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Research Article

STUDY OF KI-67 WITH CLINICOPATHOLOGICAL CORRELATION IN PATIENTS OF PSORIASIS

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ABSTRACT

Objective: To compare expression of IHC Ki-67 in clinical variants of Psoriasis and Non- Psoriatic Psoriasiform Dermatoses (NPPD) and correlate immunohistochemical findings with clinical severity

Methods: This prospective study was conducted over 18 months in the Department of Pathology at Muzaffarnagar Medical College, Uttar Pradesh. A total of 70 skin biopsy specimens with clinical diagnosis of Psoriasis (n=35) and Non-Psoriatic Psoriasiform Dermatoses (n=35) were included. Detailed clinical evaluation, histopathological examination and immunohistochemical staining for Ki-67 were performed. Statistical analysis was carried out using appropriate parametric and non-parametric tests and correlations with PASI score and Body surface area (BSA).

Results: The mean Ki-67 score was significantly higher in the Psoriasis group compared to the NPPD group (74 ± 5.0 vs 43 ± 7.0; $p < 0.001$). Classic histopathological features such as elongation of rete ridges, suprapapillary thinning, parakeratosis, diminished granular layer, vascular dilatation and Munro's microabscesses were significantly more prominent in Psoriasis ($p < 0.05$). Ki-67 expression showed statistically significant correlation with PASI score and BSA involvement in Psoriasis. Strong correlations were observed between multiple histopathological parameters in Psoriasis, reflecting coordinated epidermal and dermal changes.

Conclusion: Ki-67 expression is significantly elevated in Psoriasis compared to NPPD, highlighting its diagnostic utility as an adjunct Immunohistochemical marker. However, its limited correlation with disease severity suggests that Ki-67 should be interpreted in conjunction with clinical and histopathological findings rather than as a standalone severity marker.

KEYWORDS: Psoriasis, Non-Psoriatic Psoriasiform Dermatoses, Ki-67, Immunohistochemistry, PASI, BSA.

Introduction

Psoriasis is a chronic inflammatory disease triggered by the immune system. Psoriasis affects 2-3% of the world's population and its cause is unknown[1]. Studies from Indian continent suggest that the prevalence lies between 0.44% and 2.8%[2]. There is a complex interaction between genetic, immunological and environmental factors[1].

Psoriasis is an immune-mediated condition with a strong genetic basis and involves the skin, nails, and joints, and is often associated with multiple systemic comorbidities[2,3,4].

Psoriatic lesions typically exhibit sharply demarcated borders with silvery-white scales and a smooth, glossy underlying membrane, features that are characteristic for clinical diagnosis. Upon gently removing the scales, a shiny erythematous surface with pinpoint bleeding—resulting from the tearing of the thin suprapapillary epithelium—becomes visible, a finding clinically known as the *Auspitz*

sign. In cases where scaling is not prominent, it can be provoked by lightly scraping the lesion tangentially with a glass slide, a technique referred to as *Grattage*[5].

Psoriasis presents with diverse clinical phenotypes, including plaque, guttate, pustular, and erythrodermic variants. Among these, plaque Psoriasis is the most prevalent, accounting for approximately 80–90% of all cases[6]. These plaques most commonly appear on the scalp, trunk, buttocks, and extremities, although they may develop on any body surface. Nail changes may also occur and can sometimes be present even in the absence of cutaneous plaques. Psoriasis can also manifest through the isomorphic (Koebner) phenomenon, in which new lesions form at sites of skin trauma or injury[4,5].

The pathophysiology of Psoriasis involves a complex interplay between genetic predisposition, environmental triggers, and dysregulated immune responses. Central to this process is aberrant T-cell activation, leading to the overproduction of pro-inflammatory cytokines, particularly tumor necrosis factor-α (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23). Psoriasis is considered a polygenic disease in which genetic susceptibility interacts with environmental triggers to initiate and perpetuate disease expression[7]. Several susceptibility alleles, including HLA-Cw6, HLA-DQ*0201, CCHCR1, and CYP1A1, as* well as multiple genetic loci (PSORS1–9 and PSORSAS1), have been identified and are known to confer an increased predisposition to the development of Psoriasis. Certain medications—such as lithium, interferons, β-blockers, and antimalarials—have been linked to triggering disease onset[7]. Infections, particularly streptococcal pharyngitis and HIV, as well as corticosteroid withdrawal and major psychological stress, are also recognized as important precipitants. While streptococcal infection is strongly associated with the development of guttate Psoriasis, it may also worsen chronic plaque Psoriasis. Seasonal variations influence disease activity, with many patients experiencing improvement during summer and worsening during winter. Additionally, lifestyle factors such as smoking and alcohol consumption have been associated with the condition[7].

Assessment of Psoriasis is based on the degree of skin involvement, typically quantified by the Body surface area (BSA) affected, as well as the intensity of erythema, induration, and scaling. In secondary care settings, standardized tools such as the Psoriasis Area and Severity Index (PASI) and the Physician Global Assessment (PGA) are commonly used. These clinical measures are complemented by patient-reported outcomes, most notably the Dermatology Life Quality Index (DLQI), to evaluate the impact of the disease on quality of life[8].

The hallmark in pathogenesis of Psoriasis is sustained inflammation that leads to uncontrolled keratinocyte proliferation and dysfunctional differentiation[9].

Histologically, Psoriasis is characterized by marked acanthosis with regular elongation and clubbing of the rete ridges. The suprapapillary plates are notably thinned, and small spongiform pustules may occasionally be present. The upper epidermal layers often show pallor, along with a diminished or absent granular layer, prominent confluent parakeratosis and Munro's microabscesses. The dermal papillae contain dilated, tortuous capillaries that extend close to the surface. In addition, mitosis are not limited to the basal layer as in normal skin but are also seen above basal cell layer due to elongation of the rete ridges which results in marked increase in the number of mitoses[10].

Non-Psoriasis psoriasiform dermatoses (NPPD) are conditions that resembles Psoriasis both clinically and histologically. These include Pityriasis rubra pilaris (PRP), Pityriasis rosea, Seborrhoeic dermatitis and Lichen simplex chronicus[11]. Certain features—such as Munro's microabscesses and tortous, dilated capillaries in Psoriasis, alternating orthokeratosis and parakeratosis in PRP, parakeratotic mounds with red cell extravasation in Pityriasis rosea, spongiosis and parakeratosis in Seborrhoeic dermatitis and dermal, thickened vertical collagen bundles with orthokeratosis in Lichen simplex chronicus, provides clues to diagnosis, still many biopsies may yield a nonspecific report of "Psoriasiform dermatitis." This shows the need for more reliable markers to distinguish Psoriasis from NPPD[11].

Ki-67 was first described in 1983 by Gerdes et al. at the University of Kiel, Germany. The term "Ki-67" originates from "Ki" (Kiel) and "67", representing the antibody clone number[12].

Ki-67 antigen is a labile, 345-395 kD non-histone nuclear protein complex. It regulates the cell cycle and is the most widely used proliferation immunohistochemistry (IHC) marker. Its expression has been shown to be increased in psoriatic lesions with respect to non-lesional skin[11].

A key histopathological hallmark of Psoriasis is the marked increase in keratinocyte proliferation. The nuclear antigen Ki67 is a well-established marker of cell proliferation, being expressed during all active phases of the cell cycle (G_1, S, G_2, and M), but absent in quiescent (G_0) cells[11].

Methods

The present study was hospital-based prospective study done after the ethical clearance from institutional ethical committee (Muzaffarnagar Medical College) (MMC/IEC/2024/368) dated: 13.03.2024. The study included 70 clinically diagnosed and histologically confirmed cases of Psoriasis and NPPD. Informed consent was taken from patients who participated in the study. Clinically diagnosed cases with adequate biopsies were included, will inadequate samples, unwilling participants, patients with co-existing dermatological disease and patients already on topical or systemic treatment were excluded. Immunohistochemistry was done.

IHC Interpretation*:* Expression of Ki-67 was indicated

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by the presence of yellow to brown granules in the nucleus. Hotspot were selected and positive cells in epidermis $\text{Ki-67 percentage} = \frac{\text{Suprabasal Ki-67 positive cells}}{\text{Total epidermal Ki-67 positive cells}} \times 100$ [1].

Pasi Score: The severity of psoriatic lesions and the patient's reaction to treatment are evaluated and graded using the PASI, a tool that is frequently used in psoriasis trials.

Statistical Analysis

All data were analysed using STATA version 16. Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables were expressed as frequencies and percentages. Appropriate statistical test including *Student's t-test, Mann-Whitney U test, Chi-square test or Fisher's exact test* and *one-way analysis of variance (ANOVA)* were used based on data distribution assessed by the *Shapiro–Wilk test*. *Pearson's correlation coefficient (r)* was used to assess linear associations and $p\text{-value} < 0.05$ was considered statistically significant for all analyses.

Results

The study analyzed a total 70 clinically suspected cases of Psoriasis(n=35) and NPPD(n=35) over an 18-month period at Muzaffarnagar Medical College, all of which were confirmed histopathologically in this prospective study.

The mean age of participants (Table 1) was higher in the NPPD group compared to the Psoriasis group (41.34 ± 16.78 vs. 35.40 ± 12.84 years). Gender distribution (Table 2) was comparable between the two groups, with a male predominance observed overall.

Lesions in the Psoriasis group (Table 3) showed a more heterogeneous site distribution, with the upper extremities (31.4%) and back (28.6%) being the most common sites, followed by the abdomen (20.0%). Lesions in the chest and scalp were rare (2.9% each) whereas in the NPPD group, lesions were predominantly located in the lower extremities (60.0%), followed by the upper extremities (17.1%), with the abdomen and back each contributing 11.4% of cases.

In histopathology (Table 4) the most common findings in Psoriasis were elongation of rete ridges & regularacanthosis and elongation of dermal papillae (91.4%), followed by confluent parakeratosis, vascular dilation and suprapapillary thinning (85.7%) whereas NPPD had inflammatory infiltrate

Table 1: Age Wise distribution

Age (Years)	Cases Psoriasis (n=35)	Percentage (%)	Cases NPPD (n=35)	Percentage (%)
0-10	01	2.8	00	0
11-20	03	8.6	03	8.6
21-30	10	28.6	06	17.1
31-40	10	28.6	06	17.1
41-50	07	20	09	25.8
51-60	03	8.6	07	20
61-70	01	2.8	02	5.7
71-80	00	0	02	5.7
Age(years), mean ± SD	35.40±12.84		41.34±16.78	

Table 2: Gender distribution

Gender	Frequency Psoriasis (n=35)	Percent (%)	Frequency NPPD (n=35)	Percent (%)
Male	24	68.6	28	80
Female	11	31.4	7	20

Table 3: Site-wise distribution

Site	Cases Psoriasis Percentage %, (N=35)	Cases NPPD Percentage %, (N=35)
Upper extremities	31% (11)	17.1% (06)
Back	28.6% (10)	11.4% (04)
Abdomen	20% (07)	11.4% (04)
Lower extremities	14.% (05)	60% (21)
Scalp	2.9% (01)	0.0(00)
Chest	2.9% (01)	0.0(00)

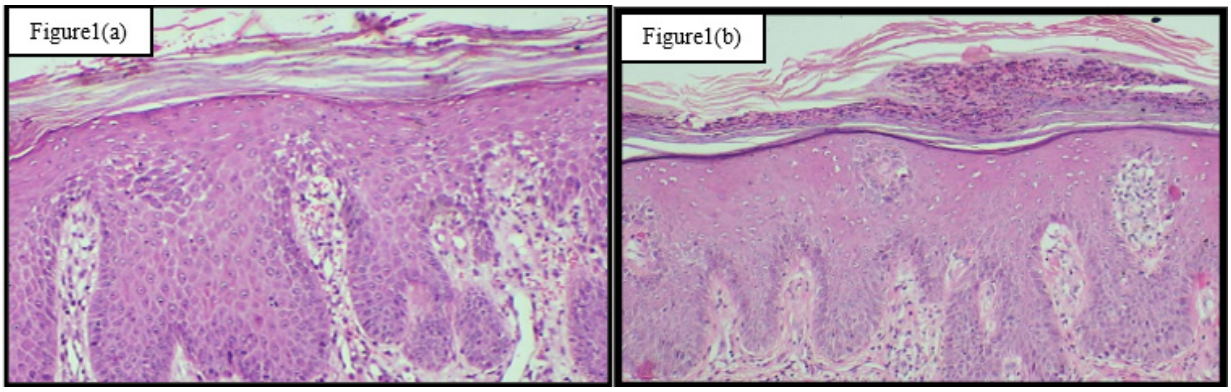

Figure 1: Psoriasis: high power view (a) showing Parakeratosis, regular acanthosis, thinning of suprapapillary plate, mild spongiosis and vascular changes (b) showing Munro's Micro abscess. (H&E stain 400x)

Table 4: Histological parameters

Histological Features	Cases Psoriasis Percentage (%)	Cases NPPD Percentage (%)
Elongation of rete ridges	91.4 (32)	-
Acanthosis	91.4 (32)	54.2 (19)
Vascular dilatation	85.7 (30)	10 (04)
Inflammatory infiltrate	71.4 (25)	71.4 (25)
Munro's microabscess	17.1 (06)	-
Parakeratosis	85.7 (30)	50 (18)
Diminshed/Absent granular layer	80 (28)	20 (07)
Spongioform pustules of kogoj	5.7 (02)	-
Suprapapillary thinning	85.7 (30)	28.6 (10)
Dermal edema	8.6 (03)	68.6 (24)
Elongation of dermal papillae	91.4	2.8 (01)

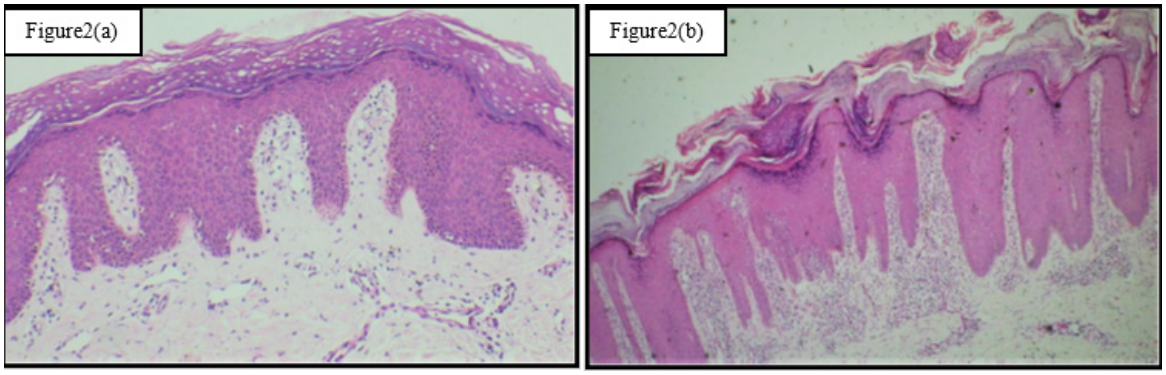

Figure 2: (a)Pityriasis rubra pilaris: Low power view showing hyperkeratosis, hypergranulosis, irregular acanthosis, thick suprapapillary plate and mild spongiosis (H&E, (b) Lichen simplex chronicus: Low power view showing hyperkeratosis, focal parakeratosis, hypergranulosis, irregular acanthosis, perivascular inflammatory infiltrate and dermal inflammation. (H&E stain, 100x).

(71.4%) followed by dermal edema (68.6%) and epidermal acanthosis (54.2%).

Table 5, the Psoriasis group was dominated by Chronic plaque Psoriasis (85.7%), followed by guttate and pustular Psoriasis. Ki-67 scores were consistently elevated across all psoriatic subtypes, with mean ranging from 59% to 81% and an overall mean of 74 ± 5%. The highest proliferative activity was observed in Pustular Psoriasis, while Chronic plaque Psoriasis also demonstrated markedly increased Ki-67 expression.

In the NPPD group, most patients were diagnosed with Pityriasis rubra pilaris (45.7%) and Eczema (34.3%). Ki-

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Table 5: Mean IHC Ki-67 percentage in Psoriasis and NPPD

Psoriasis (Histological Confirmed)	Number N (%)	Mean Ki-67 Value ± SD	NPPD (Histological Confirmed)	Number n (%)	Mean Ki-67 Value ± SD
Chronic plaque Psoriasis	30(85.7)	73±4.0	Pityriasis rubra pilaris	16 (45.71)	48±4.0
Guttate Psoriasis	03 (8.6)	68±7.0	Eczema	12 (34.29)	41±2.0
Pustular Psoriasis	02(5.7)	81±1.0	Lichen simplex	05(14.29)	35±4.0
			Pityriasis rosea	01(2.86)	43
			Parapsoriasis	01(2.86)	20
Total	35 (100)	74±5.0		35 (100)	43±7.0

67 scores in NPPD were uniformly low, with mean values ranging from 20% to 48% and an overall mean of 43 ±7.0%, indicating limited proliferative activity across NPPD.

The overall Ki-67 proliferation index was significantly higher in the Psoriasis group compared with the NPPD group

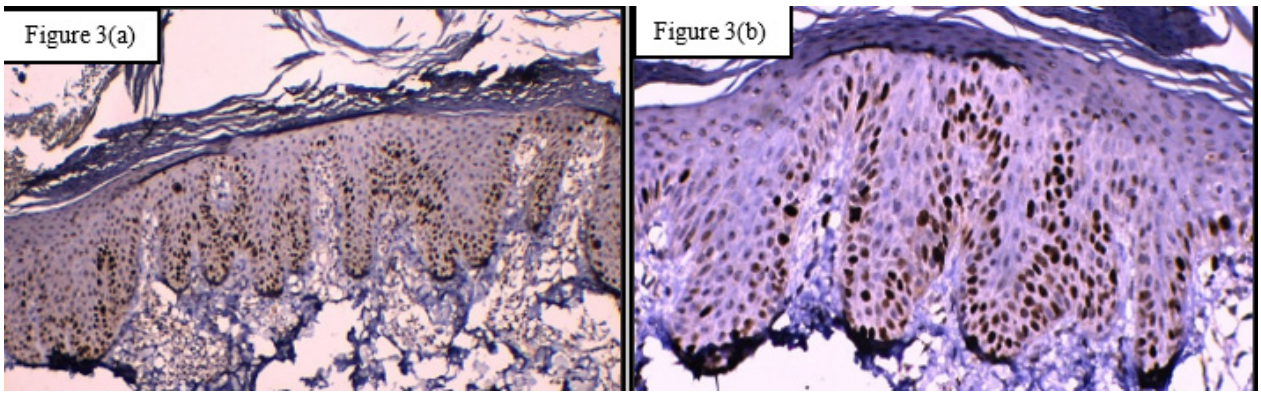

Figure 3: Psoriasis: a)Low power view showing strong positive expression (IHC Ki-67, 100x) b) High power view (IHC Ki-67, 400x)

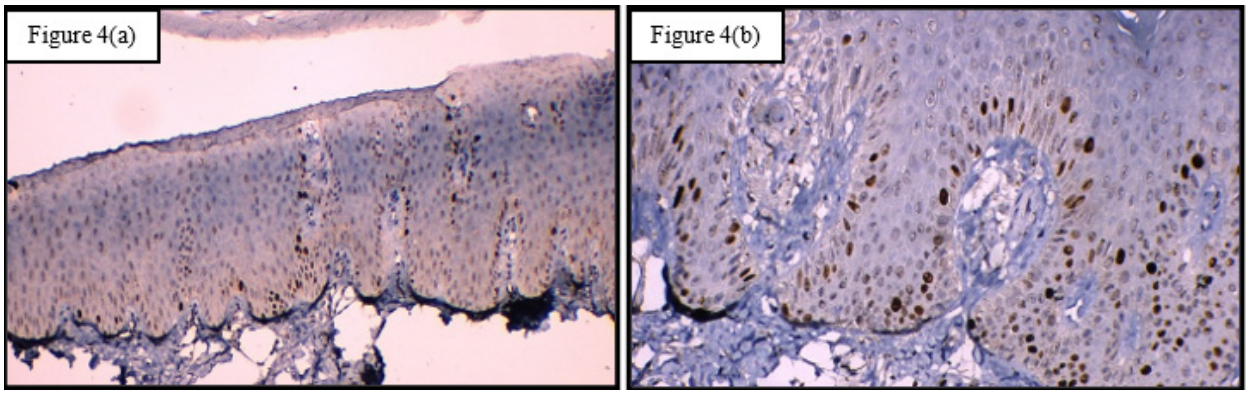

Figure 4: NPPD: a)Low power view (IHC Ki-67, 100x), b) High power view (IHC Ki-67, 400x)

Table 6: Comparison of Ki-67 score between Psoriasis and NPPD

Parameter	Psoriasis	NPPD	p-value
Ki-67 score (Mean ± SD)	74 ± 5%	43±7%	**< 0.001**
95% Confidence Interval*	67-72%	40-45%	

Table 7(a): Gender-wise Distribution of Ki-67 Score in NPPD Group

Gender	Percentage% (n)	Ki-67 Score (Mean ± SD)	p-value
Male	80 (28)	42.7 ± 7.20	**0.841**
Female	20 (07)	43.3 ± 6.30	

(74 ± 5%vs 43 ± 7%, $p < 0.001$). There was a robust and statistically reliable difference in Ki-67 expression between the two groups. This finding reflects markedly increased epidermal proliferative activity in the Psoriasis group relative to NPPD. Supported by the non overlap of 95% confidence intervals in the two group (Table 6).

Table 7(b): Gender-wise Distribution of Ki-67 Score in Psoriasis Group

Gender	Percentage% (n)	Ki-67 Score (Mean ± SD)	p-value
Male	68.6(24)	74.4 ± 4.30	**0.216**
Female	31.4 (11)	72.4± 4.80	

Table 8: Site-wise Distribution of Ki-67 Score in NPPD

Site	Psoriasis (Mean ± SD)	NPPD (Mean ± SD)
Lower extremities	77±4%	41±7%
Chest	76%	-
Abdomen	74 ± 4%	48±7%
Upper extremities	74±4%	42±5%
Back	72 ± 5%	47±4%
Scalp	70%	-

Table 9: Correlation of mean value of IHC Ki-67 with Body surface area involvement in Psoriasis

BSA	Number of patients	IHC Ki-67 (%)		p-value
		Mean	SD	
≤ 30%	4	66	0.03	<0.001
31%-50%	2	69.00	0.14	
>50%	29	75.00	0.04	

Table 10: Correlation of mean value of IHC Ki-67 with PASI score in Psoriasis

PASI Category	Number (n)	Mean value of IHC Ki-67 (%)	SD	p-value
<10	04	66.00	0.03	<0.001
>10	31	75.00	0.04	

Table 11: Mean value of IHC Ki-67 with PASI score in Chronic Plaque Psoriasis

PASI Category	Number (n)	Ki-67 Score (Mean ± SD)
< 10	**2**	**68**
> 10	**28**	**74.3 ± 3.5**

In both Psoriasis and NPPD, the mean Ki-67 scores were comparable between males and females, with no statistically significant differences observed (NPPD p= 0.841; Psoriasis p= 0.216). This indicates that Ki-67 expression is not influenced by gender in either group.(Table 7a and b).

(Table 8) The highest mean value of Ki-67 expression in Psoriasis was observed in the lower extremities(77 ± 4%) followed by chest (76%), abdomen (74 ± 4%), upper extremities(74 ± 4%), back(72 ± 5%) and scalp(70%) which showed the lowest mean value.The overall mean value in the present study was 74 ± 5%.

The highest mean Ki-67 expression in NPPD was observed in the abdomen (48 ± 7%), followed by back (47 ± 4%), upper extremities (42 ± 5%) and lower extremities (41 ± 7%). The overall mean Ki-67 index in the present study was 43 ± 7%.

The mean Ki-67 score showed only minor variation across body surface area (BSA) categories, with a slightly higher mean observed in patients with >50% BSA involvement. Ki-67 expression was significantly associated with the extent of Body surface area involvement in the present study. (demonstrated by ANOVA test, one way analysis of variance).(Table 9).

The mean Ki-67 score was higher in patients with PASI >10% compared to those with PASI <10%. However, the difference in mean Ki-67 scores between the two PASI categories was statistically significant (two-sample t test, $p = <0.001$). This indicates that Ki-67 expression differ significantly according to PASI severity category in the present study (Table 10).

In Chronic plaque Psoriasis, higher PASI scores (>10) were associated with higher mean Ki-67 values, suggesting increased epidermal proliferation with greater disease

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severity (Table 11).

Discussion

Psoriasis is a chronic immune-mediated inflammatory dermatosis marked by increased keratinocyte proliferation and aberrant differentiation. In order to assess Ki-67 expression in psoriasis and compare it with Non-psoriatic psoriasiform dermatoses (NPPD) and clinicopathological association, the current study was conducted.

In this study, patients with Psoriasis had a lower mean age (35.40 ± 12.84 years) than those with NPPD (41.34 ± 16.78 years), demonstrating that Psoriasis primarily affects younger individuals. These findings are comparable with Borade et al.[11], who showed similar age distribution, as well as epidemiological studies by Dogra et al.[3], which highlight the occurrence of Psoriasis in young to middle-aged persons. Bedi et al.[16] and Parisi et al.[13] observed peak incidence in early adulthood, corroborating the findings of the study.

In our study, there was a male predominance in the NPPD (80%) and psoriasis (68.6%) categories. This is similar to the findings of Borade et al.[11], Bedi et al.[16], Dogra et al.[3], and Asokan et al.[4], who also noted a male predominance in psoriasis, albeit in different proportions. The higher frequency among men could be due to either increased exposure to environmental factors or increased healthcare-seeking behavior.

The distribution of psoriasis sites in this study was varied, with the most prevalent sites being the back and upper extremities. Alhumidi et al.[18] and Dhar et al.[20] have reported similar findings. NPPD lesions, on the other hand, were primarily observed on lower extremities, suggesting a rather different distribution pattern.

Histopathologically, Psoriasis in the current study demonstrated classical features such as elongation of rete ridges, acanthosis, suprapapillary thinning, confluent parakeratosis, and vascular dilation. These results are in line with earlier research by Ahasan et al.[17], Gupta et al.[14], Anupama et al.[19], and Venna et al.[15], all of which highlight these traits as defining aspects of Psoriasis. Kambale et al.[22] validated their findings with morphometric analysis. However, similar to Anupama et al.[19], Munro's microabscess and Kogoj pustules were less common in our study. This could be because of differences in the biopsy site or disease stage.

On the other hand, irregular acanthosis, edema, and inflammatory infiltrate were the main characteristics of NPPD in the current investigation. These results are consistent with comparable histological patterns reported by Ahasan et al.[17] and Gupta et al.[14]. The absence of neutrophilic microabscesses and milder vascular alterations can help differentiate NPPD from Psoriasis. Venna et al.[15] and Kambale et al.[22] also emphasized spongiosis and the absence of regular epidermal hyperplasia as distinctive characteristics of NPPD.

The much higher Ki-67 expression in psoriasis (74 ± 5%) compared to NPPD (43 ± 7%) ($p < 0.001$) is one of the study's main key findings. This suggests that Psoriasis has significantly higher epidermal proliferative activity. This is consistent with the basic pathogenesis of Psoriasis, which is marked by a rapid turnover of keratinocytes. These results are consistent with those of Borade et al.[11] and Abdelsalam et al.[21], who also found that Psoriasis had much higher Ki-67 expression than Psoriasiform dermatoses.

Increased Ki-67 expression in Psoriasis has a well-established biological basis. Gerdes et al.[12] demonstrated that Ki-67 is a reliable marker of actively proliferating keratinocytes, while Weinstein et al.[25] showed that Psoriasis is associated with shortened cell cycle and increased epidermal turnover. According to Nickoloff et al.[24], immune-mediated stimulation of keratinocytes causes hyperproliferation, which raises the expression of Ki-67.

Higher Ki-67 expression in Psoriasis has also been shown in other research, such as Sharma et al.[28] and Sanasam et al.[26], indicating its diagnostic value. However, Ramezani et al.[27] reported that Psoriasiform dermatoses had increased Ki-67 expression than Psoriasis in their study. This findings contradicts the widely held belief that Psoriasis is always linked to increased keratinocytye proliferative activity. The authors suggested that rather than a proliferative pattern unique to the disease, elevated Ki-67 expression in NPPD might be the result of accelerated epidermal turnover brought on by acute inflammatory activity. The study came to the conclusion that although Ki-67 is a helpful marker of epidermal proliferation, morphological and histological characteristics should be taken into account when interpreting it.

In addition to being diagnostically significant, Ki-67 expression in this study demonstrated a strong positive correlation with both Body surface area (BSA) involvement ($r = 0.84$, $p < 0.001$) and PASI score ($r = 0.80$, $p < 0.001$), suggesting that increased proliferative activity is linked to greater disease severity and extent. These results are in line with those of Borade et al.[11] and Gupta et al.[2], who found comparable associations between disease severity indices and Ki-67 expression. Additionally, Karpouzis et al.[23] showed elevated Ki-67 expression in active psoriatic lesions, confirming its function as a disease activity marker.

The basic pathophysiology of Psoriasis, where increased keratinocyte proliferation contributes to plaque formation and disease severity, is reflected in the current study's increasing increase in Ki-67 expression with greater PASI scores and BSA involvement. These results imply that Ki-67 could be a useful biomarker for diagnosis, illness severity assessment and therapy response tracking.

The current study concludes that there is a considerable association between the severity and extent of Psoriasis and

Ki-67 expression, which is significantly higher in Psoriasis than in NPPD. These results demonstrate the usefulness of Ki-67 as an addition to routine histology in distinguishing Psoriasis from NPPD and as a potential prognostic indicator of disease, and they are corroborated by earlier research (Borade et al.[11], Abdelsalam et al.[21], Sharma et al.[28].

Conclusion

Ki-67 expression is significantly elevated in Psoriasis compared to NPPD, highlighting its diagnostic utility as an adjunct Immunohistochemical marker. More classical histopathological features of psoriasis such as regular rete ridge elongation, thinning of suprapapillary region, confluent parakeratosis diminished granular layer and vascular dilatation, Munro's microabscesses were more common in Psoriasis cases. The increased Ki-67 labeling index, supports its potential role as an accessory immunohistochemical marker for the distinction of Psoriasis from other psoriasiform dermatoses in cases with overlap.

Limitations

The current study has some limitations, including a small sample size and a high prevalence of Chronic Plaque Psoriasis, which may limit the scope of the findings. Larger multicentric investigations are needed to further validate the diagnostic cut-off values and prognostic importance of Ki-67, as the current study was limited in sample size.

Statements and Declarations Conflicts of Interest

The authors declare that they do not have conflict of interest.

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