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Original Article

Pathology Section

Differentiating Psoriasis from Psoriasiform Dermatitis using Proliferation Markers Ki-67 and Cyclin D1: A Cross-sectional Study

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ABSTRACT

Introduction: Psoriasis shares several clinical and histopathological features with other psoriasiform dermatoses. Differentiating it from other psoriasiform dermatitis is a diagnostic challenge which holds therapeutic as well as prognostic significance.

Aim: To compare the immunohistochemical expression of proliferative markers Ki-67 and Cyclin D1 in psoriasis and other psoriasiform dermatitis and to determine if these markers can help differentiate them.

Material and Methods: A cross-sectional study was conducted at the Department of Pathology, ESI-Post Graduate Institute of Medical Sciences and Research (ESI-PGIMSR), a tertiary care hospital in Basaidarapur, New Delhi, India, from April 2023 to August 2024, where 27 biopsies each of psoriasis and psoriasiform dermatitis were included. Immunohistochemistry (IHC) was performed using Ki-67 and Cyclin D1 antibodies, and their expression was evaluated. Three parameters—total epidermal cell count, suprabasal epidermal cell count, and the suprabasal-to-total epidermal cell count ratio—were compared between the two groups. Receiver Operating Characteristic (ROC) curve analysis was used to determine optimal cut-off values for these parameters. An Independent t-test was used to analyse the results using the Statistical Package for Social Sciences (SPSS) version 20.0.

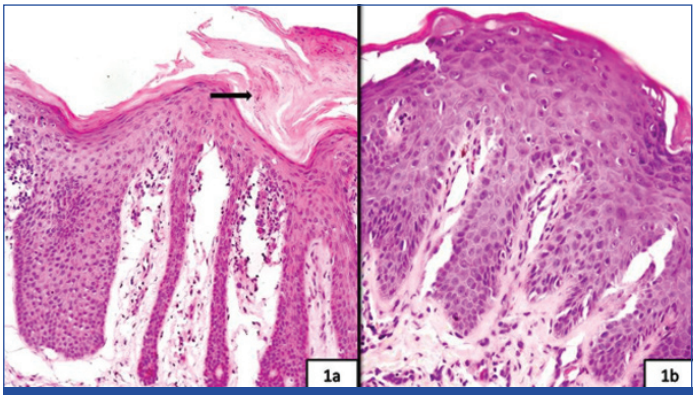
Results: The total epidermal cell count was significantly higher in psoriasis than in psoriasiform dermatitis for both markers. For Ki-67, the mean count was 386 vs 356 cells/mm² ($p<0.001$), while for Cyclin D1 it was 360 vs 329 cells/mm² ($p<0.001$). Similarly, the suprabasal epidermal cell count was also higher in psoriasis for both Ki-67 (343 vs 298 cells/mm²; $p<0.001$) and Cyclin D1 (333 vs 305 cells/mm² - $p=0.036$). The suprabasal-to-total epidermal cell count ratio was significantly higher in psoriasis, with values of 89% vs 84% for Ki-67 ($p=0.001$). ROC curve analysis yielded cut-off values of 370 and 318 cells/mm² for the total and suprabasal Ki-67 counts, respectively and 396 and 325 cells/mm² for the total and suprabasal Cyclin D1 counts, respectively.

Conclusion: Ki-67 and Cyclin D1 exhibit significantly different immunohistochemical expression patterns in psoriasis and psoriasiform dermatitis. Quantitative assessment of these markers, along with objective cut-off values, may serve as an objective adjunctive diagnostic tool. IHC can therefore be a valuable tool in challenging diagnostic cases.

Keywords: Biopsies skin, Immunohistochemistry, Psoriatic

INTRODUCTION

Psoriasis is a chronic inflammatory skin disorder characterised by hyperproliferation of keratinocytes. It exhibits marked epidermal hyperplasia with a cellular turnover rate that is approximately six to seven times higher than that of normal skin. This abnormal hyperplasia is associated with an expansion of the germinative cell layer and an increased mitotic rate [1]. Several dermatological conditions display clinical and histopathological features similar to psoriasis and are collectively referred to as psoriasiform dermatitis. Histologically, the psoriasiform reaction pattern is characterised by epidermal hyperplasia with regular elongation and widening of the rete ridges. Conditions that may exhibit a psoriasiform pattern include pityriasis rubra pilaris, pityriasis rosea, lichen simplex chronicus, reactive arthritis, parapsoriasis, and subacute or chronic spongiotic dermatitis [2]. The considerable overlap in clinical and histopathological findings between psoriasis and other psoriasiform dermatoses often makes definitive diagnosis difficult [2] [Table/Fig-1]. Traditionally, a confident diagnosis of psoriasis requires the demonstration of Munro microabscesses and spongiform pustules of Kogoj, findings that may not be evident in every biopsy specimen and sometimes necessitate repeat biopsies [3] [Table/Fig-1a]. Accurate diagnosis of psoriasis is essential because of its association with several co-morbidities, including psoriatic arthritis, cardiovascular disease, and autoimmune disorders, all of which may significantly affect patient prognosis. Furthermore, the emergence of targeted therapies, such as ustekinumab (anti-IL-12/23) and secukinumab (anti-IL-17A), has increased the importance of distinguishing psoriasis from other psoriasiform disorders, as these therapies are specifically indicated for psoriasis [4].



[Table/Fig-1]: a) Histopathology of psoriasis, arrow depicting Munro microabscess, H&E, 400X; b) Histopathology of psoriasiform dermatitis (H&E, 400x).

IHC has been investigated as a potential adjunctive diagnostic tool in this setting. Several studies have reported the usefulness of proliferative markers such as Ki-67 and Cyclin D1 in differentiating psoriasis from other psoriasiform dermatitis [5-10]. The cell proliferation marker Ki-67 is expressed in G1,S and G2 phases of the cell cycle; hence, it can be used to diagnose epidermal hyperproliferation, which is a hallmark of psoriatic lesions. Cyclin D1 is a cell cycle regulatory protein that is crucial for the G1/S

phase transition by phosphorylation of the retinoblastoma gene and release of the E2F transcription factor. Cyclin D1 overexpression causes cells to reach the G1/S phase quickly, resulting in overall cell proliferation. These two markers may be used as a diagnostic tool to distinguish psoriasis and other psoriasiform dermatitis [11].

The present study was conducted with the objective of comparing the expression of proliferative markers Ki-67 and Cyclin D1 between classic psoriasis and other psoriasiform dermatitis on IHC. The objectives were

- To observe the IHC expression of Ki-67 and Cyclin D1 in psoriasis and then calculate three parameters, i.e. total epidermal cell count, suprabasal cell count and suprabasal total epidermal cell ratio.
- The other was to compare the above-mentioned three parameters between psoriasis and psoriasiform dermatitis and to determine if the difference was significant.
- The last objective was to calculate a cut-off for each of the above parameters to aid in the differentiation between psoriasis and psoriasiform dermatitis. There are very few studies that have tried to determine a cut-off to differentiate psoriasis from psoriasiform dermatitis [5,7,12].

MATERIALS AND METHODS

A cross-sectional study was conducted at the Department of Pathology, ESI-Post Graduate Institute of Medical Sciences and Research (ESI-PGIMSR), a tertiary care hospital in Basaidarapur, New Delhi, India, from April 2023 to August 2024, after the approval of the Institutional Ethics Committee- Reg. No. IEC/2023042 dated 27-03-2023 in accordance with the 1964 Helsinki Declaration and its later amendments. Informed consent was obtained from all individual participants included in the study.

Inclusion criteria: Skin biopsies of all clinically diagnosed cases of psoriasis and other psoriasiform dermatitis sent for histopathological diagnosis were included.

Exclusion criteria: Cases with inadequate tissue in the blocks to perform IHC were excluded from the study.

Sample size: Sample size was calculated on the basis of variation in total epidermal cell count for Ki-67 in classic psoriasis and psoriasiform groups using the formula.

Calculating minimum sample size for 90% power

Given

- $Z_{1-\beta}=1.28$ for 90% power.
- $\sigma=\sqrt{(s1^2 + s2^2)/2}$ pooled SD.
- $\Delta=|\mu1 - \mu2|$ difference in means.

Study values for Suprabasal Ki-67

- $\mu1=401.2$, $s1=169.0$ (psoriasis)
- $\mu2=181.6$, $s2=97.4$ (NPPD)
- $\Delta=219.6$
- $s1^2=28561$, $s2^2=9487$
- $\sigma=\sqrt{(28561+9487)/2}=\sqrt{19024}=137.9$
- $Z_{sum}=1.96+1.28=3.24$

Formula and calculation

$$n = k\frac{\left(z_a + z_b\right)^2\left(s_1^2 + s_2^2\right)}{d^2}$$

$n=[2 (Z_{sum}^2 (\sigma)^2] / (\Delta)^2$

$n=[2 (3.24)^2 (137.9)^2] / (219.6)^2$

$n=[20.9952 * 19024] / 48224.16 \approx$

Required n per group=9

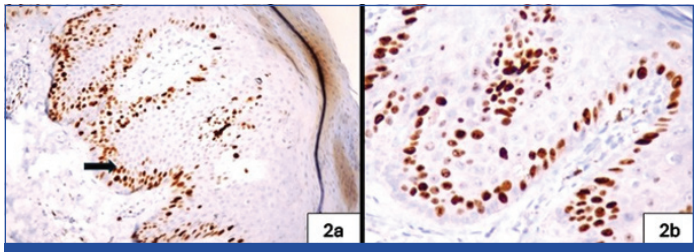
Total=18.

Since this is the minimum sample size required, more (n=27) each in both the groups were included.

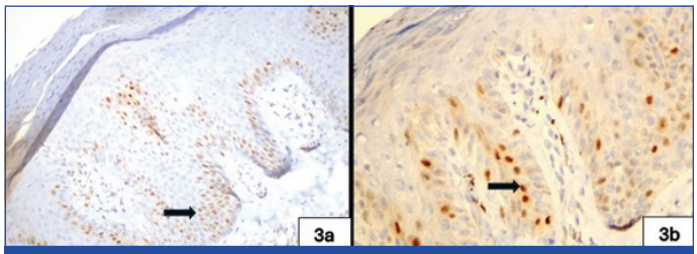
Note: The assumptions used for sample size estimation were derived from pilot observations and differed from the values ultimately observed in the study. This discrepancy likely reflects the limited precision of pilot estimates due to the small pilot sample size. While the final variability was lower than anticipated, the pilot-derived estimates were used prospectively to ensure adequate study power during the planning phase.

Study Procedure

A brief clinical data of the patient, such as age, gender and clinical diagnosis, were collected. Haematoxylin and Eosin (H&E) staining was performed and cases were reported as either psoriasis or other psoriasiform dermatitis. IHC was performed using prediluted rabbit monoclonal primary antibodies against Ki-67 (SP6 clone, BioCare) and Cyclin D1 (EP12 clone, PathnSitu) [Table/Fig-2,3]. Fully automated IHC staining was done using the Ventana Benchmark GX Instrument. Ventana UltraView Universal DAB (3, 3-diaminobenzidine) detection kit was used for secondary visualisation. Ki-67 and Cyclin D1 positive cells were counted per mm^2 in full epidermal thickness (total epidermal count) and suprabasal layer (suprabasal count). Suprabasal to total epidermal count ratios were calculated for each of the markers [Table/Fig-2,3] [5].



[Table/Fig-2]: Ki-67 showing higher suprabasal staining in psoriasis (a) 200X, depicted by an arrow as compared to lower suprabasal staining in other psoriasiform dermatitis (b) 400X, immunohistochemistry.



[Table/Fig-3]: Cyclin D1 showing higher suprabasal staining in psoriasis (a) 200X, depicted by an arrow, as compared to lower suprabasal staining in other psoriasiform dermatitis (b) 400X, immunohistochemistry.

STATISTICAL ANALYSIS

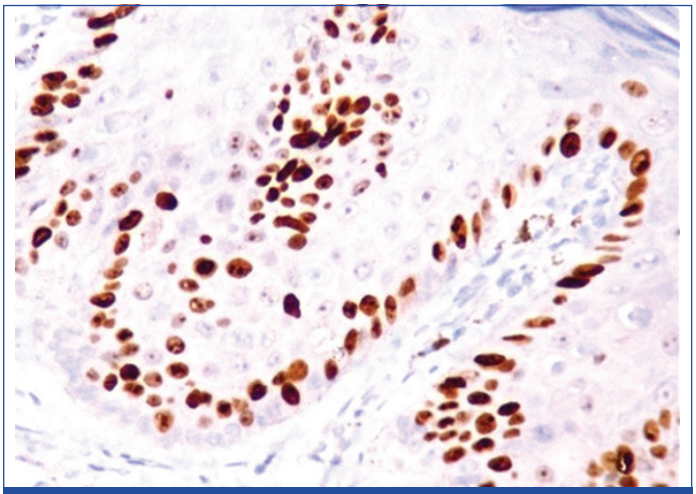
Data were analysed using SPSS version 20.0. Descriptive analysis was used to determine the mean age and gender distribution of the subjects. Variation of mean and standard deviation for total and suprabasal epidermal cell count of Ki-67, Cyclin D1 and suprabasal/total epidermal cell count ratio was analysed by using the Independent t-test. Results with a p-value less than 0.05 were considered statistically significant. A ROC curve analysis was conducted to evaluate the diagnostic accuracy of both Ki-67 and Cyclin D1 total epidermal cell count and suprabasal cell count.

To count cells per mm^2, a clear ruler was used to measure the diameter of a low-power field. It was used to calculate a constant based on the following formula:

Eyepiece Magnification x Objective Magnification x Microscopic Field Diameter=A Constant.

When the value of the constant is known, the diameter of an HPF was calculated for other objectives by using the following formula:

Unknown Field Diameter=Constant/ (Eyepiece Magnification x Objective Magnification). Half of the field diameter is the radius of the field (r), which was then used to calculate the area of the HPF: 3.1415 x r 2=Area of Microscopic Field. The total number of cells was counted in the field and divided by the area to get cells per mm^2. The diameter of the high-power field of our microscope was 0.64 mm, calculated area was 0.322 mm^2. For example, to calculate the total epidermal cell count, the total number of positive cells in the epidermis was counted, which is 150 in this figure. The area of this microscopic field is 0.322 mm^2. So count per mm^2 becomes 150/ 0.322= 466 [Table/Fig-4] [13].



[Table/Fig-4]: Example depicting the calculation of Total epidermal count/mm^2 of 466, 400X, IHC.

RESULTS

The study included patients with psoriasis and psoriasiform dermatitis with comparable demographic characteristics. The mean age of patients in the psoriasis group was 42.81±18.96 years, whereas that in the psoriasiform dermatitis group was 37.00±12.6 years. Although the mean age was higher in the psoriasis group, the Independent t-test, degrees of freedom=52).

In terms of gender distribution, males predominated in both groups. In the psoriasis group, 20 patients (74.07%) were male and 7 (25.93%) were female, while in the psoriasiform dermatitis group, 19 patients (70.37%) were male and 8 (29.63%) were female.

With respect to Ki-67 expression, the mean total epidermal cell count, mean suprabasal epidermal cell count, and the mean suprabasal-to-total epidermal cell count ratio were all higher in the psoriasis group compared to the psoriasiform dermatitis group. These differences were statistically significant for all three parameters (p<0.05) [Table/Fig-5].

Markers	Parameters	Psoriasis (Number of cells showing positivity)	Psoriasiform dermatitis (Number of cells showing positivity)	p-value
Ki- 67	Total epidermal cell count (cells/mm^2)	385.78±21.98	355.93±13.95	<0.001
	Suprabasal epidermal cell count (cells/mm^2)	342.93±20.27	297.15±18.59	<0.001
	Suprabasal/Total cell count ratio (%)	89.11±6.31	83.52±4.58	0.001
Cyclin D1	Total epidermal cell count (cells/mm^2)	360.48±22.03	329.89±8.92	<0.001
	Suprabasal epidermal cell count (cells/mm^2)	332.93±20.27	304.52±9.82	0.036
	Suprabasal/Total cell count ratio (%)	104.40±13.22	92.37±3.76	<0.001

[Table/Fig-5]: Comparison of parameters between psoriasis and psoriasiform dermatitis.

Similarly, for Cyclin D1 expression, the mean total epidermal cell count, mean suprabasal epidermal cell count, and the suprabasal-to-total epidermal cell count ratio were significantly higher in the psoriasis group (p<0.05) [Table/Fig-5].

To objectively differentiate between psoriasis and psoriasiform dermatitis, cut-off values were derived for two parameters: total epidermal cell count and suprabasal epidermal cell count, for both Ki-67 and Cyclin D1 [Table/Fig-6]. These cut-offs were determined based on ROC curve analysis.

Markers	Parameters	Psoriasis (cut-off)	Psoriasiform dermatitis (cut-off)
Ki-67	Total epidermal cell count (cells/mm^2)	>370	<370
	Suprabasal epidermal cell count (cells/mm^2)	>318	<318
	Suprabasal/Total cell count ratio (%)	>85%	<85%
Cyclin D1	Total epidermal cell count (cells/mm^2)	>396	<396
	Suprabasal epidermal cell count (cells/mm^2)	>325	<325
	Suprabasal/Total cell count ratio (%)	>85%	<85%

[Table/Fig-6]: Cut-offs to differentiate between psoriasis and psoriasiform dermatitis.

DISCUSSION

Psoriasis is the prototype of psoriasiform dermatitis. It needs to be differentiated from other psoriasiform dermatitides, which share complex and overlapping microscopic features. Diagnosis can be challenging due to clinico-histopathologic similarities [7].

The characteristic histopathological features of psoriasis are progressive epidermal hyperplasia with regular elongation of rete ridges, thin suprapapillary plates with the presence of tortuous dilated vasculature in papillary dermis, focal parakeratosis, hypogranulosis with neutrophil collections in stratum corneum (Munro micro abscess) and in the interstices of spongiotic spinosum (Kogoj pustule). As stated earlier, the diagnosis can be made with certainty only when one can demonstrate Munro microabscess and Kogoj pustule, which may not be evident in all the stages of the disease and hence may require multiple biopsies [3]. This may not be feasible due to various reasons, such as the presence of only a single lesion. Also, it may not be desirable by the patient due to cosmetic reasons or due to the presence at an unusual site. Common other psoriasiform dermatitides include pityriasis rubra pilaris, lichen simplex chronicus, Reiter's syndrome and pityriasis rosea. All of these resemble psoriasis and share one or more of the clinicopathological features of psoriasis. It becomes difficult many times to reach an exact diagnosis. Hence, the role of other modalities in conjunction with routine histology needs to be studied. A few studies have reported IHC as a successful tool to differentiate psoriasis from psoriasiform dermatitis using Ki-67 and Cyclin D1 markers [5-10].

The mean age difference between the psoriasis group and the psoriasiform dermatitis group was not statistically significant. This similarity in age distribution strengthens the comparability of the two groups, reducing the potential for age-related confounding factors. The maximum number of cases of psoriasis was in the age group 40 to 59 in the current study. The gender distribution in psoriasis (70% male, 30% female) indicates a male predominance in the study population. The significantly higher total epidermal Ki-67 count in psoriasis compared to psoriasiform dermatitis reflects the hyperproliferative state characteristic of psoriatic lesions. This aligns with the clinical presentation of psoriasis, which features thickened, scaling plaques due to accelerated keratinocyte turnover.

The higher suprabasal total epidermal cell count ratio of Ki-67 in psoriasis compared to psoriasiform dermatitis further emphasises the altered proliferation dynamics in psoriasis. This ratio suggests that not only is there more proliferation overall in psoriasis, but a greater proportion of this proliferation is occurring in the suprabasal layers, underscoring the dysregulation of epidermal homeostasis in this condition. Sanasam D et al., conducted a study in 2021 and concluded that in psoriasis, >50% of the Ki-67 positive keratinocytes were found in the suprabasal layer of the epidermis, while it was <50% in the case of psoriasiform dermatitis [6]. A 2022 cross-sectional study by Abdelsalam H et al., found that Ki-67 expression was significantly higher in psoriasis patients compared to the other dermatitis studied [7]. A few other studies have also found statistically significantly higher expression of Ki-67 [Table/Fig-7] [5-10]. Similar findings were reported in a 2024 clinicohistopathological study by Vemavarapu K et al., which demonstrated significantly increased Ki-67 immunoexpression in psoriasis compared with psoriasiform lesions, supporting the utility of proliferative markers in difficult diagnostic cases [12]. The total Cyclin D1 count was significantly higher in psoriasis, although the discriminatory performance of Ki-67 appeared superior. This could suggest that while overall cell cycle entry (as indicated by Cyclin D1) may be similar, the progression through later stages of the cell cycle (as reflected by Ki-67) differs between the two conditions. Ki-67 appears to be a more consistent differentiator across all measurements. Recent advances in the understanding of psoriasis pathogenesis further support the role of proliferative biomarkers in disease evaluation. Contemporary studies have emphasised that psoriasis is driven by complex immune-mediated epidermal hyperproliferation involving keratinocyte activation, cytokine dysregulation, and altered epidermal turnover, thereby explaining the increased expression of proliferation-associated markers such as Ki-67 and Cyclin D1 [14]. This difference could be attributed to their roles in the cell cycle: while Cyclin D1 is involved in the initiation of cell cycle entry, all active phases of the cell cycle express Ki-67, potentially making it a more comprehensive marker of ongoing proliferation. A 2015 study by Sezer E et al., found that Ki-67 is a more sensitive marker than Cyclin D1 in distinguishing psoriasis from other psoriasiform dermatitis [5]. Their results are similar to the present study. The results of this study demonstrate that Ki-67 and Cyclin D 1 are valuable proliferative markers in distinguishing psoriasis from other psoriasiform dermatitis. The significantly higher expression of these markers in psoriasis reflects the hyperproliferative nature of the disease, which is a hallmark of psoriasis. The high diagnostic accuracy of Ki-67 and Cyclin D1, as shown in the analysis, suggests that these markers can be useful tools in the diagnosis of psoriasis, particularly in cases where clinical and histopathological features are unclear. The expression of Ki-67 and Cyclin D1 in other psoriasiform dermatitis was significantly lower than in psoriasis, indicating that these markers may help differentiate psoriasis from other inflammatory skin diseases. The study's findings are consistent with previous research, which has shown that Ki-67 and Cyclin D1 are overexpressed in psoriasis. Recent literature has also explored additional molecular pathways associated with epidermal proliferation in psoriasis. Golob-Schwarzl N et al., (2024) demonstrated the importance of translational regulatory pathways such as eIF4E in sustaining keratinocyte proliferation in psoriatic lesions, highlighting the growing interest in molecular proliferation markers as potential diagnostic and therapeutic targets [15]. However, this study's results provide new insights into the diagnostic utility of these markers for differentiating psoriasis from other psoriasiform dermatitis, as we could derive cut-offs for both the markers to differentiate between Psoriasis and Psoriasiform dermatitis. This will objectify the differentiation between Psoriasis and psoriasis-like dermatitis and will be of great diagnostic help. The current study findings align with the study done by Sezer E et al., [5]. They found a cut-off of 75% for Ki-67 suprabasal to total epidermal cell count ratio, however were not able to derive any cut-off for Cyclin D1 [5].

Authors	Year	Marker	Conclusion of the study
Abdelsalam HF et al., [7]	2022	Ki-67	Mean value of Ki-67 was highly significant in psoriasis versus that in psoriasiform dermatitis
Sanasam D et al., [6]	2021	Ki-67	Significantly higher expression in suprabasal keratinocytes. >50% Ki-67 positive keratinocytes in suprabasal layer in psoriasis.
Kim SA et al., [8]	2018	Cyclin D1, Ki-67, pRB, p53	Expression levels of all were higher in chronic psoriasis.
Sezer E et al., [5]	2015	Ki-67, Cyclin D1	Ki-67 expression was significantly higher in psoriasis. Cut-off value of 75% was found for suprabasal to total epidermal count to differentiate psoriasis from other psoriasiform dermatitis.
Abdou AG et al., [9]	2013	Ki-67, Glut1	Percentage of Ki-67 expression was higher in psoriasis than normal skin and correlated with nucleolar pattern of staining. Glut1 was also upregulated in psoriasis.
Amin MM and Azim ZA [10]	2012	Osteopontin, Ki-67, CD34	Statistically significant higher expression of all was noted in lesional skin as compared to non lesional skin.
Current study	2026	Ki-67,Cyclin D1	IHC expression of Ki-67 and Cyclin D1 differs significantly between psoriasis and psoriasiform dermatitis. Objective cut offs using these IHC markers can simplify the differentiation.

[Table/Fig-7]: Studies which have utilised IHC to differentiate between psoriasis and psoriasiform dermatitis [5-10].

Limitation(s)

The results of the present study need further validation in larger cohorts. Additionally, the study did not investigate the expression of these markers in various subtypes of psoriasis and in other skin diseases, which could provide further insights into their diagnostic utility.

CONCLUSION(S)

The combination of Ki-67 and Cyclin D1 provides valuable insights into cell proliferation in psoriasis and related dermatitis. Both Ki-67 and Cyclin D1 are reliable proliferative markers for diagnosis and differentiating psoriasis from psoriasiform dermatitis. A cut-off as found in the current study will simplify the differentiation between psoriasis and other psoriasiform dermatitis. Both markers can aid in understanding the pathology and guiding treatment approaches for these conditions.

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- Manual Googling: Jun 18, 2026
- iThenticate Software: Jun 20, 2026 (10%)

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