Journal of Cardiovascular Disease Research
ISSN: 0975-3583,0976-2833 VOL 12, ISSUE 9, 2021

An Evaluation on Immunohistochemical Profiling to Differentiate Psoriasis from Psoriasiform Dermatitis at a Tertiary Care Centre of West Bengal : A Cross-Sectional Study.

Dr.Prerna Raj Gupta [1] , Dr. Aftabuddin Ahmed [2] , Dr.Advait Akash[3] , Dr. Naresh Kumar Munda[4]

[1]Assistant Professor, Department of Dermatology, Faculty of Icare Institute of Medical Sciences and Research and Dr. B C Roy Hospital, Haldia, India.

[2]Assistant Professor, Department of General Surgery , Faculty of Icare Institute of Medical Sciences and Research and Dr. B C Roy Hospital, Haldia, India.

[3]Assistant Professor, Department of General Medicine , Faculty of Gouri Devi Institute of Medical Sciences & Hospitals, Durgapur.

[4]Assistant Professor, Department of Community Medicine, Faculty of Icare Institute of Medical Sciences and Research and Dr. B C Roy Hospital, Haldia, India.

Corresponding Author: Dr. Naresh Kumar Munda, **E mail**: drnaresh2k@gmail.com.

Received-14.07.2021, Accepted-15.08.2021., published-16.09.2021

ABSTRACT

Background: Psoriasis and psoriasiform dermatitis present with strikingly similar histological features under routine haematoxylin and eosin (H&E) staining, making definitive diagnosis a persistent clinical challenge. Immunohistochemical (IHC) profiling has emerged as a reliable adjunct tool in resolving such diagnostic ambiguities. **Objectives:** To evaluate the role of immunohistochemical markers — specifically Ki-67, CD3, CD4, CD8, and HLA-DR — in differentiating psoriasis from psoriasiform dermatitis in skin biopsy specimens. **Methodology:** A hospital-based cross-sectional study was conducted on 82 patients attending the Dermatology OPD of a tertiary care centre in West Bengal. Skin biopsies were subjected to H&E and IHC staining. Sample size was calculated using the standard formula for cross-sectional studies. Consecutive sampling technique was employed. **Results:** Of 82 cases, 48 (58.5%) were confirmed as psoriasis and 34 (41.5%) as psoriasiform dermatitis. Ki-67 showed significantly higher epidermal proliferative activity in psoriasis ($p<0.001$). CD8+ T-lymphocytes were more prominent in psoriasis,

whereas CD4+ predominance was noted in psoriasiform dermatitis. HLA-DR positivity was stronger in psoriasis. **Conclusion:** IHC profiling is a valuable and reproducible tool in differentiating psoriasis from psoriasiform dermatitis, offering significant clinical and therapeutic implications. Integration of IHC into routine dermatopathological practice is strongly recommended.

Keywords: *Psoriasis, Psoriasiform Dermatitis, Immunohistochemistry, Ki-67, CD3, CD8, West Bengal*

1. INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory skin disorder affecting approximately 2–3% of the global population. It is characterised by well-defined erythematous plaques with silvery scales, typically affecting the scalp, elbows, knees, and lower back[1]. In India, the prevalence ranges between 0.44% and 2.8%, with considerable burden noted in tertiary care dermatology settings across West Bengal.

Psoriasiform dermatitis, on the other hand, is a broad histopathological term encompassing a group of inflammatory skin diseases that microscopically resemble psoriasis but have distinctly different aetiologies, clinical courses, and treatment strategies. Conditions such as seborrhoeic dermatitis, lichen simplex chronicus, pityriasis rubra pilaris, and chronic spongiotic dermatitis commonly fall under this category[2].

Routine H&E staining, although indispensable, frequently fails to draw a sharp distinction between these two entities owing to overlapping morphological features such as acanthosis, parakeratosis, hypogranulosis, and Munro's microabscesses. This diagnostic uncertainty often leads to inappropriate therapy, delayed patient recovery, and increased economic burden on both the patient and the healthcare system[3].

Immunohistochemistry (IHC) has transformed the landscape of diagnostic pathology by enabling the characterisation of cellular constituents and inflammatory infiltrates at a molecular level. Markers such as Ki-67 (a nuclear proliferation antigen), CD3, CD4, CD8 (T-lymphocyte subsets), and HLA-DR (MHC class II antigen) have shown promising results in the differential diagnosis of papulosquamous disorders[4].

Despite growing global evidence, there remains a significant paucity of IHC-based studies from Eastern India and West Bengal in particular. This study was therefore undertaken to systematically evaluate the immunohistochemical profile of psoriasis versus psoriasiform dermatitis in a tertiary care hospital in West Bengal, with the aim of generating region-specific data that may guide clinical and pathological practice.

2. OBJECTIVES

The primary objective of this study was to evaluate immunohistochemical markers (Ki-67, CD3, CD4, CD8, and HLA-DR) in skin biopsy specimens to differentiate psoriasis from psoriasiform dermatitis among patients attending a tertiary care centre in West Bengal.

The secondary objectives included assessment of the sociodemographic profile of the study participants, correlation of IHC findings with clinical and H&E morphology, and estimation of the diagnostic accuracy of IHC profiling in this clinical context.

3. METHODOLOGY

3.1 Study Design and Setting

A hospital-based, cross-sectional observational study was conducted in the Department of Pathology and Dermatology of a tertiary care medical college and hospital in West Bengal, India, over a period of eighteen months.

3.2 Sample Size Calculation

The sample size was calculated using the following standard formula for cross-sectional studies:

$$\mathbf{n = Z^2 \times P \times (1 - P) / d^2}$$

Where:

n = required sample size

Z = 1.96 (Z-value at 95% confidence interval)

P = expected prevalence of psoriasis among papulosquamous disorders = 0.50 (taken as 50% to maximise sample size)

d = allowable margin of error = 0.05 (5%)

$$n = (1.96)^2 \times 0.50 \times 0.50 / (0.05)^2 = 3.8416 \times 0.25 / 0.0025 = 384.16 \approx 385$$

Adjusting for constraints of institutional patient load and study duration, and applying a finite population correction factor along with an expected attrition rate of ~20%, the final sample size was determined to be 82 patients, which was deemed adequate for this cross-sectional analytical study.

3.3 Sampling Method

Consecutive sampling technique was employed wherein all eligible patients attending the Dermatology OPD with clinical suspicion of psoriasis or psoriasiform dermatitis during the study period were enrolled sequentially until the desired sample size was achieved. This method ensured inclusivity and minimised selection bias.

3.4 Inclusion and Exclusion Criteria

Inclusion criteria: Patients of all age groups and both sexes presenting with clinical features suggestive of psoriasis or psoriasiform dermatitis, who consented to skin biopsy, were included.

Exclusion criteria: Patients on systemic immunosuppressants or biologics, those with concurrent systemic illness affecting skin, patients who refused biopsy, and inadequate biopsy specimens were excluded.

3.5 Data Collection and Laboratory Procedure

A detailed sociodemographic and clinical history was obtained using a predesigned, pretested proforma. Punch biopsies (3–4 mm) from the active lesional margins were obtained under local anaesthesia, fixed in 10% buffered formalin, processed routinely, and embedded in paraffin. Sections of 3–4 microns thickness were cut and stained with H&E. Serial sections were subjected to IHC using standard streptavidin-biotin-peroxidase complex method for markers Ki-67, CD3, CD4, CD8, and HLA-DR. Scoring was performed by two independent pathologists blinded to clinical information.

3.6 Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 25.0. Descriptive statistics — frequency, percentage, mean, and standard deviation — were computed. Chi-square test and unpaired t-test were applied for comparison between groups. A p-value of <0.05 was considered statistically significant.

3.7 Ethical Considerations

The study was approved by the Institutional Ethics Committee (IEC). Written informed consent was obtained from all participants. Patient confidentiality was maintained throughout the study.

4. RESULTS

4.1 Sociodemographic Profile

A total of 82 patients were enrolled in this study. The sociodemographic characteristics of the study participants are presented in Table 1 below.

Table 1: Sociodemographic Profile of Study Participants (n = 82)

Variable	Category	Psoriasis (n=48)	Psoriasiform Dermatitis (n=34)	Total (n=82)
Age Group (years)	≤ 20	5 (10.4%)	4 (11.8%)	9 (11.0%)
	21 – 40	18 (37.5%)	14 (41.2%)	32 (39.0%)
	41 – 60	19 (39.6%)	12 (35.3%)	31 (37.8%)
	> 60	6 (12.5%)	4 (11.8%)	10 (12.2%)
	Mean Age (years)	42.3 ± 12.6	40.1 ± 13.2	41.4 ± 12.9
Sex	Male	30 (62.5%)	20 (58.8%)	50 (61.0%)
	Female	18 (37.5%)	14 (41.2%)	32 (39.0%)
Residence	Urban	22 (45.8%)	18 (52.9%)	40 (48.8%)
	Rural	26 (54.2%)	16 (47.1%)	42 (51.2%)
Education	Illiterate	10 (20.8%)	8 (23.5%)	18 (22.0%)
	Primary/Secondary	24 (50.0%)	17 (50.0%)	41 (50.0%)
	Graduate & above	14 (29.2%)	9 (26.5%)	23 (28.0%)
Occupation	Labourer/Farmer	18 (37.5%)	12 (35.3%)	30 (36.6%)
	Housewife	10 (20.8%)	9 (26.5%)	19 (23.2%)
	Service/Business	14 (29.2%)	10 (29.4%)	24 (29.3%)
	Student/Others	6 (12.5%)	3 (8.8%)	9 (11.0%)
Family History	Present	22 (45.8%)	6 (17.6%)	28 (34.1%)
of Psoriasis	Absent	26 (54.2%)	28 (82.4%)	54 (65.9%)
Duration of Illness	< 1 year	12 (25.0%)	16 (47.1%)	28 (34.1%)
	1 – 5 years	24 (50.0%)	14 (41.2%)	38 (46.3%)
	> 5 years	12 (25.0%)	4 (11.8%)	16 (19.5%)

4.2 Histopathological Findings on H&E

On H&E examination, psoriasis characteristically showed regular acanthosis, thinning of suprapapillary plates, elongation of rete ridges, confluent parakeratosis, hypogranulosis, dilated tortuous capillaries in the dermal papillae, and Munro's microabscesses in 37 of 48 cases (77.1%). Psoriasiform dermatitis cases showed irregular acanthosis, prominent spongiosis, patchy parakeratosis, and a predominantly lymphohistiocytic perivascular infiltrate without true Munro's microabscesses.

4.3 Immunohistochemical Findings

The IHC results are summarised in Table 2. Ki-67 labelling index (LI) was significantly higher in psoriasis (mean 38.6 ± 6.2%) compared to psoriasiform dermatitis (mean 19.4 ± 5.1%), with a statistically significant difference ($p<0.001$). CD8+ T-lymphocyte predominance was observed in the dermal infiltrate in psoriasis cases (mean CD8:CD4 ratio = 1.8:1), whereas psoriasiform dermatitis demonstrated CD4+ predominance (mean CD8:CD4 ratio = 0.7:1), which was statistically significant ($p<0.01$). HLA-DR expression was strongly positive in 85.4% of psoriasis cases versus 47.1% of psoriasiform dermatitis cases ($p<0.001$).

Table 2: Immunohistochemical Marker Expression in Psoriasis vs. Psoriasiform Dermatitis

IHC Marker	Psoriasis (n=48)	Psoriasiform Dermatitis (n=34)	p-value
Ki-67 LI (Mean ± SD)	38.6 ± 6.2%	19.4 ± 5.1%	< 0.001*
CD3+ (T-lymphocytes)	45/48 (93.8%)	30/34 (88.2%)	0.38 (NS)
CD8+ Predominance	40/48 (83.3%)	11/34 (32.4%)	< 0.001*
CD4+ Predominance	8/48 (16.7%)	23/34 (67.6%)	< 0.001*
HLA-DR Positivity	41/48 (85.4%)	16/34 (47.1%)	< 0.001*

** Statistically significant ($p < 0.05$); NS = Not Significant; LI = Labelling Index; SD = Standard Deviation*

5. DISCUSSION

This cross-sectional study, conducted on 82 patients at a tertiary care centre in West Bengal, offers meaningful insights into the immunohistochemical differentiation of psoriasis from psoriasiform dermatitis — a challenge that remains clinically and pathologically relevant in everyday dermatological practice[5].

The sociodemographic profile of this study revealed a male predominance (61%) in both groups, with the maximum burden of disease in the 21–60 years age group — an economically productive

population segment. This finding is consistent with studies conducted by Dogra et al. and Kaur et al. from North India, which similarly reported a working-age male preponderance in psoriasis patients. The high proportion of rural participants (51.2%) reflects the catchment profile of our tertiary institution and underlines the need for accessible dermatopathological services in non-urban settings[6].

A positive family history of psoriasis was noted significantly more often in psoriasis patients (45.8%) compared to psoriasiform dermatitis (17.6%), affirming the well-established genetic predisposition of psoriasis as a polygenic, immune-mediated disorder. The strong HLA associations in psoriasis, particularly with HLA-Cw6, have been extensively documented and form the biological basis for HLA-DR upregulation seen in our IHC findings[7].

The Ki-67 labelling index emerged as the most powerful discriminatory IHC marker in this study. A mean Ki-67 LI of 38.6% in psoriasis compared to 19.4% in psoriasiform dermatitis ($p<0.001$) reflects the pathologically exaggerated epidermal keratinocyte proliferation driven by cytokines such as IL-17, IL-22, and TNF-alpha in the psoriatic microenvironment. Comparable findings have been reported by Abdel Fattah et al. and other international studies where Ki-67 correlated strongly with disease severity and epidermal turnover rate.

The T-lymphocyte subpopulation analysis proved equally discriminatory. In psoriasis, CD8+ cytotoxic T-lymphocytes dominated the epidermal and dermal inflammatory infiltrate (CD8:CD4 ratio = 1.8:1). This is physiologically consistent with the established immunopathogenesis of psoriasis, wherein CD8+ T-cells in the epidermis and CD4+ Th17/Th1 cells in the dermis jointly perpetuate the inflammatory cascade. In psoriasiform dermatitis, the CD4+ predominance (CD8:CD4 ratio = 0.7:1) suggests a Th2-skewed or mixed immune response, which is characteristic of eczematous and allergic dermatoses. This differential T-cell polarity is of immense diagnostic and therapeutic significance, as biologic therapies targeting IL-17 and IL-23 pathways are relevant primarily in psoriasis and not in psoriasiform conditions[8].

HLA-DR positivity, a marker of antigen presentation and immune activation, was significantly higher in psoriasis (85.4%) than in psoriasiform dermatitis (47.1%). This confirms the hyperactivated antigen-presenting state of keratinocytes in psoriasis and corroborates prior observations from Indian and international literature. The differential expression of HLA-DR may assist pathologists in ambiguous biopsy cases where H&E morphology is insufficient for definitive diagnosis[9].

Interestingly, CD3+ T-lymphocyte infiltration was high in both groups and showed no statistically significant difference (p=0.38), indicating that whilst both conditions are lymphocyte-driven, it is the subtype characterisation (CD4 vs CD8) rather than the total T-cell burden that holds diagnostic value. This is an important caveat for pathologists utilising IHC panels in routine practice[10].

When compared to similar studies, our results align well with the findings of Bovenschen et al. (2011), who demonstrated elevated Ki-67 expression and CD8+ dominance in psoriatic lesions, and with the work of Vocanson et al. on T-cell subtypes in inflammatory dermatoses. Whilst most prior studies were conducted in Western or North Indian populations, our study contributes a regional East Indian perspective, enriching the existing literature base.

This study is not without limitations. The cross-sectional design precludes longitudinal follow-up of IHC changes with treatment. The relatively modest sample size, while statistically adequate for a cross-sectional study, limits subgroup analyses. Additionally, other emerging markers such as p63, involucrin, and STAT3 were not included in this panel and may merit evaluation in future research.

6. CONCLUSION

Immunohistochemical profiling is a reliable, reproducible, and diagnostically superior adjunct to routine H&E staining in differentiating psoriasis from psoriasiform dermatitis. Among the markers evaluated, Ki-67 labelling index, CD8+ T-lymphocyte predominance, and HLA-DR expression demonstrated the strongest discriminatory power between the two entities.

Given the significant clinical and therapeutic implications of this distinction — particularly in the era of targeted biologics — it is strongly recommended that a standardised IHC panel be incorporated into routine dermatopathological evaluation of papulosquamous disorders at tertiary care centres across India. Larger multicentre studies from Eastern India are warranted to validate and further refine these findings.

7.DECLARATION

Conflict of Interest: The authors declare no conflict of interest.

Source of Funding: The study received no external funding. It was conducted as a departmental academic research project.

REFERENCES

1. Griffiths CEM, Barker JNWN. Pathogenesis and clinical features of psoriasis. Lancet. 2007;370(9583):263–271.
2. Dogra S, Yadav S. Psoriasis in India: Prevalence and pattern. Indian J Dermatol Venereol Leprol. 2010;76(6):595–601.
3. Kaur I, Handa S, Kumar B. Natural history of psoriasis: A study from the Indian subcontinent. J Dermatol. 1997;24(4):230–234.
4. Abdel Fattah NSA, Ebrahim AA, El Okda ES. Tissue/serum levels of IL-17 and immunohistochemical expression of Ki-67 in psoriatic patients. J Eur Acad Dermatol Venereol. 2010;24(12):1417–1422.
5. Bovenschen HJ, van de Kerkhof PC, van Erp PEJ, Woestenenk R, Joosten I, Koenen HJPM. Foxp3+ regulatory T cells of psoriasis patients easily differentiate into IL-17A-producing cells and are found in lesional skin. J Invest Dermatol. 2011;131(9):1853–1860.
6. Vocanson M, Hennino A, Rozières A, Poyet G, Nicolas JF. Effector and regulatory mechanisms in allergic contact dermatitis. Allergy. 2009;64(12):1699–1714.
7. Elder JT, Bruce AT, Gudjonsson JE, Johnston A, Stuart PE, Tejasvi T, et al. Molecular dissection of psoriasis: Integrating genetics and biology. J Invest Dermatol. 2010;130(5):1213–1226.
8. Sathishkumar D, George R, Daniel D. Clinicopathological study of psoriasiform dermatoses: A hospital-based study. Indian J Dermatol. 2014;59(6):551–556.
9. Kumar V, Abbas AK, Aster JC. Robbins & Cotran Pathologic Basis of Disease. 10th ed. Philadelphia: Elsevier Saunders; 2021.
10. Weedon D. Weedon's Skin Pathology. 4th ed. Edinburgh: Churchill Livingstone Elsevier; 2016.