

Clinical Phenotype and Disease Severity in Psoriasis with Genotype Correlation: A Cross-Sectional Study from Eastern India

Abhijit Chattopadhyay¹, Neeladri Majumder², Shanwer Harlalka³, Agnik Pal⁴

¹Assistant Professor, Department of Dermatology, Santiniketan Medical College, Bolpur, West Bengal 731204

²Assistant Professor, Department of Pharmacology, Santiniketan Medical College, Bolpur, West Bengal 731204

³Assistant Professor, Department of Pharmacology, Santiniketan Medical College, Bolpur, West Bengal 731204

⁴Professor and Head, Department of Pharmacology, Santiniketan Medical College, Bolpur, West Bengal 731204

Corresponding Author: Prof. (Dr.) Agnik Pal

DOI: <https://doi.org/10.52403/ijshr.20260402>

Received: 02/05/2026; Revised: 10/08/2026; Accepted: 03/10/2026

ABSTRACT

Background: Psoriasis is a chronic immune-mediated inflammatory skin disease with a substantial genetic component. HLA-Cw6 and variants in the endoplasmic reticulum aminopeptidase 1 (ERAP1) gene have been implicated in psoriasis susceptibility and clinical heterogeneity. However, the relationship between these genetic markers and disease severity remains insufficiently characterized in Indian patients.

Objective: To describe the demographic, clinical, and genotypic characteristics of patients with psoriasis and to assess associations of HLA-Cw6 status and ERAP1 rs26653 genotype with Psoriasis Area and Severity Index (PASI) scores and selected clinical features.

Methods: This cross-sectional study included 100 patients with psoriasis. Demographic and clinical variables, including age, age at disease onset, disease duration, sex, clinical phenotype, scalp involvement, nail changes, and arthralgia, were recorded. Disease severity was assessed

using the PASI. HLA-Cw6 status was determined, and ERAP1 rs26653 was genotyped using allele-specific polymerase chain reaction. Continuous and categorical variables were summarized descriptively. The Mann–Whitney U test was used for comparisons of PASI between two groups, while the Kruskal–Wallis test was used for comparisons involving three or more groups. Spearman’s rank correlation was used to assess associations between PASI and continuous variables, and Pearson’s chi-square test was used to evaluate associations between categorical variables. Statistical significance was set at $p < 0.05$.

Results: The mean age of participants was 39.15 ± 15.93 years, and 70% were male. The mean PASI score was 12.13 ± 10.20 . HLA-Cw6 was present in 52% of patients, while ERAP1 rs26653 genotypes were CC in 27%, GC in 41%, and GG in 32%. PASI did not differ significantly according to sex ($p = 0.491$), HLA-Cw6 status ($p = 0.136$), or ERAP1 rs26653 genotype ($p = 0.933$). No significant correlations were observed between PASI and age ($\rho = 0.14$, $p = 0.154$),

age at onset ($p = 0.07$, $p = 0.514$), or disease duration ($p = 0.14$, $p = 0.159$). PASI differed significantly across clinical-phenotype categories (Kruskal–Wallis, $p = 1.23 \times 10^{-9}$).

Conclusion: In this cohort of Indian patients with psoriasis, HLA-Cw6 status and ERAP1 rs26653 genotype were not significantly associated with PASI-defined disease severity. PASI varied significantly according to clinical phenotype. Larger studies with appropriate control groups and multivariable analyses are warranted to further characterize genotype–phenotype relationships in psoriasis.

Keywords: Psoriasis; Psoriasis Area and Severity Index; HLA-C Antigens; Genetic Association Studies; ERAP1; Disease Severity

1. INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory skin disease affecting roughly 2–3% of the world's population, with a lower reported prevalence of 0.44–2.8% in Indian hospital-based series. [1] It arises from an interaction between genetic susceptibility and environmental triggers that drives keratinocyte hyperproliferation and cutaneous inflammation. [2] Among genetic markers, HLA-Cw6 (HLA-C*06:02), located within the PSORS1 locus on chromosome 6p21, is the single strongest and most consistently replicated psoriasis susceptibility allele worldwide, though its carrier frequency varies widely by ethnicity and is generally lower in Asian than in Caucasian populations. [3–6] HLA-Cw6 positivity has been linked with earlier age at onset, guttate morphology and other distinguishing clinical features in several populations. [3,4]

A genome-wide association study additionally identified a statistical interaction between HLA-C and endoplasmic reticulum aminopeptidase-1 (ERAP1), an enzyme that trims peptides for loading onto HLA class I molecules, suggesting that ERAP1 variants may modulate psoriasis risk conferred by HLA-Cw6. [3] The non-synonymous

ERAP1 single-nucleotide polymorphism rs26653 has shown disease association in some cohorts, including a suggested age-restricted effect confined to onset in the second decade of life, [7] while a subsequent meta-analysis of case–control studies found no overall association between rs26653 and psoriasis susceptibility, in contrast to the ERAP1 variants rs30187 and rs27524, which were significantly associated. [8] Whether HLA-Cw6 and ERAP1 rs26653 additionally influence disease severity, as opposed to susceptibility alone, remains inadequately studied, particularly in Indian patients, in whom epidemiological data are largely limited to demographic and morphological description rather than genotype–phenotype correlation. [1,9]

The Psoriasis Area and Severity Index (PASI), which quantifies erythema, induration and desquamation weighted by body-surface-area involvement across four body regions, remains the most widely used and validated instrument for grading psoriasis severity in clinical and research settings. [10] Combining PASI with genotypic data allows exploration of whether individual genetic markers track with more severe or more extensive disease, information that is potentially relevant to risk stratification and to the design of future pharmacogenetic studies.

This study was undertaken to describe the demographic and clinical profile of a cohort of patients with psoriasis and to examine whether HLA-Cw6 status and ERAP1 rs26653 genotype are associated with PASI score, sex, scalp involvement, nail change or arthralgia, and whether PASI varies with age, age at onset, disease duration or clinical phenotype.

2. MATERIALS AND METHODS

2.1 Study design and population

This was a cross-sectional, single-centre analytical study conducted among patients with clinically diagnosed psoriasis attending dermatology outpatient services of a tertiary care medical college hospital. A total of 100

consecutive consenting patients constituted the analysed cohort.

2.2 Data collection

This is an OPD based study where all patients with a clinical diagnosis of psoriasis were included after eliminating those who does not match inclusion and exclusion criteria.

Inclusion Criteria

- a) All the patients presenting with clinical diagnosed cases of psoriasis and histopathological study in doubtful cases and its subsets irrespective of duration of symptom.
- b) Patients willing to participate in the study through a written consent form.

Exclusion Criteria

- a) Patients who were unable or unwilling to provide an informed consent for any reason.
 - b) Seriously ill patients.
 - c) Any other reported autoimmune disease.
- For each patient, age and age at onset of psoriasis were recorded and converted to years from the documented year/month format; disease duration was derived as age minus age at onset. Patients presenting to Dermatology OPD with skin lesion consistent with psoriasis and its subsets were thoroughly examined and a proper history was taken and punch biopsy of the suspected lesion was sent for histopathological examination and findings were meticulously recorded in case record form. 5 ml of patient's blood were taken for ERAP1 gene typing by allele specific PCR. DNA isolation from blood samples were done and allele specific PCR were performed for ERAP1 (rs26653) typing. Appropriate routine laboratory tests were done. A punch biopsy was taken only from suspected lesion under aseptic precaution to be sent for histopathological examination. PASI score of severity was done for each patient.

Study Tool

- a) Data of all patients attending Dermatology OPD.
- b) Blood sampling materials.

c) PCR.

The purpose of PCR is to make a huge number of copies of gene. An allele specific PCR method was developed to detect single nucleotide polymorphism (SNP) in human DNA sample. There are three major steps in a PCR, which are repeated for 30 or 40 cycles.

During the denaturation, the double strand melts and opens to single stranded DNA. During annealing the primer jiggle around, caused by Brownian motion. Ionic bonds are constantly formed and broken between the single stranded primer and the single stranded template. The more stable bonds last a little bit longer and on that little piece of double stranded DNA (template and primer), the polymerase can attach and starts coping the template. Extension was done at 72 degree centigrade. The bases (complementary to the template) are coupled to primer on the 3' side (the polymerase adds dNTPs from 5' to 3', reading the template from 3' to 5' side, bases are added complementary to template). The PCR results in selective amplification of a chosen region of a DNA molecule. Since both strands are copied during PCR, there is an exponential increase in the number of copies of genes.

In this study, SNP rs26653 in ERAP1 gene was selected. SNP was genotyped by PCR using allele specific primer (designed using primer 3 software). The amplicons were analyzed by electrophoresis on 1% agarose gel in 1X TBE buffer against a DNA molecular size marker and bands were then visualized in a gel documentation system after staining with ethidium bromide (0.5 microgram/ml).

Sex, clinical diagnosis/phenotype (generalized plaque, localized plaque, guttate, generalized pustular, localized pustular, and generalized plaque with arthritis), scalp involvement, presence of any nail change and presence of arthralgia were recorded on clinical examination. Disease severity was quantified using the PASI Index. HLA-Cw6 status (present/absent) and ERAP1 rs26653 genotype (CC, GC, GG)

were determined and recorded for each patient.

2.3 Statistical analysis

Continuous variables were summarized using mean $\pm$ standard deviation (SD) and median, with ranges reported where appropriate. Categorical variables were summarized as frequencies and percentages. The distribution of PASI scores was compared between two independent groups using the Mann–Whitney U test, including comparisons according to sex and HLA-Cw6 status. For comparisons involving three or more independent groups, the Kruskal–Wallis test was used, including comparisons across ERAP1 rs26653 genotype groups and clinical-phenotype categories.

Associations between PASI and continuous variables, including age, age at onset, and disease duration, were assessed using Spearman’s rank correlation coefficient (ρ). Associations between categorical variables were evaluated using Pearson’s chi-square test, including the relationships of HLA-Cw6 status and ERAP1 rs26653 genotype with sex, scalp involvement, nail changes, and arthralgia, as well as the joint distribution of HLA-Cw6 status and ERAP1 rs26653 genotype. All statistical tests were two-sided,

and a p-value <0.05 was considered statistically significant. As this was an exploratory, single-cohort analysis without a healthy control group, the statistical analyses were interpreted as assessing within-cohort genotype–phenotype associations rather than disease susceptibility. All the analyses were done at department of Pharmacology.

3. RESULTS

3.1 Demographic and clinical profile

The cohort comprised 100 patients with a mean age of 39.15 ± 15.93 years (median 40.0; range 7.5–80 years) and a male predominance (70 men, 30 women; 70%). Mean age at onset was 32.06 ± 15.52 years (median 30.5; range 4.5–77 years), and mean disease duration was 7.09 ± 7.69 years (median 4.5; range 0–40 years). The largest age group was 21–40 years (41%), followed by 41–60 years (39%), 0–20 years (13%) and older than 60 years (7%).

Mean PASI was 12.13 ± 10.20 (median 9.00; range 1.2–45.6), indicating substantial inter-individual variation in severity. Scalp involvement was present in 60% of patients, making it the most frequent extra-plaque clinical feature, followed by nail change in 28% and arthralgia in 16%. Table 1 summarises the descriptive results.

Table 1: Descriptive statistics of demographic, clinical and genotypic variables (n= 100).

Variable	Result (mean $\pm$ SD; median)	Range	Notes
Age (years)	39.15 ± 15.93 ; 40.0	7.5–80	—
Age at onset (years)	32.06 ± 15.52 ; 30.5	4.5–77	—
Disease duration (years)	7.09 ± 7.69 ; 4.5	0–40	—
PASI	12.13 ± 10.20 ; 9.00	1.2–45.6	—
Sex	Male 70 (70%); Female 30 (30%)	—	Male predominance
HLA-Cw6	Present 52 (52%); Absent 48 (48%)	—	Near-even split
ERAP1 rs26653	CC 27 (27%); GC 41 (41%); GG 32 (32%)	—	GC most frequent
Scalp involvement	60 (60%)	—	Most common feature
Any nail change	28 (28%)	—	—
Arthralgia	16 (16%)	—	—

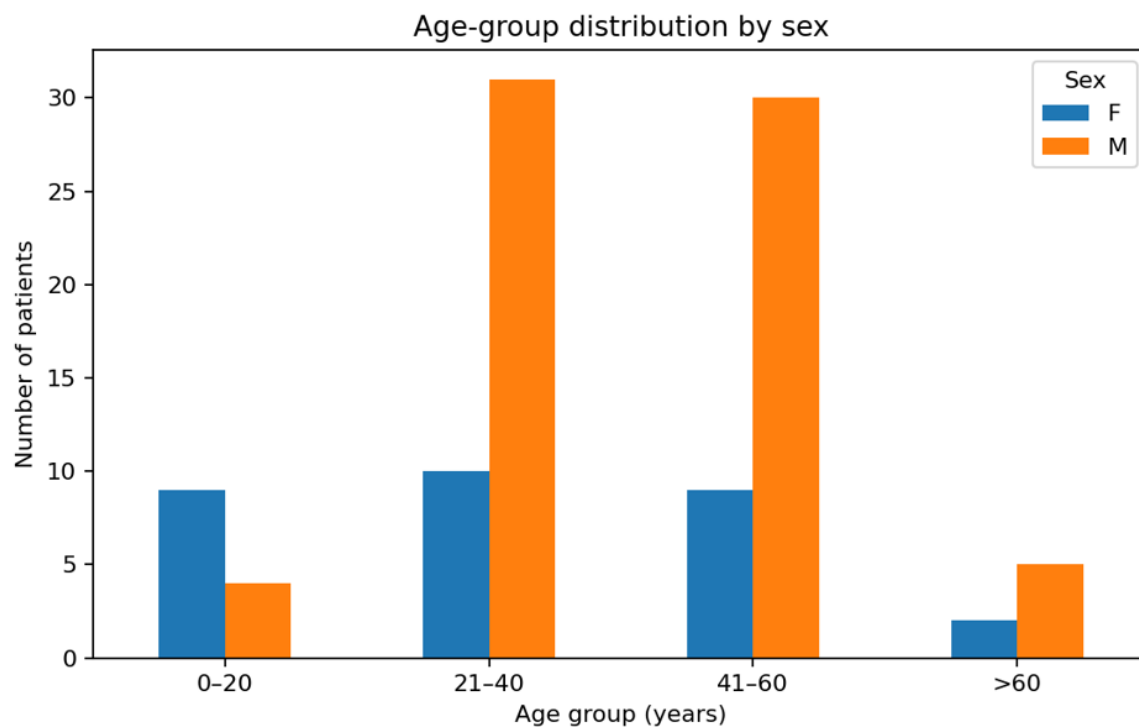


Figure 1: Age and sex distribution

3.2 Genotype distribution

HLA-Cw6 was present in 52 patients (52%) and absent in 48 (48%), a near-even distribution. For ERAP1 rs26653, genotype frequencies were CC 27%, GC 41% and GG 32%, with the heterozygous GC genotype being most common.

3.3 PASI in relation to demographic and genotypic variables

Table 2: PASI and gender differences.

Sex	n	Mean	SD	Median
M	70	12.49	10.04	9.00
F	30	11.28	10.68	9.15

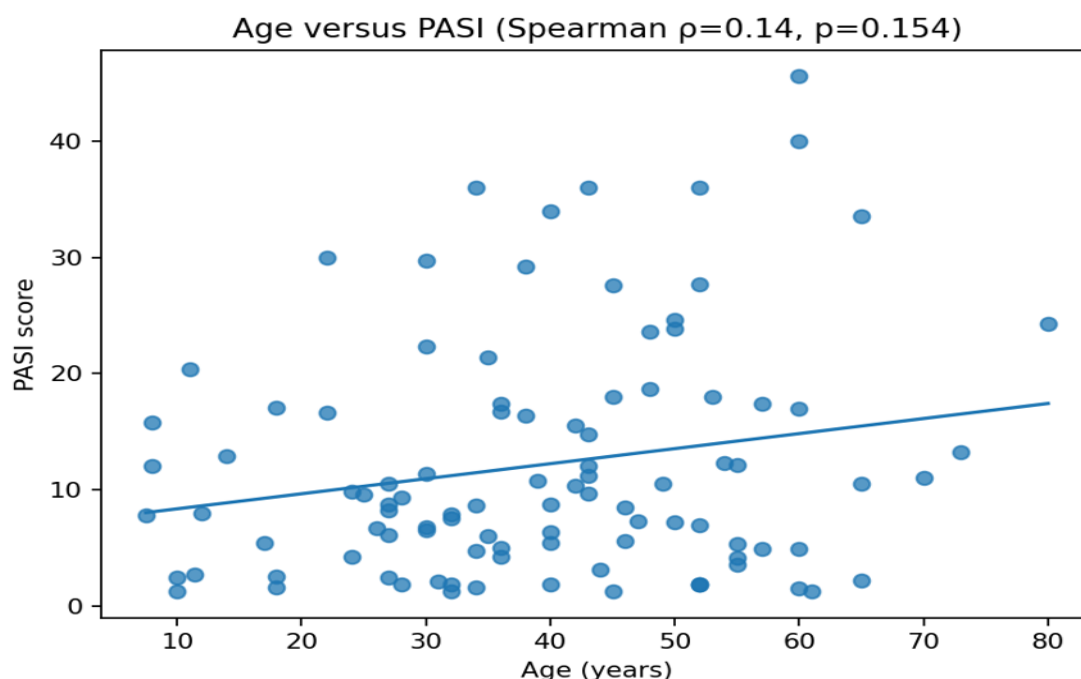


Figure 2: Correlation of PASI with age.

PASI did not differ significantly between men (mean 12.49 ± 10.04) and women (mean 11.28 ± 10.68) (Mann–Whitney U, $p = 0.491$). There was no significant correlation between PASI and age (Spearman $\rho = 0.14$, $p = 0.154$), age at onset ($\rho = 0.07$, $p = 0.514$) or disease duration ($\rho = 0.14$, $p = 0.159$). PASI did not differ significantly by HLA-Cw6 status ($p = 0.136$) or across ERAP1 rs26653 genotypes (Kruskal–Wallis, $p = 0.933$).

3.4 Genotype associations with clinical features

HLA-Cw6 status was not significantly associated with sex ($p = 0.694$), scalp involvement ($p = 0.465$), nail change ($p = 1.000$) or arthralgia ($p = 1.000$). Similarly, ERAP1 rs26653 genotype was not significantly associated with sex ($p = 0.428$), arthralgia ($p = 0.207$), scalp involvement ($p = 0.115$) or nail change ($p = 0.281$). The joint distribution of HLA-Cw6 and ERAP1 genotype showed a non-significant trend toward association ($p = 0.089$).

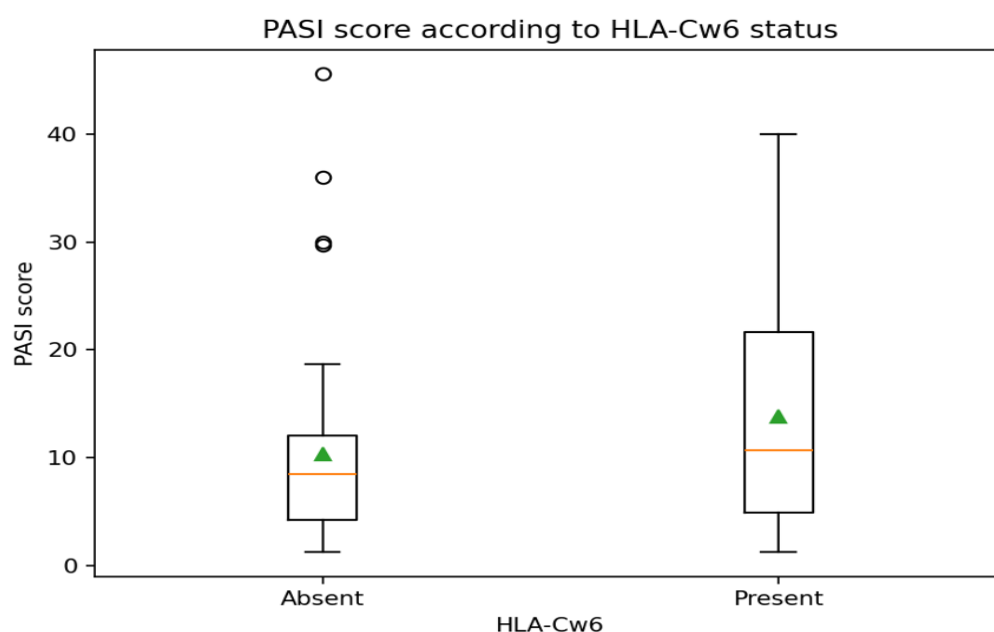


Figure 3: Correlation of PASI with HLA Cw6 status

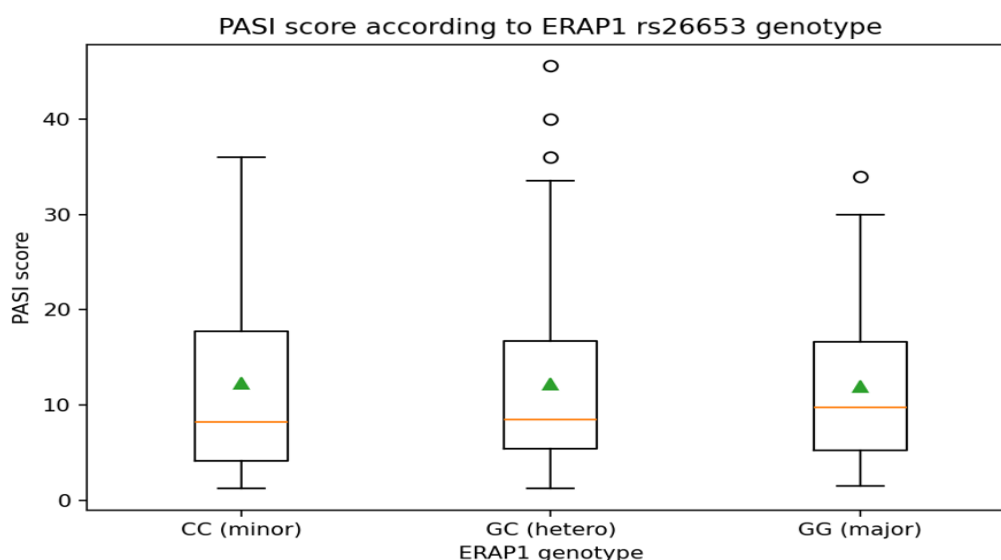


Figure 4: Correlation of PASI with ERAP1 rs26653 genotype

3.5 PASI and clinical phenotype

Generalized plaque psoriasis was the predominant phenotype (66%), followed by localized plaque psoriasis (23%), guttate psoriasis (4%), generalized pustular psoriasis (3%), localized pustular psoriasis (2%) and generalized plaque disease with arthritis (2%). PASI differed significantly across

these clinical-phenotype categories (Kruskal–Wallis, $p = 1.23 \times 10^{-9}$). Because several categories contained very few patients, this finding should be interpreted cautiously, and confirmatory post-hoc pairwise testing with correction for multiplicity is warranted before it is treated as a robust conclusion.

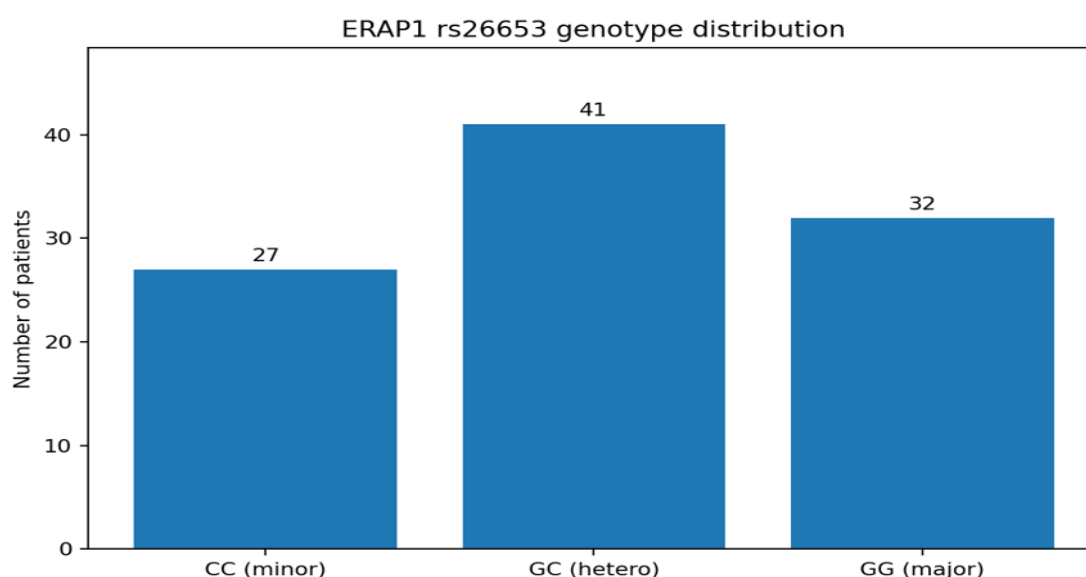


Figure 5: ERAP1 rs26653 genotype distribution

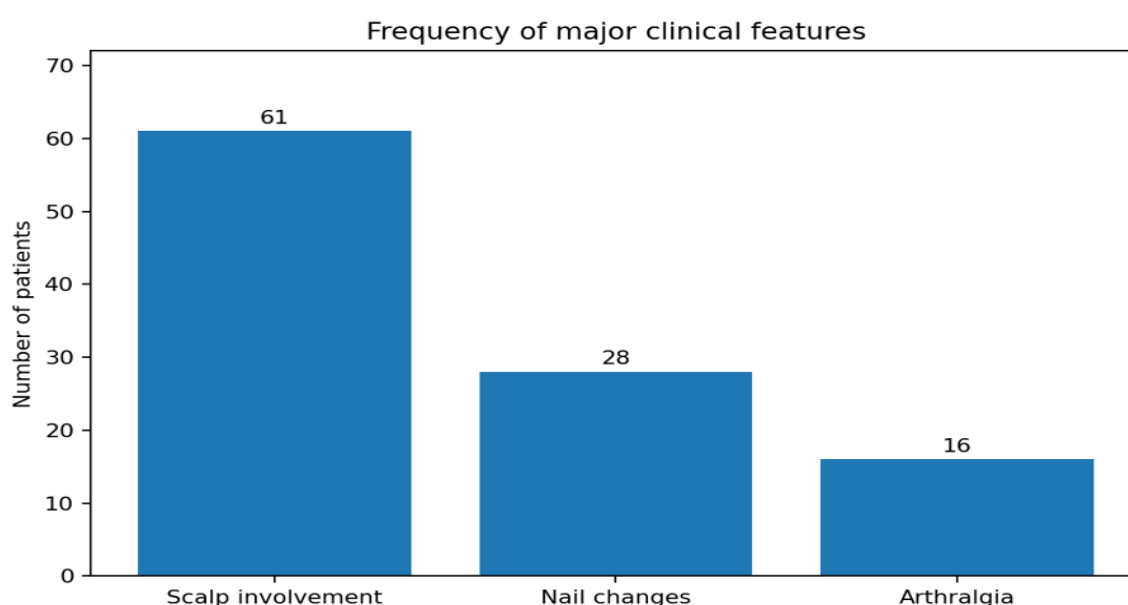


Figure 6: Frequency of major clinical features

4. DISCUSSION

In this cohort of 100 Indian patients with psoriasis, the demographic profile - a mean age in the fourth decade, male predominance, and a mean age at onset in the early thirties -

closely mirrors previous studies, which have consistently reported male preponderance and onset predominantly in the third and fourth decades of life. [9-12] In the present study, out of 100 psoriasis patients, 30 (30%)

female and 70 male (70%) reported to us. This may be due to females from far away village get lesser opportunity to reach tertiary care centre as compared to male counterpart. A higher prevalence in male has been noted in most Indian studies. A study done in India on North population by Bedi showed males are most commonly affected than females.

[9]

The prevalence of scalp involvement (60%) and nail change (28%) is likewise broadly consistent with earlier Indian reports describing scalp as one of the most frequently affected sites and nail changes present in roughly a quarter to half of patients at presentation. [12] The predominance of generalized plaque disease (66%) followed by localized plaque disease is also concordant with the well-established observation that chronic plaque psoriasis constitutes the great majority of cases in Indian and other populations. [10]

In our study out of 100 patients, 21 patients were in service, 14 farmer, 19 patients were involved in different businesses, 15 patients were student, 19 were homemakers, 5 patients were drivers, 3 patients were unemployed, 3 patients were retired, and 1 patient was priest. Thus, among men, service job was the most common occupation and most of the females were housewives. In our study, there were 46 patients (46%) of rural background and 54 patients (54%) of urban. A study from Norway by Braathen LR et al. found a higher prevalence of psoriasis among rural females. [13] Another study done by Jesić S et al. in West Serbia found equal participation of urban and rural population.

[14]

In present study, there were only 7 patients with family history of psoriasis. A study by Farber EM et al. reported 36% of familial incidence in psoriasis. [15] However, most Indian studies reported lower familial incidence similar to our study. [9]

PASI score ranges from 0 to maximum 72. Mean PASI score in our study was 12.13 with a median of 9. The minimum PASI score was 1.2 and maximum was 45.6; the standard deviation was 10.2 with standard

error of mean 1.02. Our results vary from other studies as PASI score varies with disease severity which varies with the type of study population.

In our study, no significant PASI difference was found by HLA-Cw6 status ($p = 0.136$). HLA-Cw6 was not associated with sex ($p = 0.694$), scalp involvement ($p = 0.465$), nail changes ($p = 1.000$), or arthralgia ($p = 1.000$). ERAP1 rs26653 genotype was not associated with PASI (Kruskal–Wallis $p = 0.933$), sex ($p = 0.428$), arthralgia ($p = 0.207$), scalp involvement ($p = 0.115$), or nail changes ($p = 0.281$). The HLA-Cw6 $\times$ ERAP1 distribution showed a non-significant trend ($\chi^2 p = 0.089$). The HLA-Cw6 carrier frequency of 52% observed here sits within the range reported for Asian psoriasis cohorts, which is generally lower than the 55–80% reported in Caucasian series but overlaps with several East and South Asian estimates. [3,6] HLA-Cw6 has been repeatedly identified as the strongest psoriasis susceptibility allele and has been associated in other cohorts with earlier disease onset and a higher frequency of guttate morphology. [3-5] In the present cohort, however, HLA-Cw6 status was not significantly associated with PASI severity, sex, scalp involvement, nail change or arthralgia. This is consistent with the broader literature distinction between susceptibility and severity: HLA-Cw6 is primarily a marker of disease risk and of certain phenotypic tendencies (notably early onset and guttate presentation) rather than a consistent determinant of quantitative severity once disease is established, and several studies examining HLA-Cw6 in relation to treatment response and severity have likewise reported divergent or null findings. [4,5]

For ERAP1 rs26653, the genotype distribution in this cohort (CC 27%, GC 41%, GG 32%) did not show significant association with PASI, sex, or the examined clinical features, and the joint HLA-Cw6 $\times$ ERAP1 distribution showed only a non-significant trend. This finding is compatible with a meta-analysis of case–control studies that found no significant overall association

between rs26653 and psoriasis susceptibility, in contrast to significant associations for the ERAP1 variants rs30187 and rs27524. [8] It also aligns with reports suggesting that any ERAP1-related genetic effect in psoriasis may be age-of-onset-restricted, being most apparent in patients with onset in the second decade of life, a subgroup that was not separately powered in the present analysis. [7] Because the present study did not include a healthy control group, it cannot address susceptibility to psoriasis itself; the negative genotype–severity associations observed here should be interpreted as evidence against a severity-modifying role for HLA-Cw6 and ERAP1 rs26653 in this cohort, not as evidence against their established role in disease susceptibility.

We found 28 patients (28%) with nail involvement. This finding is almost similar to a study conducted by Narayan R et al. showing 31% nail involvement when disease is not associated with arthritis and 81% of patients with associated psoriatic arthritis. [16] A study by Schons KR et al. showed 46.1% nail involvement in psoriasis. [17] In our study, 61% patients had scalp involvement. This is in slight discordance with a study conducted by Frez ML et al. showing 75-90% have scalp involvement in psoriasis. [18] In present study, 16% patients had arthralgia. A study conducted by Radtke MA et al. showed 19% of psoriatic patients having arthritis, in which 84.9% had arthralgia. [19] A study, conducted by Lysell et al. on Swedish population, where association of ERAP1 (rs26653) was confined to cases with age of disease onset between 10-20 years; this study also showed there was borderline association for ERAP1 (rs26653) with age of disease onset between 21-40 years. [7] Nevertheless, Oostveen et al. did not find any such association between age of onset and ERAP1 in the Dutch pediatric psoriasis patients. [20]

In our study, we further divided the psoriasis patients according to age of onset into two groups, early age of onset (≤ 40 years) referred to as Type I and late age of onset (>40 years) referred to Type II psoriasis.

There were 75 patients of Type I and 25 patients belong to Type II psoriasis in our study. We could not find any significant association with ERAP1 SNP (rs26653) at genotype level with any types of psoriasis (Type I cases P value: 0.3737, and Type II cases P value: 0.8414). Similarly, allele frequency distribution also showed no significant association with any type of psoriasis (Type I cases P value: 0.2264, Type II cases P value: 0.5655). This is in contrast to a study done on Chinese Han population, which showed ERAP1 was associated with Type I (early onset) psoriasis. [21]

The absence of correlation between PASI and age, age at onset or disease duration suggests that, in this cohort, chronological and disease-duration variables were not major drivers of instantaneous severity, which is plausible given that PASI reflects current activity rather than cumulative disease burden. In contrast, PASI varied strongly across clinical-phenotype categories, with pustular and arthritis-associated phenotypes generally representing more severe disease. This is biologically expected, since generalized pustular psoriasis and psoriatic disease with joint involvement are recognised as more severe, less common variants; however, the very small numbers in several phenotype subgroups (as few as 2–4 patients) limit the precision and generalisability of this comparison, and multiplicity-corrected post-hoc testing is required before this result can be considered definitive.

4.1 Limitations

This study has several limitations. First, the cross-sectional, single-centre design with a modest sample size ($n = 100$) limits statistical power, particularly for subgroup comparisons involving uncommon phenotypes. Second, the absence of a healthy control group precludes any inference about genetic susceptibility to psoriasis, restricting conclusions to within-cohort genotype–phenotype correlation. Third, potential confounders such as body-mass index, smoking, alcohol use, comorbid metabolic

syndrome and treatment history were not incorporated into a multivariable model. Future work should include age-, sex- and geographically matched controls, larger sample sizes to adequately power comparisons across phenotype subgroups, multivariable regression of PASI adjusting for demographic and clinical covariates, and reporting of effect sizes with 95% confidence intervals alongside p-values.

5. CONCLUSION

In this cross-sectional cohort of Eastern Indian patients with psoriasis, HLA-Cw6 and ERAP1 rs26653 genotypes were common but were not significantly associated with PASI severity or with sex, scalp involvement, nail change or arthralgia. PASI was not correlated with age, age at onset or disease duration but differed significantly across clinical-phenotype categories, reflecting the greater severity of pustular and arthritis-associated disease, albeit in small subgroups. These findings suggest that, within an established psoriasis cohort, HLA-Cw6 and ERAP1 rs26653 may be more relevant to disease susceptibility and early clinical phenotype than to quantitative severity, and they underscore the need for larger, case-control genotype-phenotype studies incorporating multivariable analysis to clarify these relationships in Indian patients.

Declaration by Authors

Ethical Approval: Approved

Acknowledgement: None

Source of Funding: None

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

Creative Commons Attribution License 4.0

This is an open-access article published under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

<https://creativecommons.org/licenses/by/4.0>

REFERENCES

1. Dogra S, Mahajan R. Psoriasis: epidemiology, clinical features, comorbidities, and clinical scoring. Indian

- Dermatol Online J. 2016;7(6):471-480. doi:10.4103/2229-5178.193906.
2. Rendon A, Schäkel K. Psoriasis pathogenesis and treatment. Int J Mol Sci. 2019;20(6):1475. doi:10.3390/ijms20061475.
3. Strange A, Capon F, Spencer CC, Knight J, Weale ME, Allen MH, et al. Genome-wide association study identifies new psoriasis susceptibility loci and an interaction between HLA-C and ERAP1. Nat Genet. 2010;42(11):985-990. doi:10.1038/ng.694.
4. Guðjónsson JE, Kárasón A, Antonsdóttir AA, Rúnarsdóttir EH, Gulcher JR, Stefánsson K, et al. HLA-Cw6-positive and HLA-Cw6-negative patients with psoriasis vulgaris have distinct clinical features. J Invest Dermatol. 2002;118(2):362-365. doi:10.1046/j.0022-202x.2001.01656.x.
5. Fan X, Yang S, Sun LD, Liang YH, Gao M, Zhang KY, et al. Comparison of clinical features of HLA-Cw*0602-positive and -negative psoriasis patients in a Han Chinese population. Acta Derm Venereol. 2007;87(4):335-340. doi:10.2340/00015555-0253.
6. Pham NTU, Nguyen TV, Nguyen HT. HLA-Cw6 increases the risk of psoriasis and early onset before twenty-seven years of age among the Vietnamese population. Dermatol Rep. 2023;16(2):9854. doi:10.4081/dr.2023.9854.
7. Lysell J, Padyukov L, Kockum I, Nikamo P, Ståhle M. Genetic association with ERAP1 in psoriasis is confined to disease onset after puberty and not dependent on HLA-C*06. J Invest Dermatol. 2013;133(2):411-417. doi:10.1038/jid.2012.280.
8. Wu X, Zhao Z. Associations between ERAP1 gene polymorphisms and psoriasis susceptibility: a meta-analysis of case-control studies. Biomed Res Int. 2021;2021:5515868. doi:10.1155/2021/5515868.
9. Bedi TR. Clinical profile of psoriasis in North India. Indian J Dermatol Venereol Leprol. 1995;61(4):202-205.
10. Fredriksson T, Pettersson U. Severe psoriasis—oral therapy with a new retinoid. Dermatologica. 1978;157(4):238-244. doi:10.1159/000250839.
11. Henseler T, Christophers E. Psoriasis of early and late onset: characterization of two types of psoriasis vulgaris. J Am Acad Dermatol. 1985;13(3):450-456. doi:10.1016/S0190-9622(85)70188-0.

12. Kaur I, Kumar B, Sharma KV, Kaur S. Epidemiology of psoriasis in a clinic from North India. *Indian J Dermatol Venereol Leprol.* 1986;52(4):208-212.
13. Braathen LR, Botten G, Bjerkedal T. Prevalence of psoriasis in Norway. *Acta Derm Venereol Suppl (Stockh).* 1989;142:5-8.
14. Jesić S, Hosovski E. Prevalence of psoriasis vulgaris in West Serbia. *Srp Arh Celok Lek.* 1994;122(3-4):61-64.
15. Farber EM, Nall ML. The natural history of psoriasis in 5,600 patients. *Dermatologica.* 1974;148(1):1-18. doi:10.1159/000251595.
16. Narayan R, Kar HK, Gautam RK, Jain RK, Bagga GR. Psoriasis and nail involvement. *Indian J Dermatol Venereol Leprol.* 1995;61(3):182-183.
17. Schons KRR, Beber AAC, Beck MO, Monticelo OA. Nail involvement in adult patients with plaque-type psoriasis: prevalence and clinical features. *An Bras Dermatol.* 2015;90(3):314-319. doi:10.1590/abd1806-4841.20153736.
18. Frez MLF, Asawanonda P, Gunasekara C, Koh C, Loo S, Oon HH, et al. Recommendations for a patient-centered approach to the assessment and treatment of scalp psoriasis: a consensus statement from the Asia Scalp Psoriasis Study Group. *J Dermatolog Treat.* 2014;25(1):38-45. doi:10.3109/09546634.2012.742176.
19. Radtke MA, Reich K, Blome C, Rustenbach S, Augustin M. Prevalence and clinical features of psoriatic arthritis and joint complaints in 2009 patients with psoriasis: results of a German national survey. *J Eur Acad Dermatol Venereol.* 2009;23(6):683-691. doi:10.1111/j.1468-3083.2009.03159.x.
20. Oostveen AM, Bergboer JGM, van de Kerkhof PCM, Zeeuwen PLJM, de Jong EMGJ, Schalkwijk J, et al. Genotype-phenotype correlations in a prospective cohort study of paediatric plaque psoriasis: lack of correlation between HLA-C*06 and family history of psoriasis. *Acta Derm Venereol.* 2014;94(6):667-671. doi:10.2340/00015555-1810.
21. Sun LD, Cheng H, Wang ZX, Zhang AP, Wang PG, Xu JH, et al. Association analyses identify six new psoriasis susceptibility loci in the Chinese population. *Nat Genet.* 2010;42(11):1005-1009. doi:10.1038/ng.690.

How to cite this article: Chattopadhyay A, Majumder N, Harlalka S, Pal A. Clinical phenotype and disease severity in psoriasis with genotype correlation: a cross-sectional study from Eastern India *Int. J. Sci. Healthc. Res.* 2026; 11(4): 8-18. doi: [10.52403/ijshr.20260402](https://doi.org/10.52403/ijshr.20260402)
