Evaluation Of Card14 Gene (rs34367357)Polymorphism in Egyptian Psoriatic patients

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Abstract

Background: Psoriasis is a chronic, immune-mediated skin disease affecting millions, marked by abnormal keratinocyte proliferation. Genetic factors, including the CARD14 gene, epigenetic mechanisms, alongside environmental triggers, drive immune hyperactivation and epidermal barrier dysfunction, influencing disease susceptibility and severity.

Objectives: To assess the role of CARD14 gene(rs34367357) polymorphism in Egyptian psoriatic patients.

Patients and methods: This cross-sectional study at Qena University Hospital included 40 adults with mild psoriasis vulgaris and 40 age and sex matched healthy controls, excluding individuals with other autoimmune disorders, pregnancy, or genetic immune conditions. Clinical assessment recorded demographics, disease history, BMI, and psoriasis severity using the PASI score (<10 defined as mild). Blood samples were collected for DNA extraction, and CARD14 rs34367357 polymorphism was genotyped using a TaqMan assay with real-time PCR.

Results: In mild psoriasis patients, demographics and lifestyle factors were similar to controls, but family history was significantly associated with disease (30% vs 0%, p<0.001). Mean PASI score was 3.53±0.78, with disease duration 2.34±1.39 years. Lesions primarily affected upper extremities (91.7%) and lower extremities (68.3%). The CARD14 rs34367357 G allele and AG/GG genotypes were significantly more frequent in cases than controls(p<0.001), and GG carriers had higher PASI scores(p<0.001). BMI positively correlated with PASI score(p=0.035), and family history also predicted higher severity(p=0.015), while other parameters and genotype showed no significant effect on disease course.

Conclusion: CARD14 rs34367357 G allele and GG genotype are key genetic risk factors for Egyptian psoriasis, associated with greater disease severity. Family history and higher BMI further exacerbate severity, emphasizing the combined influence of genetic and systemic factors.

Keywords: Psoriasis vulgaris; CARD14 gene; Genetic polymorphism

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Introduction

Psoriasis is a lifelong and clinically heterogeneous skin disease that manifests in several forms, including plaque, flexural, guttate, pustular, and erythrodermic types. Globally, it affects nearly 60 million individuals. The disease is considered an immune-mediated inflammatory disorder with a strong genetic component that contributes significantly to its development and persistence (**Raharjaetal.,2021**).

In addition to genetic predisposition, epigenetic mechanisms also play an essential role in regulating gene expression associated with psoriasis. These mechanisms include DNA methylation, histone modifications, and non-coding RNAs, all of which influence immune responses and keratinocyte behavior. Environmental factors such as stress, infections, and lifestyle habits can modify these epigenetic processes, thereby altering disease susceptibility and severity. Consequently, epigenetic regulation is critical for proper keratinocyte differentiation, and disturbances in this regulation can contribute to psoriatic pathogenesis. The interaction between genetic susceptibility and epigenetic modifications is therefore fundamental to understanding psoriasis, as together they contribute to the epidermal barrier dysfunction and immune hyperactivation characteristic of the disease (**Moltrasio et al., 2022**).

A major hallmark of psoriasis is the abnormal proliferation and differentiation of keratinocytes (KCs). Among the genes associated with increased activity in psoriasis is CARD14, which is highly expressed in keratinocytes located in the skin epidermis and encodes an activator of nuclear factor kappa-B (NF-κB). Early studies suggested that CARD14 expression was mainly limited to the placenta; however, later research demonstrated that it is predominantly expressed in mucosal tissues as well, highlighting its broader biological relevance in inflammatory skin conditions (**Singh et al., 2024**).

This study aimed to assess the role of the Card14 gene (rs34367357) polymorphism in Egyptian psoriatic patients.

Patients and methods

Study Design and Participants

This cross-sectional study included patients attending the dermatology outpatient clinics who fulfilled the required inclusion and exclusion criteria. Eligible participants were adults aged 18 years or older diagnosed with mild psoriasis vulgaris according to the Psoriasis Area and Severity Index (PASI) score **(Manchanda et al., 2023).** Patients with other autoimmune or inflammatory diseases, such as rheumatoid arthritis or systemic lupus erythematosus, were excluded. Pregnant or lactating women and individuals with known genetic disorders affecting immune system function were also excluded. The study was conducted at Qena University Hospital, Department of Dermatology, Venereology and Andrology.

Sample size calculation:

The sample size was estimated based on a previous study that reported a significant association between CARD14 gene polymorphisms and psoriasis with a moderate to large effect size **(Suleman et al., 2022).** Using the standard formula for case–control genetic association studies and assuming a two-sided α level of 0,05 and 80% statistical power, the frequency of the G allele was reported to be approximately 65% in cases and 12.5% in controls.

The sample size was calculated based on the following formula:

$$n = [(Z\alpha/2 + Z\beta)^2 \times (p1(1-p1) + p2(1-p2))] / (p1 - p2)^2$$

Where:

- $Z_{\alpha/2} = 1.96$ for a 95% confidence level
- $Z_{\beta} = 0.84$ for 80% power
- p1 = proportion of the risk allele among cases = 65%
- p2 = proportion of the risk allele among controls = 12.5%.

Substituting these values into the equation:

- $n = [(1.96 + 0.84)^2 \times (0.65\times0.35 + 0.125\times0.875)] / (0.65 - 0.125)^2$
- $n = [7.84 \times (0.2275 + 0.1094)] / (0.525)^2$
- $n = [7.84 \times 0.3369] / 0.2756$
- $n \approx 9.60$

Considering the need for adequate statistical power, adjustment for variability and improvement of the reliability of results, the sample size was increased to 40 subjects per group. Therefore, a total of 80 participants were included in the study.

Ethical Consideration:

Written informed consent was obtained from all participants before enrollment, and the study was conducted in accordance with the Declaration of Helsinki. Ethical approval was granted by the Institutional Review Board of the Faculty of Medicine, Qena University (approval code: SVU-MED-DVA021-1-24-12-1016).

Clinical Assessment and Data Collection

All participants underwent detailed history taking and clinical examination. Personal data included age, sex, occupation, marital status, and special habits. Disease-specific history focused on age of onset, duration, course, progression, lesion distribution, previous treatments, and family history of psoriasis or other autoimmune disorders. The cclinical examination included body mass index (BMI) measurement and a complete dermatological examination to determine the clinical type and severity of psoriasis using the PASI score.

PASI Scoring System

Fredriksson and Pettersson first used the PASI score in 1978 to evaluate psoriasis severity **(Manchanda et al., 2023).** The score ranges from 0 to 72 and is calculated through assessment of both lesion severity and body surface area (BSA) involvement. According to clinical guidelines, mild psoriasis is defined as PASI < 10 **(Menter et al., 2019).**

The body is divided into four regions: head (h), upper limbs (u), trunk (t), and lower limbs (l), representing 10%, 20%, 30%, and 40% of the total body surface area respectively (**Fredriksson & Pettersson, 1978**). In each region, lesions are evaluated based on erythema (E), induration (I), and scaling or desquamation (D). Each parameter is graded from 0 to 4 (0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = very severe). The affected area is also scored from 1 to 6 according to the percentage of involvement: 1 (<10%), 2 (10–29%), 3 (30–49%), 4 (50–69%), 5 (70–89%), and 6 (>90%). The final PASI score is calculated using the formula:

PASI = 0.1 (Eh + Ih + Dh) Ah + 0.2 (Eu + Iu + Du) Au + 0.3 (Et + It + Dt) At + 0.4 (El + Il + Dl) Al. In this study, body surface area affected by psoriasis was estimated using the patient's palm as approximately 1% of total skin surface. area

Control Group

The control group consisted of healthy individuals without any clinical signs or family history of psoriasis or autoimmune diseases such as diabetes mellitus, rheumatoid arthritis, or systemic lupus erythematosus. Controls were matched with patients according to age and gender to ensure comparability between groups.

Laboratory Procedures

A total of 40 patients with mild psoriasis and 40 healthy controls were included. For genetic analysis, 2 mL of venous blood was collected from each participant under sterile conditions in EDTA vacutainer tubes and stored at −80°C for later analysis of CARD14 gene (rs34367357) polymorphism.

DNA Extraction and Genotyping

Genomic DNA was extracted from whole blood samples using the QIAamp DNA Blood Mini Kit (QIAGEN, Germany) according to the manufacturer's instructions. Approximately 200 µL of whole blood was lysed with QIAGEN protease and the buffer AL, incubated at 56°C for 10 minutes, followed by ethanol addition and purification using QIAamp spin columns. The columns were washed sequentially with Buffer AW1 and Buffer AW2, and DNA was finally eluted in Buffer AE or RNase-free water and stored at −80°C. DNA concentration and purity were assessed using a Nanodrop spectrophotometer.

Genotyping of the CARD14 rs34367357 (A/G) polymorphism was performed using a TaqMan SNP Genotyping Assay on a 7500 Fast Real-Time PCR System (Applied Biosystems, USA). The PCR reaction volume was 20 µL, containing 10 µL TaqMan Genotyping Master Mix (2×), 5 µL genomic DNA (10 ng/µL), 0.3 µL SNP assay, and 4.7 µL RNase-free water. Thermal cycling involved initial activation at 95°C for 7 minutes, followed by 40 cycles of denaturation at 95°C

for 20 seconds and annealing/extension at 59°C for 60 seconds. Allelic discrimination was performed using the system software to determine the distribution of the studied polymorphism.

Statistical analysis

Numerical variables are represented as mean and standard deviation (SD), while categorical variables are presented as frequencies and percentages. Comparisons between two groups were performed using the independent samples t-test for normally distributed continuous variables and the chi-square test (χ^2) for categorical variables. For comparisons across genotypes, one-way ANOVA was applied for continuous variables. Associations between continuous variables were assessed using Pearson correlation coefficients. Multiple linear regression analysis was used to identify independent predictors of disease severity (PASI score). A p-value < 0.05 was considered statistically significant.

Results

Demographic and lifestyle characteristics were comparable between mild psoriasis cases and controls, with no significant differences in age, BMI, sex, occupation, marital status, or smoking status (all $p > 0.05$). However, family history of psoriasis was significantly associated with the disease, being present in 30% of patients and absent in controls ($p < 0.001$) **(Table 1).**

Among 40 patients with mild psoriasis, the mean PASI score was 3.53 ± 0.78, with a disease duration of 2.34 ± 1.39 years. Lesions most frequently involved the upper (91.7%) and lower extremities (68.3%), followed by the palms (46.7%) and soles (20.0%). The disease course was predominantly progressive (76.7%). Regarding prior therapy, 60% received combined topical treatment and phototherapy, while 40% used topical therapy alone **(Table 2)**.

Genotype and allele distributions differed significantly between psoriasis cases and controls ($p < 0.001$). The AG and GG genotypes were significantly more frequent among cases, whereas the AA genotype predominated in controls. The G allele was significantly associated with psoriasis susceptibility ($p < 0.001$). Both dominant (AG + GG vs AA) and recessive (GG vs AG + AA) genetic models showed significant associations with psoriasis risk ($p < 0.001$) **(Table 3).**

No significant differences were observed in demographic or lifestyle characteristics across the three genotypes (AA, AG, GG), including age ($p = 0.161$), BMI ($p = 0.830$), sex, occupation, marital status, and smoking status (all $p > 0.05$) **(Table 4).**

PASI score differed significantly among genotypes, with higher scores in GG carriers ($p < 0.001$). No significant differences were observed regarding disease duration ($p = 0.871$), site of involvement (all $p > 0.05$), or disease course ($p = 0.504$) **(Table 5).**

Family history was a significant predictor of PASI score, with higher severity in patients with a positive history ($p = 0.015$). BMI showed a non-significant positive trend ($p = 0.056$); sex, age, disease duration, and genotype were not associated (all $p > 0.05$) (**Table 6**).

BMI showed a significant positive correlation with PASI score ($r = 0.335$, $p = 0.035$). No significant correlations were found between PASI score and age, treatment duration, or disease duration (all $p > 0.05$) (**Table 7, Fig 1**).

Table (1). Demographic Characteristics of Psoriasis Cases and Controls

Variables		Psoriasis cases (N=40)	Controls (N=40)	P-value
Age (years)	Mean ±SD	39.45 ± 11.87	35.25 ± 12.29	0.124^{t}
Gender	Male	20 (50.0%)	22 (55.0%)	0.654^{χ^2}
	Female	20 (50.0%)	18 (45.0%)	
BMI (kg/m²)	Mean ±SD	22.21 ± 1.84	22.22 ± 2.02	0.983^{t}
Occupation	Worker	18 (45.0%)	23 (57.5%)	0.263^{χ^2}
	Not worker	22 (55.0%)	17 (42.5%)	
Marital status	Married	34(85.0%)	37(92.5%)	0.288^{χ^2}

	Not married	6(15.0%)	3(7.5%)	
Comorbidities	None	40 (100.0%)	40 (100.0%)	------
Smoking		6 (15.0%)	8 (20.0%)	0.556$^{\chi^2}$
Family history		12 (30.0%)	0 (0.0%)	<0.001$^{\chi^2}$*

χ; Chi-square; t: independent sample t-test.

Table (2). Clinical characteristics of psoriatic cases

Variables		**Psoriasis cases (N=40)**
PASI score		3.53 ± 0.78
Disease duration (years)		2.34 ± 1.39
Duration of treatment (months)		1.72 ± 0.73
Site of involvement	**Palm involvement**	28 (46.7%)
	Upper extremities	55 (91.7%)
	Sole involvement	12 (20.0%)
	Lower extremities	41 (68.3%)
	Trunk involvement	7 (11.7%)
	Scalp involvement	5 (8.3%)
Course / Progression	**Stationary**	9 (15.0%)
	Regressive	5 (8.3%)
	Progressive	46 (76.7%)
Previous treatment	**Topical**	16 (40.0%)
	Topical &phototherapy	24 (60.0%)

Table (3). Card 14 (rs34367357) genotype and allele distribution in psoriasis cases and controls

Variable	**Total (N=80)**	**Psoriasis cases (N=40)**	**Controls (N=40)**	**P value**
Genotype				<0.001* $^{\chi^2}$
AA	36 (45.0%)	4 (10.0%)	32 (80.0%)	
AG	26 (32.5%)	20 (50.0%)	6 (15.0%)	
GG	18 (22.5%)	16 (40.0%)	2 (5.0%)	
Allele frequency				<0.001* $^{\chi^2}$
A allele	98 (61.25%)	28 (35.0%)	70 (87.5%)	
G allele	62 (38.75%)	52 (65.0%)	10 (12.5%)	
Dominant model (AG+GG vs AA)				<0.001* $^{\chi^2}$
AG+GG	44 (55.0%)	36 (90.0%)	8 (20.0%)	
AA	36 (45.0%)	4 (10.0%)	32 (80.0%)	
Recessive model (GG vs AG+AA)				<0.001* $^{\chi^2}$
GG	18 (22.5%)	16 (40.0%)	2 (5.0%)	
AG+AA	62 (77.5%)	24 (60.0%)	38 (95.0%)	

*: significant; χ; Chi-square.

Table (4). Association between CARD14 (rs34367357) genotypes and demographic characteristics among total study population (N=80)

Variable	**Category**	**AA (n=36) n (%)**	**AG (n=26) n (%)**	**GG (n=18) n (%)**	**P value**
Age (years)		34.81 ± 12.20	40.81 ± 11.11	37.44 ± 13.05	0.161#
Sex	**Male**	19 (52.8%)	14 (53.8%)	9 (50.0%)	0.968 $^{\chi^2}$
	Female	17 (47.2%)	12 (46.2%)	9 (50.0%)	

BMI (kg/m²)		22.14 ± 2.02	22.14 ± 2.04	22.46 ± 1.54	0.830#
Occupation	**Worker**	18 (50.0%)	15 (57.7%)	8 (44.4%)	0.674 χ^2
	Not worker	18 (50.0%)	11 (42.3%)	10 (55.6%)	
Marital status	**Married**	34 (94.4%)	23 (88.5%)	14 (77.8%)	0.188 χ^2
	Not married	2 (5.6%)	3 (11.5%)	4 (22.2%)	
Smoking		8 (22.2%)	5 (19.2%)	1 (5.6%)	0.303 χ^2

*: significant; χ; Chi-square; #:one-way ANOVA test.

Table (5). Association between CARD14 (rs34367357) genotypes and clinical involvement among psoriasis patients (N=40)

Variable	AA (n=4) n (%)	AG (n=20) n (%)	GG (n=16) n (%)	P value
PASI score	2.25 ± 0.50	3.35 ± 0.49	4.06 ± 0.68	<0.001*#
Disease Duration (years)	2.08 ± 2.04	2.29 ± 1.36	2.46 ± 1.34	0.871#
Palm	1 (25.0%)	10 (50.0%)	7 (43.8%)	0.651 χ^2
Upper extremities	4 (100.0%)	15 (75.0%)	11 (68.8%)	0.435 χ^2
Lower extremities	1 (25.0%)	10 (50.0%)	11 (68.8%)	0.237 χ^2
Trunk	0 (0.0%)	2 (10.0%)	0 (0.0%)	0.349 χ^2
Sole	0 (0.0%)	2 (10.0%)	2 (12.5%)	0.757 χ^2
Course / Progression				
Stationary	3 (75.0%)	7 (35.0%)	8 (50.0%)	0.504 χ^2
Regressive	1 (25.0%)	5 (25.0%)	4 (25.0%)	
Progressive	0 (0.0%)	8 (40.0%)	4 (25.0%)	

*: significant; χ; Chi-square; #:one-way ANOVA test.

Table (6). Multiple linear regression analysis of factors associated with PASI score

Variable	B	Standardized β	P value	95% CI
Family history	0.669	0.396	0.015*	0.137 – 1.200
BMI	0.129	0.301	0.056	-0.004 – 0.261
Sex	-0.283	-0.183	0.264	-0.789 – 0.223
Disease duration	-0.070	-0.125	0.416	-0.244 – 0.103
Genotype	0.097	0.083	0.599	-0.274 – 0.468
Age	0.003	0.049	0.749	-0.017 – 0.024

*: significant.

Table (7). Correlation between PASI Score and clinical parameters in psoriasis cases

Variable	r (Pearson)	P value
Age (years)	-0.023	0.887
BMI (kg/m²)	0.335	0.035*
Duration of treatment (months)	-0.068	0.677
Disease duration (years)	-0.039	0.811

*: significant.

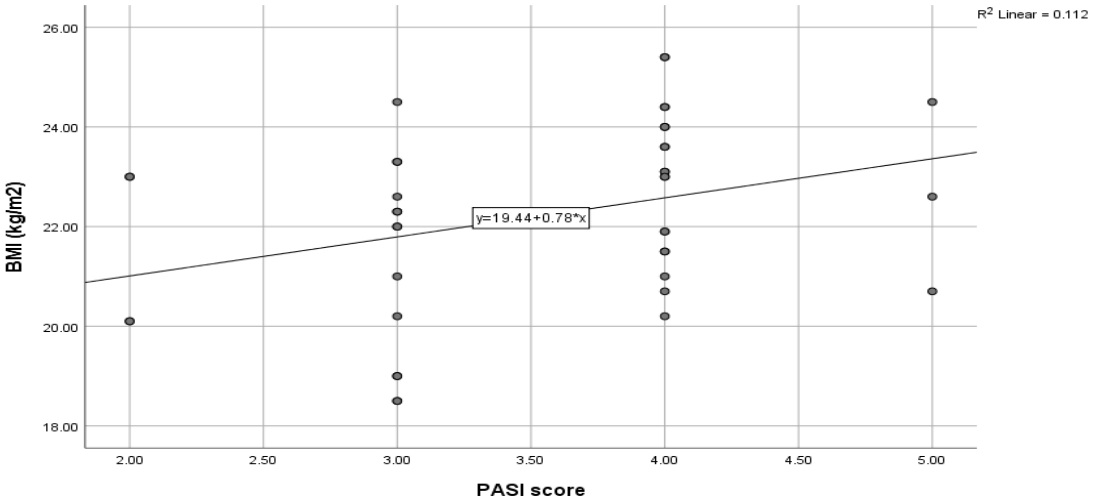

Fig 1. Ccorrelation between PASI score and BMI (kg/m²)

Discussion

Psoriasis is a chronic immune-mediated inflammatory disease characterized by keratinocyte hyperproliferation and impairment of the epidermal barrier. Hyperactivation of the NF-κB pathway drives the release of proinflammatory cytokines and chemokines. The CARD14 gene (rs34367357) encodes a keratinocyte scaffold protein that forms the CBM complex, and gain-of-function mutations in CARD14 lead to excessive immune activation, resulting in the recruitment of neutrophils and T cells to the skin (**Suleman et al., 2022**).

Our study demonstrated that patients with mild psoriasis and controls were comparable regarding demographic characteristics and lifestyle factors; however, a positive family history was observed exclusively among patients. The disease exhibited a predominantly progressive course, mainly affecting the upper (91.7%) and lower (68.3%) extremities, while also involving keratinocytes, nails, joints, and mucosa. Previous treatments consisted of topical therapies alone (40%) or in combination with phototherapy (60%). Consistent with our findings, **El-Komy et al. (2023)** evaluated 200 Egyptian psoriatic patients and reported upper limb involvement in 78% of cases, with a progressive course observed in 75%, closely aligning with the high upper extremity involvement and progressive pattern identified in our cohort.

The CARD14 rs34367357 polymorphism induces a gain-of-function mutation (V585I), leading to hyperactivation of the CBM signaling complex in keratinocytes and subsequent activation of the NF-κB and MAPK pathways. CARD14 is predominantly expressed in epidermal keratinocytes, underscoring its role in localized cutaneous inflammation. This activation results in excessive release of pro-inflammatory cytokines and chemokines (IL-8, CCL20), which recruit Th17 cells and neutrophils, thereby promoting keratinocyte hyperproliferation and progressive plaque formation, as observed in our study (**Niedźwiedź et al., 2025**).

Our results revealed a significant difference in genotype distribution between patients and controls, with the G allele and AG/GG genotypes predominantly associated with cases, whereas the A allele and AA genotype were more frequently observed among controls. Both dominant and recessive genetic models confirmed a strong association between the G allele and disease susceptibility. Similarly, **Suleman et al. (2022)** analyzed 123 subjects (63 patients and 60 controls) and reported a significant association of the rs34367357 (V585I) variant with psoriasis ($p < 0.05$), supporting the allelic distribution and genetic predominance observed in our cohort.

No significant differences in baseline characteristics or lifestyle factors were observed across the three genotypes in our study. CARD14 is primarily expressed in epidermal keratinocytes,

where variants such as rs34367357 promote assembly of the CARD14-BCL10-MALT1 (CBM) complex, leading to hyperactivation of NF-κB and mitogen-activated protein kinase (MAPK) signaling pathways and driving localized skin inflammation. As this effect is confined to the epidermis, systemic variables such as BMI, age, and lifestyle factors remain unaffected (**Suleman et al., 2022**). In agreement, **Abbas and Al-Kenan (2024)** assessed 120 individuals (60 patients and 60 controls) and reported a mean patient age of 30.11 ± 8.07 years, with no significant differences between groups ($p = 0.321$), consistent with our findings.

Disease severity, as measured by the PASI score, was highest among GG genotype carriers in our study, whereas disease duration, site of involvement, and progression did not differ significantly across genotypes. The GG genotype likely contributes to elevated PASI scores through gain-of-function effects in keratinocytes, enhancing CBM complex assembly and NF-κB hyperactivation, which in turn leads to overproduction of IL-36 and IL-19 and activation of the IL-23/IL-17 axis (**Hamid, 2025**). The predominance of mild psoriasis cases within our cohort may explain the limited variation in other clinical parameters across genotypes. Similarly, **Suleman et al. (2022)** reported that rs34367357 significantly influenced clinical disease severity in 123 subjects, confirming its impact on PASI scores in agreement with our findings ($p < 0.001$). Family history emerged as a significant predictor of disease severity in our cohort, with higher PASI scores observed among patients with affected relatives. BMI demonstrated a positive trend, whereas sex, disease duration, genotype, and age were not significantly associated with severity. The association between family history and disease severity likely reflects a cumulative genetic burden, with high-penetrance alleles such as CARD14 rs34367357 disrupting autoinhibition and activating NF-κB signaling (**Jiang et al., 2024**). In contrast, **Hu et al. (2026)**, in an analysis of 11,134 psoriasis patients using machine learning models, identified significant associations between BMI ($p = 0.007$), gender ($p = 0.001$), and disease duration ($p = 0.024$) with treatment outcomes. This differs from our findings, likely due to variations in cohort size, genetic background, and methodological approaches.

Our study also identified a positive correlation between BMI and disease severity, whereas age, treatment duration, and disease duration were not significantly associated. This relationship may be attributed to the endocrine activity of adipose tissue, which secretes pro-inflammatory cytokines such as TNF-α, IL-6, and leptin, thereby amplifying systemic inflammation and the Th17/IL-23 axis, ultimately promoting keratinocyte hyperproliferation (**Vata et al., 2023**). Supporting this observation, **Prakasita et al. (2025)** reported a significant positive correlation between BMI and PASI score ($r = 0.233$, $p = 0.002$) in a cohort of 181 patients with psoriasis vulgaris.

Our study has some limitations, including its small single-center cohort, and focus on predominantly mild psoriasis, which restricts causal inference and generalizability. Additionally, examining only the CARD14 (rs34367357) variant does not capture other genetic, epigenetic, or environmental factors influencing psoriasis pathogenesis.

Conclusion

Our study identifies the CARD14 (rs34367357) variant as a key genetic factor in Egyptian psoriasis, with the G allele and GG genotype linked to greater disease severity through NF-κB–driven skin inflammation. Family history strongly predicts severity, and higher BMI further exacerbates disease, highlighting the interplay of genetic and systemic factors.

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