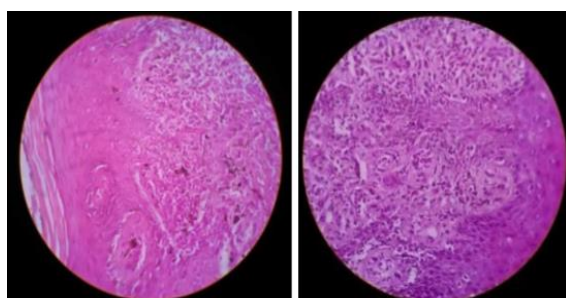


Figure 5: (a) Histopathology of classical lichen planus with basal cell vacuolation and melanin incontinence; and (b) lymphocytic infiltrate at dermo epidermal junction in a case of classical lichen planus.

Histopathology

Epidermis

All cases exhibited hypergranulosis and acanthosis along with basal cell vacuolation. 12 of 13 cases (92.31%) had hyperkeratosis and 11 of 13 cases (84.62%) cases had civatte bodies and saw-toothed rete ridges on histopathological examination.

Dermis

All cases showed lymphocytic infiltrate at dermo-epidermal junction. 5 of 13 cases (38.46%) had mild and 8 of 13 cases (61.54%) had moderate intensity of inflammation. Melanin incontinence was seen in all cases and perivascular lymphocyte cuffing in 6 of 13 cases (46.15%).

Thus, the clinicopathological features can be correlated as follows-Wickham striae are because of underlying hypergranulosis, orthokeratosis

(hypergranulosis without parakeratosis) and acanthosis explain the appearance of flat-topped papules. Epidermal basal cell damage, being the characteristic feature of interface dermatoses is seen in all cases in the form of basal cell vacuolation. This vacuolar change is seen in lichenoid (as in above cases) interface dermatitis apart from vacuolar group of interface dermatitis. Filamentous degeneration of basal cells is represented by civatte bodies, which is also responsible for pigment incontinence (released from destructed basal cells). This is correlated by the number of cases showing civatte bodies and pigment incontinence in the above study. Saw toothing of rete ridges can be explained by the intense infiltrate that is obscuring the normal rete ridge pattern.

Hypertrophic lichen planus

9.3% of all cases and 12.12% of lichen planus cases were hypertrophic LP. All cases presented as pruritic plaques, most commonly over legs. Oral cavity was involved in 75% cases. Wickham striae was noted in all cases and 1 case also had Koebner's phenomenon.

Histopathology

All cases exhibited band like lymphocytic infiltrate at DEJ with pigment incontinence. All cases exhibiting saw tooth rete ridges can be explained by band like inflammatory infiltrate in all the cases, which is characteristic of hypertrophic lichen planus as described by Attili.¹ The same intense infiltrate is responsible in 1 case for obscuring the visualization of basal cell vacuolation.

Lichen planopilaris

Histopathology

All cases had follicular plugging and perifollicular inflammation. 5 of 6 cases had pigment incontinence. These findings of perifollicular infiltrates are in concordance with other studies done by Kumar et al, Dhar et al and Dixit et al.^{5,10,14} This in turn explains the scarring alopecia seen in lichen plano pilaris [Figures 6 and 7].



Figure 6: Violaceous plaque over scalp with cicatricial alopecia in a case of lichen planopilaris

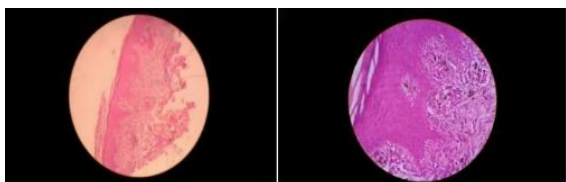


Figure 7: (a) Perifollicular inflammation in a case of lichen planopilaris, and (b) pigment incontinence with MaxJoseph space in a case of lichen planopilaris.



Figure 8: A case of nail lichen planus with trachyonychia. Lichenoid drug eruptions

Genital lichen planus.

Clinically presented as itchy, hyperpigmented to violaceous flat-topped papules around genitalia and groin with violaceous hue and lacy pattern in buccal mucosa and longitudinal ridging over nails. Differentials were bowenoid papulosis and lichen sclerosus et atrophicus, which could only be differentiated on histopathology. On microscopy- it exhibited hyperkeratosis, parakeratosis, hypergranulosis, acanthosis, basal cell vacuolation, Civatte bodies and saw toothing of retes. Also had inflammation at DEJ and perivascular lymphocytic infiltrate. Thus, stressing the necessity of histological correlation.

Similarly, the diagnosis of nail lichen planus [Figure 8] was confirmed as the underlying cause of trachyonychia based only on classical histopathological findings.

There was 1 case in the present study (2.3%). Kumar et al reported 3 cases (3.33%) of LDE and Manjunatha et al reported 1 case (1.1%) in their studies.^{5,7} Absence of elongated retes, Munro's microabscesses and presence of granular layer with interface dermatitis helped in ruling out psoriasis in one case.

Vitiligo

A 25 years male was diagnosed outside as vitiligo solely based on histopathology. He had for 1 atrophic patch with loss of hair but intact sensations along with multiple hypopigmented patches, hence there was necessity of histopathology to rule out

lichen sclerosus and Hansens. Later, after few months when patient approached GGH, Kurnool in view of phototherapy, he had full blown vitiliginous depigmented macules and patches. Microscopy of his initial lesions revealed atrophy, basal cell vacuolation and perivascular lymphocyte infiltrate with features consistent of early vitiligo with focal inflammatory change. This scenario explains the early lichenoid infiltrates that are responsible for the loss of melanocytes and late destructive phase with loss of even appendages also as described by Attili et al, Sharquie et al, Hann et al, Montes et al and Gokhale et al.^[18-22]

Fixed drug eruption

2 of 3 cases had basal cell vacuolation along with pigment incontinence and perivascular lymphocyte infiltrate consistent with changes of late lesions without any specific diagnostic finding. But 1 case which presented as bullous FDE had subepidermal bulla with epidermal necrosis. These findings are consistent with description mentioned by Joshi et al.^[23]

Discoid lupus erythematosus

It constituted 7% of all cases. Manjunatha et al reported 13.3% cases in their study and 0.68% of cases in a study done by Dixit et al.^{7,14} In a study by Kumar et al, lupus erythematosus constituted 9% cases.⁵ All 3 cases presented as atrophic hypopigmented plaques of cicatricial alopecia over scalp, forearms and neck.

On histopathology- all cases had atrophy, basal cell vacuolation and follicular plugging along with mild inflammation at DEJ, perifollicular inflammation and pigment incontinence. Similar findings were seen in a study conducted by Manjunatha et al.⁷ Basement membrane was destroyed, there was no evidence of thickening as such. Basement membrane thickening, (usually present in late stages as mentioned by Attili) is the differentiating feature for DLE as mentioned by Joshi et al.^[1,23]

Follicular plugging with keratin plugs denotes early lesions whereas predominant follicular destruction with fibrosis represents late lesions. As described by Sarin et al, hypopigmentation can be explained by collateral damage of melanocytes as a result of vacuolar alteration of basal layer and atrophic epidermis (no chances of epidermopoiesis) [Figure 9].^[6]

Erythema multiforme

Histopathology

There was orthokeratosis, basal cell vacuolation and pigment incontinence with perivascular infiltrate. Histopathology per se was not specific in our case but showed features of interface dermatitis.

Histopathological features of interface dermatoses in this study were comparable with other studies as represented in the [Table 5]. Some discrepancies may exist between findings of different studies as the spectrum of diagnosis chosen differs and histopathological features vary with the pathophysiology underlying the different dermatoses.



Figure 9: (a) Cicatricial alopecia of scalp with atrophic hypopigmented plaques in a case of discoid lupus erythematosus, and (b) Focal basal vacuolation, pigment incontinence and lymphocytic infiltrate in papillary dermis in a case of DLE.

Frequency of different types of interface dermatitis according to Le Boit groups is depicted in the [Table 6].^[24]

As per [Table 7] clinicopathological concordance was consistent with other studies.

List of discordant cases in the present study

It included cases clinically suspected to have interface dermatitis but no suggestive features on histopathology.

Clinically suspected erythema dyschromicum perstans but no interface changes, clinically suspected actinic LP but no lichenoid changes, LE panniculitis but had only fibrotic changes with no interface dermatitis, suspected hypertrophic LP but turned-out verruca on histopathology, suspected nevus/lichen planus pigmentosus and histopathology ruled out LPP, inflammatory linear verrucous epidermal nevus/hypertrophic LP and microscopy was in favour of ILVEN, and bullous pemphigoid/lichen planus pemphigoides but no microscopic features suggestive of lichenoid interface dermatitis.

As the study was carried out over a limited time period with a limited number of cases, it may not be large enough to be of perfect precision. All the facts and figures may vary considerably from those of large series covering multiple cases throughout the spectrum of interface dermatitis.

CONCLUSION

In interface dermatoses, the combination of clinical acumen and detailed analysis of secondary pathological features plays a pivotal role in reaching a specific diagnosis. Hence stressing upon the paramount importance of clinicopathologic correlation.

Recommendations

Based upon the observations in our study and previous studies, we can suggest all the clinicians that clinicopathological correlation with the aid of dermatopathologists is always recommended, especially when there is a diagnostic dilemma. Such large multicentric studies with large sample size

may be considered in future to strengthen the diagnostic and therapeutic processes.

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