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Original Research Article

Clinicohistopathological study of lichenoid interface dermatitis

Simran Mehra, Shweta Grover*, Vineet Relhan, Anupam Varshney

Department of Pathology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India

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***Correspondence:**
Dr. Shweta Grover,
E-mail: grovershweta75@gmail.com

ABSTRACT

Background: Lichenoid interface dermatitis (LID) represents a heterogeneous group of inflammatory dermatoses characterized by immune-mediated basal keratinocyte injury and interface inflammation, often posing diagnostic challenges due to overlapping clinical and histopathological features. The study was done to evaluate the clinicohistopathological features of LID, to identify the spectrum of clinical and microscopic findings, and to assess histopathological features useful in subtyping variants of lichenoid interface dermatoses.
Methods: This was hospital-based observational study conducted in the Department of Pathology, Muzaffarnagar Medical College and Hospital, from March 2024 to September 2025. A total of 70 skin biopsy specimens clinically suspected and histopathologically diagnosed as LID were included. Biopsies were processed routinely, and 4-5 µm sections were stained with hematoxylin and eosin. Detailed histopathological evaluation was performed and correlated with clinical data. Statistical analysis was carried out using SPSS version 17/20, and results were expressed as percentages.
Results: Out of the total of 70 cases, female preponderance was noted with male to female ratio as 1:1.33. Most patients belonged to the 31-40 years group of which most of the cases were diagnosed as classic lichen planus (44.3%). Most common site reported in the present cohort was lower limbs. Clinico-pathological concordance was seen in 80% of the cases.
Conclusions: Meticulous histopathological examination, coupled with clinical correlation, is essential for precise diagnosis and subtyping, thereby fulfilling the study objectives and reinforcing the central role of histopathology in managing lichenoid interface dermatoses.

Keywords: Lichenoid, Interface, Histopathology

INTRODUCTION

Lichenoid interface dermatitis (LID) represents a group of inflammatory skin diseases that share a common pattern of damage at the junction of the epidermis and dermis. Pinkus first described lichenoid tissue reactions as a set of clinically diverse and relatively uncommon dermatoses that are united by similar microscopic changes in the year 2013.[1] Later, this pattern was termed "interface dermatitis" by other authors, emphasizing that the main pathology is seen at the dermoepidermal junction rather than in the superficial epidermis alone.[2]

Interface dermatitis is broadly divided into two main patterns based on the density of inflammatory cells. The lichenoid or "cell-rich" pattern is typified by classical lichen planus, where dense band of lymphocytes obscures the dermoepidermal junction. The vacuolar or "cell-poor" pattern is seen in diseases such as erythema multiforme, where basal cell damage is evident but the inflammatory infiltrate is relatively sparse.[3] Basic mechanism in many LID conditions is thought to be cell-mediated cytotoxicity directed against basal keratinocytes.[4]

The spectrum of conditions that fall under LID includes classical lichen planus, oral lichen planus, hypertrophic

lichen planus, lichen planopilaris, lichen planus pigmentosus, lichenoid drug eruptions, discoid lupus erythematosus, erythema multiforme and localized entities such as lichenoid keratosis. Each has characteristic clinical features, but many share overlapping signs such as violaceous papules, scaling, or pigmentation.[5] Because of this overlap, histopathology plays a central role in distinguishing between these entities, especially when lesions are atypical or localized to special sites like mucosa, scalp, or genitalia.[6]

The aim of the present study was to evaluate the clinicohistopathological features of LID and to assess histopathological features useful in subtyping variants of lichenoid interface dermatoses.

METHODS

This present study is a hospital-based observational study conducted in the Department of Pathology, Muzaffarnagar Medical College and Hospital, from March 2024 to September 2025. A total of 70 skin biopsies clinically diagnosed as LID and confirmed on histopathological examination falling within the specified period of time is considered under inclusion criteria. Autolyzed/Necrotic tissue specimens were excluded from the study. All biopsy specimens were fixed in 10% buffered formalin and was routinely processed and embedded in paraffin wax. Serial sections of 4-5-micron thickness were taken from each block for hematoxylin and eosin staining (H and E). Further detailed histopathological examination of the samples was conducted and correlated with clinical findings.

Data collection method

Clinical and histopathological analysis data of the LID cases (included according to the inclusion criteria) was entered in Microsoft excel.

Statistical analysis

Results on categorical measurements were tabulated and presented as frequencies and percentages. Statistical software- SPSS version 17/20, Statcal 2 software was used for analysis of the data.

RESULTS

The study included 70 cases, with 30 males and 40 females (M:F ratio 1:1.33). Most patients were in the 31-40 years age group (28.6%), with a mean age of 33 years. Classic lichen planus accounted for 31 cases (44.3%), while the remaining 39 cases (55.7%) were its variants.

In the present cohort, females constituted 40 cases (57.1%), whereas males accounted for 30 cases (42.9%), indicating a modest female predominance. The male-to-female (M:F) ratio was 30:40, (equivalently, M:F=1:1.33).

Biopsy-site categories in the study included lower limb in 26 cases (37.1%), face in 10 cases (14.3%), and abdomen in 10 cases (14.3%). Upper limb sites accounted for 9 cases (12.9%), while scalp contributed 8 cases (11.4%) and neck 5 cases (7.1%). Oral lesions and genital sites were each recorded in 1 case (1.4%). Overall, 70 biopsies (100.0%) were included and lower limb were most commonly involved site in this cohort with progressively fewer samples obtained from cephalic, mucosal and abdominal regions.

Table 1: Distribution of cases according to age group.

Age group (in years)	N (%)
1-10	2 (2.9)
11-20	14 (20.0)
21-30	15 (21.4)
31-40	20 (28.6)
41-50	10 (14.3)
51-60	6 (8.6)
61-70	2 (2.9)
71-80	1 (1.4)

The age distribution showed that most patients belonged to the 31-40 years group (20; 28.6%). This was followed by 21-30 years (15; 21.4%) and 11-20 years (14; 20.0%). The 41-50 years group contributed 10 patients (14.3%), while 51-60 years accounted for 6 patients (8.6%). Very few cases were seen at the extremes of age, with 1-10 years and 61-70 years each having 2 patients (2.9%), and only 1 patient (1.4%) in the 71-80 years group.

Table 2: Distribution of cases by gender.

Gender	N (%)
Male	30 (42.9)
Female	40 (57.1)

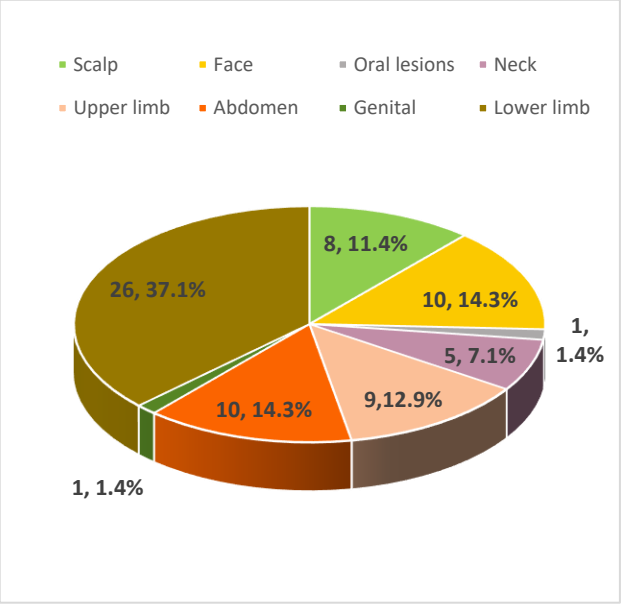

Figure 1: Distribution of biopsy sites by category.

Skin manifestations observed clinically

In the present cohort, lesions in different interface dermatitis were analysed as follows-All classical lichen planus lesions presented as itchy and scaly skin lesions i.e., papules and plaques. Other clinical presentations include slate grey and hyperpigmented macules in Lichen planus pigmentosus and ashy dermatosis. Cicatricial plaques over scalp was noted in lichen planopilaris. Papules coalescing to form linear bands following Blaschko lines was characteristic feature of lichen striatus. Tiny shiny flat-topped skin colored to pinkish papules was noted in lichen nitidus. Erythema multiforme presented with characteristic targetoid lesion whereas thick and scaly plaques was noted in lichen simplex chronicus. Atrophic shiny porcelain white plaques around genital area was seen in lichen sclerous whereas atrophic hypopigmented scarring over scalp was seen in discoid lupus erythematosus. Lichenoid drug eruption presented as erythematous to violaceous macules, papules and plaques.

Among the 70 cases, the histopathological diagnoses included classic lichen planus in 31 cases (44.3%) and lichen planus pigmentosus in 14 cases (20.0%). Hypertrophic lichen planus and lichen planopilaris were recorded in 5 cases each (7.1% each), while lichen simplex chronicus was seen in 4 cases (5.7%). Ashy dermatosis, lichen nitidus, and lichen striatus accounted for 2 cases each (2.9% each). The remaining diagnoses-discoid lupus erythematosus, erythema multiforme, lichen sclerosus et atrophicus, lichenoid drug eruptions, and oral lichen planus-were each noted in 1 case (1.4%) (Table 3).

Among the key histological features (n=70), hyperkeratosis was observed in 91.4%, while basal cell vacuolar degeneration-the principal interface change-was present in 84.3% of cases. Epidermal alterations included acanthosis (62.9%), atrophy (8.6%) and max-joseph spaces in 7.14% cases. These are the clefts formed between the epidermis and dermis due to extensive BM degeneration. Parakeratosis was noted in 4.2% cases only. In the dermis, a predominantly lymphocytic infiltrate was noted in 85.7%, and melanin incontinence in 77.1%. A band-like chronic mononuclear inflammatory infiltrate over the DEJ and perivascular/periadnexal inflammatory infiltrate were each seen in 48.6% of cases. Less common findings included perifollicular inflammatory infiltrate (7.1%), lymphohistiocytic infiltrate in expanded dermal papillae (2.9%), and eosinophils (1.4%) (Table 4).

A total of 70 cases were included in this study. Out of these 30 cases were male and 40 cases were female with a male to female ratio of 1:1.33. Majority of patients were in the age group of 31-40 years (28.6%) with a mean age of 33 years. Of these, majority of cases were of classic lichen planus (i.e., 31 cases; 44.3%) and 39 cases (55.7%) were its variants. Among lichen planus and its variants-classic lichen planus was the most common variant in our study contributing for 31 cases (44.3%). Lichen planus pigmentosus was the second most common LP variant (14 cases; 20.0%) in our study followed by hypertrophic lichen planus and lichen planopilaris with equal frequency (5 cases; 7.1%). Two cases (2.9%) each of lichen striatus, lichen nitidus and ashy dermatosis was diagnosed. Other histopathological diagnosis were lichen simplex chronicus 4 cases (5.7%), one case (1.4%) each of discoid lupus erythematosus, erythema multiforme, lichen sclerosus et atrophicus and lichenoid drug eruptions. Fifty-six cases (80%) were concordant on histopathology. These were classic lichen planus, hypertrophic lichen planus, lichen planopilaris, ashy dermatosis, lichen striatus, lichen nitidus, erythema multiforme, lichen sclerosus and discoid lupus erythematosus. Discordance was seen in fourteen cases including nine (12.8%) cases of classic lichen planus, 3 (4.28%) cases of lichen planus pigmentosus and 2 (2.9%) cases of lichen planopilaris. Hyperkeratosis was noted in 91.4% of the cases. About 4.2% of the cases revealed parakeratosis, a key feature observed in oral lichen planus, lichenoid drug eruptions and discoid lupus erythematosus which helps to differentiate it from classic lichen planus. The 44 cases exhibited acanthosis reflecting epidermal hyperplasia with pointed or saw-toothing of rete ridges was identified in 33 cases (47.1%). Spongiosis was observed in 5 cases (7.1%) of which four cases were diagnosed as lichen simplex chronicus and one case as lichenoid drug eruption. Wedge-shaped hypergranulosis was observed in 31.4% cases of which predominant category was of classic lichen planus. Epidermal atrophy was seen in six cases i.e., five cases (7.1%) of lichen planus pigmentosus and one case (1.4%) of discoid lupus erythematosus, indicating that thinning of epidermis was a rare feature within this study. Max-Joseph spaces are slit-like clefts formed between the epidermis and dermis, resulting from the vacuolated degeneration of the basal epithelial cell layers and was noted in 7.14% of cases in our study. Damage to the basal cell layer with vacuolar degeneration was the most predominant finding contributing about 84.2% (59 cases) along with apoptosis of the basal cells. Civatte bodies accounted for 41.4% (29 cases), predominantly in classic lichen planus. Basal cell damage and melanin incontinence accounted for 77.1% (54 cases) in our study. Circumscribed lymphohistiocytic infiltrate in expanded dermal papillae was seen in lichen nitidus (2.9%). 48.6% cases exhibited band-like chronic mononuclear inflammatory infiltrate at dermo-epidermal junction. Lichenoid drug eruption was seen in one case (1.4%) showed few eosinophils in dermal infiltrate and perivascular lymphocytic infiltrate.

Table 3: Distribution of histopathologic diagnosis of lichenoid skin lesions (n=70).

Histopathological diagnosis	N	Percentage (%)
Classic lichen planus	31	44.3
Lichen planus pigmentosus	14	20

Continued.

Histopathological diagnosis	N	Percentage (%)
Hypertrophic lichen planus	5	7.1
Lichen planopilaris	5	7.1
Lichen simplex chronicus	4	5.7
Ashy dermatosis	2	2.9
Lichen nitidus	2	2.9
Lichen striatus	2	2.9
Discoid lupus erythematosus	1	1.4
Erythema multiforme	1	1.4
Lichen sclerosus et atrophicus	1	1.4
Lichenoid drug eruptions	1	1.4
Oral lichen planus	1	1.4
Total	70	100

Table 4: Key Histological features, (n=70).

Parameters	Percentage
Epidermal + dermo-epidermal junction features	
Hyperkeratosis	91.4
Parakeratosis	4.2
Acanthosis	62.9
Atrophy	8.6
Basal cell vacuolar degeneration	84.3
Max-Joseph space	7.14
Dermal features	
Band like chronic mononuclear inflammatory infiltrate over DEJ	48.6
Melanin incontinence	77.1
Predominantly lymphocytic infiltrate	85.7
Perivascular/ periadnexal inflammatory infiltrate	48.6
Perifollicular inflammatory infiltrate	7.1
Lymphohistiocytic infiltrate in expanded dermal papillae	2.9
Eosinophils	1.4

Table 5: Distribution of epidermal and dermal histopathological features across histopathological diagnoses in lichenoid interface dermatoses, (n=70).

Histopathological diagnosis	HK	Acanthosis	BCVD	Max-Joseph space	CB	MI	Inflammation			
							DEJ	PV/PA	PF	Expanded dermal papillae
Classic lichen planus (31)	29	31	31	5	28	27	31	14	0	0
Lichen planus pigmentosus (14)	14	0	13	0	0	14	1	6	0	0
Hypertrophic lichen planus (5)	5	5	2	0	1	5	0	1	0	0
Lichen planopilaris (5)	2	0	3	0	0	1	0	3	5	0
Lichen simplex chronicus (4)	4	4	0	0	0	1	0	3	0	0
Ashy dermatosis (2)	2	0	2	0	0	2	0	2	0	0
Lichen nitidus (2)	2	0	2	0	0	1	0	1	0	2
Lichen striatus (2)	2	0	2	0	0	2	0	1	0	0
Discoid lupus erythematosus (1)	1	1	0	0	0	0	0	1	0	0
Erythema multiforme (1)	1	1	1	0	0	0	0	1	0	0
Lichen sclerosus et atrophicus (1)	1	0	1	0	0	0	0	0	0	0
Lichenoid drug eruptions (1)	1	1	1	0	0	0	1	1	0	0
Oral lichen planus (1)	0	1	1	0	0	1	1	0	0	0
Total (70)	64	44	59	5	29	54	34	34	5	2

*Abbreviations: HK=Hyperkeratosis; BCVD=Basal cell vacuolar degeneration; CB=Civatte bodies; MI=Melanin incontinence; DEJ=Dermoepidermal junction; PV/PA=Perivascular/periadnexal infiltrate; PF=Perifollicular infiltrate; Expanded dermal papillae=Lymphohistiocytic infiltrate in expanded dermal papillae

DISCUSSION

The age-wise distribution in the present study showed a clear predominance in the 31-40-year age group accounting for 28.6% of cases, followed by 21-30 years (21.4%) and 11-20 years (20.0%), indicating that LID was most frequently encountered in young to middle-aged adults. Similar age clustering has been reported by Pawar et al, Dhar et al and Manjunatha et al where the majority of cases were concentrated between the third and fifth decades.[4,7,8] In contrast, the present study documented very few cases at the extremes of age, with only 2.9% each in the 1-10 and 61-70-year groups and 1.4% above 70 years, a finding less emphasized in comparator studies and likely reflective of lower biopsy rates and diagnostic suspicion in these age groups rather than true disease rarity.

Gender distribution in the present series showed female predominance with 57.1% females and 42.9% males. This indicates higher prevalence of interface dermatitis in females as compared to males with male-to-female ratio of 1:1.33. This pattern closely aligns with observations by Dhar et al where the ratio of male to females was 1:1.4 whereas Malathi et al where the ratio was 1:1.57 respectively.[4,9]

Clinically, papules were the most frequent manifestation in the present study, observed in 51 cases, predominantly within classic lichen planus and its variants (46 cases), followed by plaques in 16 cases and macules in only three cases, with no pustular lesions identified. This distribution is comparable to findings by Vijayalakshmi et al and Malathi et al where papular and plaque-based presentations dominated, particularly in lichen planus and hypertrophic variants.[9,10] The predominance of papules reinforces lichen planus as the prototype of cell-rich lichenoid reactions. Discoid lupus erythematosus, erythema multiforme, lichen sclerosus et atrophicus, and lichenoid drug eruption were infrequent in the present study, each accounting for isolated cases, mirroring the lower prevalence of these entities reported by Dixit et al, Dhar et al and Chauhan et al.[4,11,12]

The lower limb was the most common biopsy site, accounting for 37.1% of cases, followed by the face and abdomen (14.3% each), upper limb (12.9%), scalp (11.4%), neck (7.1%), and infrequent involvement of oral and genital sites (1.4% each). A similar predilection for extremities, particularly the lower limbs, has been documented by Vijayalakshmi et al and Dhar et al who reported distal lower limb involvement as the most frequent site in interface dermatitis, largely reflecting the distribution of classic lichen planus.[4,10] Batchu et al also observed a higher frequency of limb lesions compared to mucosal sites.[2] In comparison, studies focusing on discoid lupus erythematosus, such as Kamath et al have emphasized facial and scalp involvement, whereas in our study only one case (1.4%) of DLE was reported which exhibited scalp involvement with scarring.[13] However, in our study oral and genital sites were each recorded in one case (i.e., 1.4%) indicating lower frequency. Similar observation was found in study by Maheshwari et al and Chauhan et al.[14,12]

Classic lichen planus shows orthokeratosis, wedge-shaped hypergranulosis, and “saw-toothed” acanthosis. There is basal cell degeneration with Civatte bodies and a dense band-like lymphocytic infiltrate at the dermo-epidermal junction, along with pigment incontinence.[15,16] Clinically, it presents as violaceous papules with Wickham striae due to $CD8^+$ T-cell–mediated damage.[17]

Dixit et al studied 166 cases of lichenoid tissue reactions and found lichen planus constituted over 90% of confirmed diagnoses. Dense lichenoid infiltrates, basal vacuolar degeneration, and pigment incontinence were consistent histologic findings. The study highlighted LP as the dominant entity within the lichenoid spectrum.[11] Histopathologically, classic lichen planus was the most common diagnosis in the present study (44.3%), followed by lichen planus pigmentosus (20.0%), with hypertrophic lichen planus and lichen planopilaris each contributing 7.1%. Similar pattern was observed by Agarwala et al and Pathave et al in their study from Delhi and Mumbai respectively.[18,19]

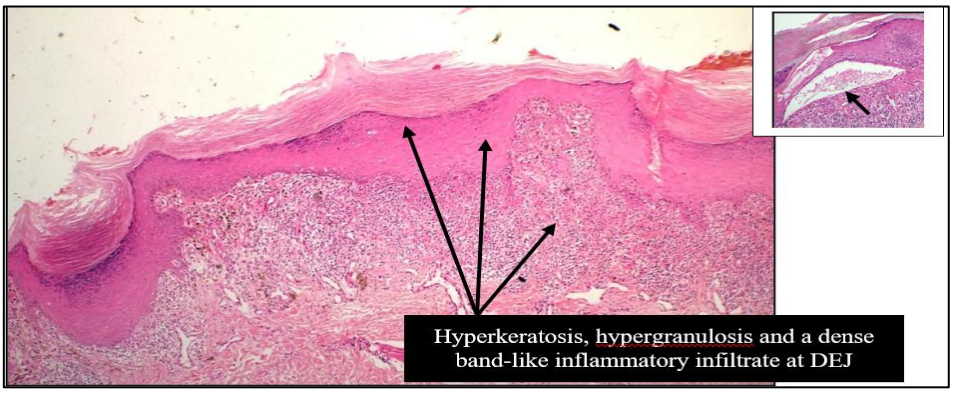

Figure 2: Low power view (40× image, H and E) of LP.

*Hyperkeratosis, wedge-shaped hypergranulosis, irregular rete ridges, dense, band-like lymphocytic infiltrate in superficial dermis and vacuolar alteration of the basal layer. Inlet figure showing Max-Joseph space (arrow), this is an artifactual small subepidermal clefts formed between the epidermis and dermis.

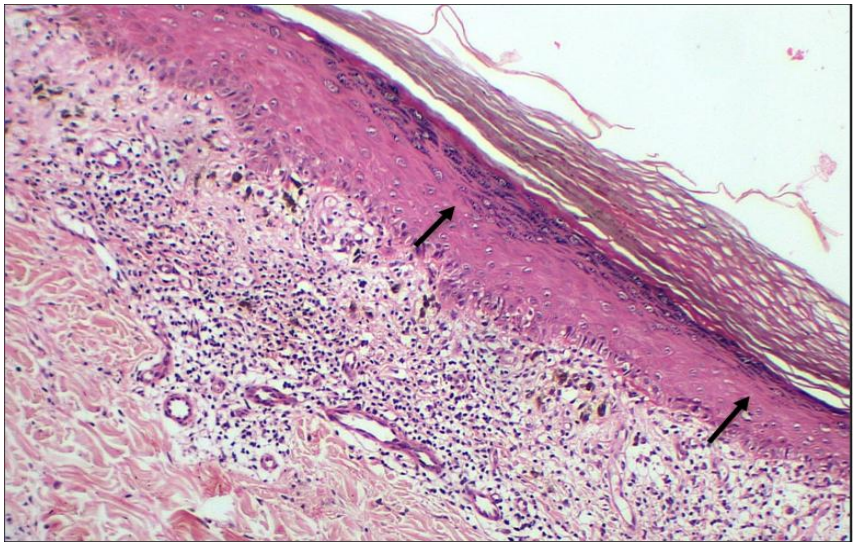

Figure 3: Lichen planus pigmentosus (100×image, H and E) showing epidermal thinning, focal basal cell vacuolar degeneration, marked melanin incontinence (arrow) and mild perivascular infiltrate.

Marked melanin incontinence and abundant dermal melanophages was prominent finding in lichen planus pigmentosus, which was second most common entity in our study. Comparable findings were reported in studies conducted by Parihar et al and Kamyab et al and in case series comprising of 42 cases by Beso et al.[20-22]

Hypertrophic lichen planus was characterised histologically by marked epidermal hyperplasia with vacuolar or lichenoid change accentuated at the bases of elongated rete ridges rather than forming a continuous band. This entity was observed in only 5 cases (7.1%). Approximately similar observations were noted in studies by Batchu et al (9.3%), Muralidhar et al (5.3%), Agarwala et al (3.8%), Sabeena et al (11%), Varsha et al (6.5%) and Poudel et al (4.5%).[2,18,23-25]

Lichen planopilaris in this study was distinguished by perifollicular inflammation in all 5 cases (7.1%) with limited interfollicular epidermal changes, consistent with folliculotropic patterns emphasized by Muralidhar et al (4%), Dhar et al (5.8%) and Batchu et al (14.0%).[2,4,23]

Lichen simplex chronicus accounted for 4 cases (5.7%) in our study which illustrated acanthosis, elongation of rete ridges, lymphocytic exocytosis and vertical streaks of collagen bundles along with perivascular inflammatory infiltrate of lymphocytes. This aligns well with observations of studies reported by Agarwala et al (3.26%) and Manjunatha et al (5.5%), Dhar et al (13.9%) and Malathi et al (14.8%).[4,8,9,18]

Classic "claw clutching the ball" architecture with characteristic circumscribed lymphohistiocytic infiltrate in expanded dermal papillae was observed in our study in both the cases of lichen nitidus. Similar histopathological feature was also noted by various other authors-Chauhan et al, Agarwala et al, Manjunatha et al, Dixit et al, Maheshwari et al, Wankhade et al and Reddy et al.[8,12,11,14,18,27,28]

Lichen striatus showed characteristic alignment of inflammation along adnexal and eccrine structures in both the cases in our study i.e., 2.9%, which was analogous with study by Agarwala et al (2.7%), Dhar et al (1.1%), Manjunatha et al (1.1%) and Maheshwari et al (4.2%).[4,8,14,18] However, Muralidhar et al (0.6%) highlighted fewer cases within broader spectrum of interface dermatitis.[23]

Discoid lupus erythematosus comprises of 1.4% of all cases and presented as atrophic hypopigmented scarring plaque over scalp. Histopathologically, it revealed vacuolar degeneration, follicular plugging, BM thickening and superficial and deep perivascular or periappendageal inflammatory infiltrate of lymphocytes. Studies done by Dixit et al (0.68%), Chauhan et al (4.54%), Batchu et al (7%), Manjunatha et al (8.8%) and Neelima et al (8.93%) noted similar histopathological finding.[2,8,11,12,29]

A case of erythema multiforme (1.4%) presented clinically with characteristic targetoid lesion, while histopathology showed extensive basal cell vacuolation with sparse lymphocytes at DEJ, focal dermal infiltrate and RBC extravasation in dermis was also noted in our study. Similar findings was also reported in study done by Dixit et al (1.35%) and Batchu et al (2%).[2,11]

One case (1.4%) of Lichen sclerosus et atrophicus was noted in our study which was comparable with study done by Dhar et al (1.1%).[4] Histopathology showed thinned out epidermis, densely homogenised collagen in upper dermis along with perivascular lymphocytic infiltrate.

Focal parakeratosis, basal cell vacuolation and necrotic keratinocytes along with presence of eosinophils within lichenoid infiltrate were noted as histopathological

findings in our study in a case (1.4%) of lichenoid drug eruption. Similar findings are also observed by Chauhan R et al (1.5%) and Maheshwari et al (1.7%).[12,14] Only one case (1.4%) of Oral lichen planus was reported in our study exhibiting parakeratosis, acanthosis, saw-toothed rete ridges, basal cell vacuolar degeneration and dense subepithelial band-like inflammatory infiltrate. Similar histopathological findings were noted in studies by Chauhan et al, Agarwala et al, Poudel et al, Osipowicz et al, Manchanda et al and Lodolo M et al.[12,18,26,30-32]

Table 6: Comparison of histopathologic changes.

Parameters Epidermal+dermoepidermal junction features	Present study	Batchu et al[2] (2022)	Muralidhar et al[23] (2020)	Chauhan et al[12] (2015)	Agarwala et al[18] (2024)	Sabeena et al[24] (2025)
Hyperkeratosis	91.4%	72.10%	80.64%	71.21%	37.5%	72%
Acanthosis	62.9%	67.44%	56.45%	60.60%	44.02%	69%
Saw-tooth rete ridges	48.6%	41.90%	-	6.06%	1.63%	52%
Civatte bodies	41.4%	44.20%	41.93%	25.75%	1.63%	33%
Hypergranulosis	31.4%	67.44%	22.58%	65.15%	34.23%	91%
Atrophy	8.6%	14%	1.74%	21.21%	23.91%	8%
Spongiosis	7.1%	-	-	9.09%	9.7%	-
Parakeratosis	4.2%	7%	12.9%	16.66%	14.13%	27%
Basal cell vacuolar degeneration	84.3%	97.70%	95.16%	74.24%	61.41%	100%
Max-Joseph space	7.14%	-	4.83%	3.03%	5.97%	-
Dermal features						
Band like infiltrate over DEJ	48.6 %	83.72%	58.06%	48.48%	27.17%	77%
Melanin incontinence	77.1%	90.70%	90.32%	63.63%	51.08%	64%
Predominantly lymphocytic infiltrate	85.7%	58.13%	100%	72.72%	47.28%	77%
Perivascular/periadnexal inflammatory infiltrate	48.6%	20.90%	50.6%	36.36%	32.60%	39%
Perifollicular inflammatory infiltrate	7.1%	-	4.6%	-	17.39%	11%
Lymphohistiocytic infiltrate in expanded dermal papillae	2.9%	-	4%	27.27%	4.89%	2.7%
Eosinophils	1.4%	-	8.06%	4.54%	-	-

As shown in Table 6, epidermal changes in the present study were dominated by hyperkeratosis i.e., 91.4% which was comparable to study by Batchu et al (72.10%), Muralidhar et al (80.64%), Chauhan et al (71.21%), Sabeena et al (72%), Pathave et al (81.5%) and Varsha et al (78.9%).[2,12,19,23-25]

Acanthosis was reported in 62.9% of the cases which is in concordance with the studies done by Batchu et al (67.44%), Muralidhar et al (56.45%), Chauhan et al (60.60%), Sabeena et al (69%), Pathave et al (74.5%) and Varsha et al (67.1%) respectively.[2,12,19,23-25]

Dense infiltrate that obscures the normal rete ridge configuration can lead to saw-toothing of rete ridges. This feature was observed in 48.6% of the cases in our study. However, study by Batchu et al and Sabeena et al observed it in 41.90% and 52% of the cases which shows conformity.[2,24] Whereas, Chauhan et al and Agarwala et al reported it in fewer cases (6.06% and 1.63%) comparatively.[12,18]

Singly, scattered apoptotic civatte (colloid) bodies were noted in 41.4% of the cases in our study. Parallel findings was also reported by Batchu et al (44.20%), Muralidhar et al (41.93%), Chauhan et al (25.75%) and Sabeena et al (33%) whereas Pathave et al showed high variability which exhibited in 64.1% of cases and Agarwala et al observed it in 1.63% of cases denoting it as less frequent feature in their study.[2,1,18,19,24]

About 31.4% cases exhibited hypergranulosis which was comparable to studies by Maheshwari et al (29%) and Muralidhar et al (22.58%).[14,23] However, studies by Chauhan et al (65.15%), Batchu et al (67.44%), Pathave et al (88%) and Sabeena et al (91%) reported higher number of cases with hypergranulosis respectively.[2,12,19,24]

Atrophy was noted in 8.6% of cases and spongiosis in 7.1% cases which was comparable with findings in study done by Sabeena et al (8%) and Chauhan et al (9.09%).[12,24]

Parakeratosis was reported as most prominent finding in case of oral lichen planus in our study which was analogous to findings of Chauhan et al and Poudel et al.[12,26] Parakeratosis strongly suggests other lichenoid dermatoses, such as lichenoid drug eruption or lichen planus-like keratosis, and is not seen in cases of classic lichen planus.[26] Overall, the present study shows parakeratosis in 4.2% cases only (1 in Oral lichen planus, 1 in lichenoid drug eruption and 1 in discoid lupus erythematosus).

Basal cell vacuolar degeneration, a key feature in Lichen planus, was seen in 84.3% of cases, particularly in classic lichen planus and lichen planus pigmentosus. Similar observation has been reported by Batchu et al (97.70%), Muralidhar et al (95.16%), Chauhan et al (74.24%) and Pathave et al (82.6%), where it was found to be one of the most predominant features.[2,12,19,23]

Max-joseph spaces were observed in minority of the cases (7.14%) in our study as well as in other studies.[12,18,19,23] Upto 18.5% have been identified in other studies.[19]

Dermal findings were characterized by band-like chronic mononuclear inflammatory infiltrate at dermo-epidermal junction in 48.6% of cases in our study, comparable to that of Muralidhar et al (58.06%), Chauhan et al (48.48%) and Maheshwari et al (52%).[12,14,23] Predominantly lymphocytic infiltrate was noted in 85.7% of cases in our study which is comparable to other similar studies.[4,12,23,24]

As a result of basal cell damage, there is "incontinence" of melanin into papillary dermis which accounts for 77.1% of cases in our study, which was found similar to studies reported by Sabeena et al (64%), Chauhan et al (63.63%) and Maheshwari et al (52.99%).[12,14,24] While the number of cases showing pigment incontinence in various other studies reported it over ≥90% and was observed as the most consistent finding.[2,4,10,20,23]

Perivascular and periadnexal infiltrates in mid and deep reticular dermis was found to be 48.6% of cases. This was in accordance with study of Muralidhar et al (50.6%), Chauhan et al (36.36%) and Sabeena et al (39%) respectively.[12,23,24]

Perifollicular inflammation is seen in all cases of lichen planopilaris (5 cases, 7.1%) in our study which was in concordance with other studies done by Vijayalakshmi et al, Muralidhar et al, Parihar et al and Sabeena et al.[10,19,20,24]

All cases (2.9%) of lichen nitidus showed lymphohistiocytic infiltrate in expanded dermal papillae which was similarly noted by various authors.[2,4,12,14,18,23,24]

Lichenoid drug eruptions showed presence of parakeratosis, mild basal vacuolar alteration and eosinophils and sometimes plasma cells in dermal infiltrate while presence of eosinophils (1.4%) was noted as distinct feature in our study. Similar finding was also noted by other authors.[2,4,9,12,23]

Figure 4 (A-I): Clinical photomicrographs of lichenoid interface dermatitis.

Comparative clinicohistological concordance rates ranging from 70% to 85% was noted in various studies accentuating the need of clinical correlation with final histopathological diagnoses.[2,7,10,14,23,33] The rate of concordance is 80% observed in this study. Appropriate clinicopathologic correlation and role of histopathology is helpful in further categorization of various lesions of interface dermatitis.

Despite the high concordance, 20% discordance was observed in this study indicating the cases where clinical diagnoses did not correspond with final histopathological diagnoses. A major source of discordance is the overlap between histopathological features of all the lesions of interface dermatitis which shows very minute difference in each of the variants.

Proper in-depth analysis should be done to avoid misdiagnosis, taking following clinical attributes into consideration while examining the patient. These features include lesion's location, duration and type, drug intake history and any other disease association. Significant number of treatments drop out instances, patients lost to follow up and patients who may not receive appropriate treatment due to over-, mis- or underdiagnosis, will increase the progression of the condition, potentially resulting in worse patient outcomes. The biopsy should be taken from a recent untreated lesion that is morphologically suggestive of clinical diagnosis.

CONCLUSION

Overall, the study fulfills its objectives by establishing the clinicohistopathological profile of LID and identifying key histological parameters essential for accurate classification. These observations emphasize role of histopathology as the cornerstone for diagnosis and subtyping. Appropriate clinicopathological correlation facilitates better clinical management and improving the understanding of disease spectrum within Lichenoid interface dermatoses.

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