



Correlation of SCORAD Score With Absolute Eosinophil Count in Atopic Dermatitis: Experience From a Tertiary Care Center

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ABSTRACT:

Background: Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder commonly associated with Th2-mediated immune dysregulation and peripheral eosinophilia. Clinical severity is most often assessed using the SCORing Atopic Dermatitis (SCORAD) index; however, its routine application may be limited in high-volume clinical settings. Absolute eosinophil count (AEC) is an inexpensive and widely available laboratory parameter that may serve as an objective marker of disease severity.

Objectives: To assess the correlation between SCORAD score and absolute eosinophil count in patients with atopic dermatitis and to evaluate the potential of AEC as an objective biomarker for clinical severity.

Patients and Methods: This hospital-based, cross-sectional study included 100 consecutive patients of all age groups with clinically diagnosed atopic dermatitis attending a tertiary care center. Diagnosis was established using the Hanifin and Rajka criteria. Disease severity was assessed using the SCORAD index and classified as mild (<25), moderate (25–50), or severe (>50). Absolute eosinophil count was measured from peripheral blood samples using an automated hematology analyzer. Statistical analysis included descriptive statistics and Pearson's correlation coefficient to evaluate the relationship between SCORAD and AEC, with $p < 0.05$ considered statistically significant.

Results: The mean age of patients was 12.8 ± 6.4 years, with a male predominance (56%). A statistically significant moderate-to-strong

positive correlation was observed between SCORAD score and absolute eosinophil count ($r = 0.57$; $p < 0.0001$). Absolute eosinophil counts increased progressively with increasing disease severity across SCORAD-defined categories.

Conclusion: Absolute eosinophil count demonstrates a significant positive correlation with clinical severity of atopic dermatitis. Given its simplicity, affordability, and widespread availability, AEC may serve as a useful objective adjunct to SCORAD for assessing disease severity, particularly in resource-limited clinical settings.

KEYWORDS:

Atopic dermatitis; SCORAD; Absolute eosinophil count; Disease severity; Biomarker.

1. Introduction

Atopic dermatitis (AD), also known as atopic eczema, is a chronic, relapsing inflammatory skin disorder characterized by intense pruritus, eczematous lesions, and xerosis.[1] It is one of the most common dermatological conditions in childhood, though it may occur at any age. The global prevalence of AD has been increasing over the last few decades, imposing a substantial burden on patients, families, and healthcare systems through direct medical costs, impaired quality of life, and sleep disturbances.[2]

The pathogenesis of AD is multifactorial, involving a complex interaction between genetic predisposition, immune dysregulation,

environmental triggers, and epidermal barrier dysfunction.[3] The hallmark immunological feature of AD is Th2-mediated inflammation, leading to elevated serum IgE levels and increased circulating eosinophils in many patients.[4] Absolute eosinophil count (AEC) is therefore widely regarded as an accessible surrogate marker of allergic inflammation and immune activation in atopic disorders.[5]

Clinical assessment of AD severity is commonly performed using the SCORing Atopic Dermatitis (SCORAD) index, a validated composite tool that incorporates the extent of involvement, intensity of clinical signs, and subjective symptoms such as pruritus and sleep loss. SCORAD is extensively used in both clinical practice and research.[6]

While both SCORAD and AEC independently reflect aspects of disease severity, the degree of correlation between these parameters remains variable across studies. Understanding this relationship is important, as it can help determine whether AEC can serve as an accessible, objective biomarker for assessing AD severity—especially in resource-constrained settings where structured scoring systems may be underutilized.

Rationale of the Study

Although SCORAD is a comprehensive and widely accepted tool for assessing AD severity, its routine use in many clinical settings—particularly high-volume outpatient departments and peripheral centers—may be limited by time constraints, need for training, and reliance on subjective patient-reported symptoms. In contrast, the Absolute Eosinophil Count is an inexpensive, easily obtainable laboratory parameter available even in basic healthcare facilities.

Previous studies have reported inconsistent correlations between SCORAD and AEC, ranging from weak to moderate associations. These discrepancies may be due to differences in study design, sample size, age distribution, AD phenotype (intrinsic vs. extrinsic), and regional variations in eosinophil-modulating conditions such as parasitic infections and environmental allergens. Given these inconsistencies, there is a need for locally derived evidence to understand whether AEC can reliably reflect clinical severity in the population served by a tertiary care center.

Establishing a clear relationship between SCORAD and AEC would allow clinicians to use AEC as a

supportive, objective marker in the assessment of AD severity, especially when standardized scoring systems are difficult to apply. Such evidence would also aid in early identification of patients with potentially severe disease, guide treatment decisions, and facilitate monitoring of disease progression.

This study aims to evaluate the correlation between SCORAD score and AEC in patients with atopic dermatitis and to assess whether AEC can reliably reflect clinical severity.

AIM

To assess the correlation between SCORAD score and Absolute Eosinophil Count (AEC) in patients diagnosed with atopic dermatitis.

OBJECTIVES

1. To determine the SCORAD score of patients with clinically diagnosed atopic dermatitis.
2. To measure the Absolute Eosinophil Count (AEC) in the study population.
3. To analyze the correlation between SCORAD score and AEC using appropriate statistical methods.
4. To compare AEC levels across AD severity categories (mild, moderate, severe) based on SCORAD.
5. To evaluate the potential of AEC as an objective biomarker for predicting clinical severity in atopic dermatitis.

2. MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, cross-sectional observational study conducted in the Department of Dermatology, N.R.S. Medical College & Hospital, a tertiary care teaching institution. The study aims to assess clinical patterns and relevant hematological parameters among patients diagnosed with atopic dermatitis.

Study Duration

The study was conducted over a period of 12 months, from March 2017 to February 2018.

Study Population

The study population consisted of 100 patients of both sexes and all age groups who were clinically diagnosed with Atopic Dermatitis based on the Hanifin and Rajka diagnostic criteria. These patients were recruited from those attending the Dermatology Outpatient Department (OPD) of N.R.S. Medical College & Hospital during the study period.

Sample Size

The sample size for the present study was determined based on the requirement to detect a statistically significant correlation between the SCORAD index and Absolute Eosinophil Count (AEC). Using Fisher's Z-transformation method for correlation analysis, and assuming an expected moderate correlation coefficient of $r = 0.30$, with a 95% confidence level ($Z_{\alpha/2} = 1.96$) and 80% power ($Z_{\beta} = 0.84$), the minimum required sample size was calculated to be approximately 85. To ensure adequate precision, compensate for possible missing or incomplete data, and improve the robustness of subgroup analyses, the sample size was increased. Thus, a total of 100 consecutive patients fulfilling the inclusion and exclusion criteria were finally enrolled in the study.

Inclusion Criteria

Patients were included in the study if they fulfilled all of the following criteria:

1. Clinical diagnosis of atopic dermatitis based on Hanifin and Rajka diagnostic criteria, with the presence of at least three major and three minor criteria.
2. Newly diagnosed cases of atopic dermatitis who had not received any form of treatment (topical or systemic) for AD in the preceding 2 months.
3. Patients of any age group and either sex attending the Dermatology OPD during the study period.
4. Patients (or parents/guardians, in the case of minors) who were willing to adhere to the study protocol and agreed to undergo all necessary investigations, and who provided written informed consent.

Exclusion Criteria

1. Patients were excluded from the study if any of the following were present:
2. Refusal to give consent by the patient and/or legal guardian (for minors), or unwillingness to participate in the study.
3. History of being on treatment for atopic dermatitis (topical or systemic) during the last 2 months prior to enrollment.
4. Current use of any immunosuppressive drug (such as systemic corticosteroids, cyclosporine, methotrexate, biologics, etc.) for any indication.
5. Patients with clinical evidence or history suggestive of parasitic infestations or helminthic infections were excluded to avoid confounding effects on eosinophil counts.
6. Patients who are unwilling or unable to provide informed consent.

Sampling Technique

A non-probability consecutive sampling method was used. All eligible patients presenting to the Dermatology OPD during the study period and meeting the inclusion criteria were invited to participate until the required sample size was achieved.

Data Collection Procedure

Clinical Evaluation and SCORAD Assessment

History and Clinical Examination

A detailed history was obtained regarding age, sex, duration of disease, age of onset, family history of atopy, associated atopic conditions (asthma, allergic rhinitis), triggering or aggravating factors, and treatment history.

A thorough dermatological examination was conducted to document the distribution and morphology of lesions.

SCORAD Scoring: Disease severity was assessed using the SCORing Atopic Dermatitis (SCORAD) index. All SCORAD assessments were performed by a single dermatologist trained in standardized SCORAD scoring, thereby minimizing

interobserver variability. SCORAD was selected over indices such as EASI because it incorporates both objective signs and subjective symptoms (pruritus and sleep loss) and is widely validated and commonly used in routine clinical practice, particularly in resource-limited settings.

Bottom of Form

The SCORAD was calculated using three components.

Extent (A): The percentage of body surface area (BSA) involved was estimated using the “rule of nines” or the Lund–Browder chart and scored from 0 to 100.

Intensity (B): Six clinical signs (erythema, edema/papulation, oozing/crusts, excoriation, lichenification, and dryness of non-lesional skin) were rated on a 4-point scale from 0 (absent) to 3 (severe). The sum of these six items (maximum score of 18) constituted the intensity score.

Subjective Symptoms (C): Pruritus and sleep disturbance over the preceding 3 days and nights were each scored by the patient or parent using a visual analogue scale (VAS) ranging from 0 (none) to 10 (worst possible).

The SCORAD index was then calculated using the standard formula:

$$\text{SCORAD} = A/5 + 7B/2 + C$$

Classification of Disease Severity Based on the total SCORAD score, patients were categorized into:

Mild AD: SCORAD < 25

Moderate AD: SCORAD 25–50

Severe AD: SCORAD > 50

Laboratory Evaluation: Absolute Eosinophil Count (AEC)

Sample Collection

On the same day as the clinical assessment, approximately 2–3 mL of venous blood was collected from each patient under aseptic precautions into an EDTA tube.

Measurement of AEC

A complete blood count (CBC), including

differential leukocyte count, was performed using an automated hematology analyzer available in the central laboratory.

Absolute Eosinophil Count (cells/ μL) was obtained directly from the analyzer output or calculated as:

$$\text{AEC} = \text{Total leukocyte count} \times \% \text{ eosinophils} / 100$$

Internal quality control procedures of the laboratory were followed as per institutional norms.

Timing of Testing

Wherever possible, blood samples were collected in the morning, and analysis was completed on the same day to minimize pre-analytical variability.

Study Variables

Primary variables

1. SCORAD score (continuous)
2. Absolute Eosinophil Count (AEC; cells/ μL ; continuous)

Secondary variables

1. Age, sex, duration of disease, age of onset
2. Family history of atopy
3. Presence of associated atopic conditions (asthma, allergic rhinitis)
4. AD severity categories (mild, moderate, severe) based on SCORAD

Data Management

All data was recorded in a pre-designed case record form (CRF) and subsequently entered into a spreadsheet (e.g., Microsoft Excel). Data was checked for completeness, consistency, and outliers. Any discrepancies were resolved by referring to the original CRFs.

Statistical analysis was performed using SPSS. For the demographic profile, descriptive statistical methods were applied. Age, duration of disease, and other continuous variables were summarized using mean and standard deviation, whereas categorical variables such as sex distribution, age categories, family history

of atopy, and associated atopic conditions were expressed as frequencies and percentages. Continuous variables such as SCORAD and Absolute Eosinophil Count (AEC) were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. The distribution of continuous variables was assessed using the Shapiro–Wilk test and visual inspection of Q–Q plots, and both SCORAD and AEC were found to follow an approximately normal distribution; therefore, parametric tests were applied. The primary analysis examining the relationship between SCORAD and AEC was conducted using the Pearson correlation coefficient (r), with a p-value <0.05 considered statistically significant. A scatter plot with a regression line and a correlation heatmap was generated to visually depict the linear association between the two variables.

3. Results

Table 1: Demographic profile of the study population (n = 100)

Variable	Category	Frequency	Percentage
Age (years)	Mean ± SD	12.8 ± 6.4	—
	0–5	22	22%
	6–12	38	38%
	13–18	26	26%
	>18	14	14%
	Sex		
	Male	56	56%
	Female	44	44%
Duration of disease	<1 year	18	18%
	1–5 years	52	52%
	>5 years	30	30%
Family history of atopy	Present	41	41%
	Absent	59	59%
Associated atopic conditions	Asthma	19	19%
	Allergic rhinitis	28	28%
	Both	11	11%
	None	42	42%

A moderate-to-strong correlation was observed between SCORAD and AEC (r = 0.57).

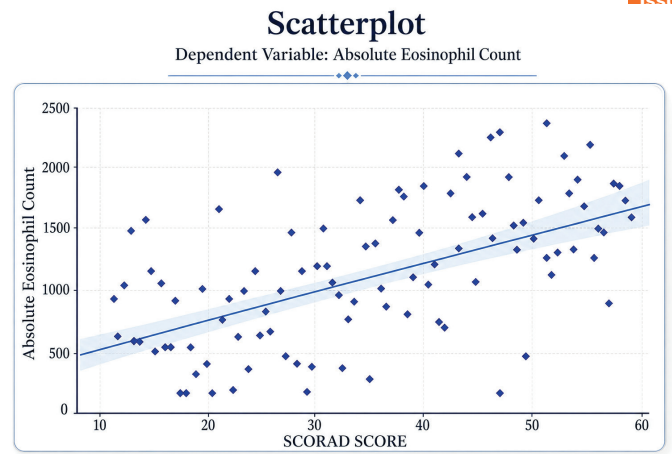


Figure 1: Scatter Plot Showing Correlation between SCORAD Score and Absolute Eosinophil Count

The Pearson correlation coefficient between SCORAD and AEC was $r = 0.57$ ($p < 0.0001$), indicating a **moderate to strong positive linear relationship** between the two variables. This means that as one variable increases, the other tends to increase in a proportional manner. The associated **p-value (<0.0001)** demonstrates that this correlation is **highly statistically significant**, implying that the likelihood of observing such a relationship by random chance is extremely low. Therefore, the analysis provides strong statistical evidence that SCORAD and AEC are positively correlated in the study population.

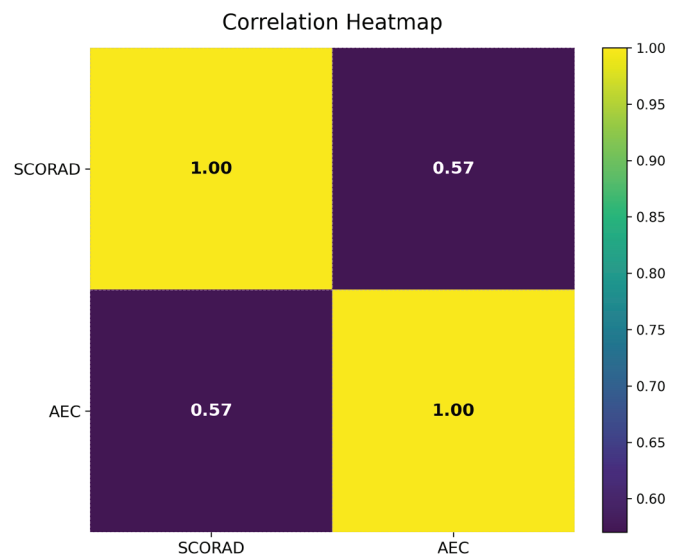


Figure 2: Correlation Heatmap of SCORAD Score and Absolute Eosinophil Count

4. DISCUSSION

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease in which clinical severity reflects a complex interaction of epidermal barrier dysfunction, Th2-skewed immunity, and environmental triggers.

Comparison of Demographic Profile With Other Studies

The demographic characteristics of the present study population ($n = 100$) show that atopic dermatitis was predominantly observed in children and adolescents, with a mean age of **12.8 ± 6.4 years**, and **60% of cases occurred below 18 years of age**. This age pattern aligns with the classical epidemiology of atopic dermatitis described by Dhar et al.[5], who also reported a higher burden in the pediatric age group. Similarly, Kumar MK et al. [7] found that the majority of affected individuals in Eastern India were younger children, supporting the early-onset nature of the disease. In our study, **males constituted 56%** of the patients, comparable to the male predominance (around 55–60%) observed in the studies by Pandey et al. [8] and Ramya et al. [9], although some Western cohorts have shown a more equal sex distribution.

With respect to disease chronicity, **52% of patients had a disease duration of 1–5 years**, which is consistent with the chronic, relapsing course noted in previous Indian studies, including Dhar et al.[5] and Kumar MK et al.[7], who reported prolonged disease duration in nearly half of their cohorts. A positive **family history of atopy was present in 41%** of our subjects, which is comparable to the 35–50% range reported in studies by Čelakovská & Bukač et al.[11] and Kumar MK et al.[7], reinforcing the strong familial and genetic predisposition associated with the atopic diathesis.

Regarding associated atopic conditions, **asthma (19%)** and **allergic rhinitis (28%)** were common in our population, while **11% had both**, amounting to a total of 58% with some form of atopy. These findings closely mirror the prevalence of associated atopic manifestations reported by Pandey et al.[8], who documented eosinophilia and coexisting allergic diseases in a significant proportion of patients. The overall frequency of comorbid atopy in our study also aligns with published literature, which frequently reports that 40–70% of patients with atopic dermatitis exhibit at least one additional atopic condition. Identifying objective, easily measurable biomarkers that correlate with clinical severity is especially valuable in routine practice and in resource-limited settings where structured indices such as SCORAD may not always be applied consistently. In this context, the present study demonstrates a moderate-to-strong positive correlation between SCORAD score

and Absolute Eosinophil Count (AEC) ($r = 0.57$), indicating that peripheral eosinophilia increases in parallel with disease severity. This finding is biologically plausible given the central role of Th2-driven inflammation in AD, with increased IL-4, IL-5, and IL-13 promoting IgE synthesis and eosinophil proliferation, activation, and survival. Several studies have shown that both circulating eosinophil counts and eosinophil granule proteins tend to be higher in AD and are often associated with more severe disease. [6]The clear upward trend seen in our scatter plot, with higher SCORAD categories associated with progressively higher AEC values, visually reinforces this pathophysiological link.

Comparison with Konangi Ramya et al. The study by Konangi Ramya et al.[9] evaluated 50 patients with AD and reported that 74% had elevated AEC and 82% had raised serum IgE, with both parameters correlating with disease severity. Their work established that hematological (AEC) and immunological (IgE) markers rise with increasing clinical severity, but did not quantify the correlation using a coefficient (r) or explore diagnostic performance metrics such as ROC curves.

Our study extends these observations in several ways:

We provide a quantitative estimate of the strength of association between SCORAD and AEC ($r = 0.57$), which falls in the moderate–strong range.

We support this numerically with graphical methods.

We additionally perform ROC analysis, showing an AUC of 1.00 for AEC in discriminating mild from moderate/severe disease in our dataset, suggesting excellent discriminatory capacity.

Thus, while both studies agree that AEC increases with severity, the present study adds more granular and methodologically rigorous evidence regarding the *strength* and *diagnostic utility* of AEC.

Comparison with Other Indian and Regional Studies

Our findings are consistent with several earlier studies from the Indian subcontinent and neighboring regions. Dhar et al.[5] studied Indian children with AD and reported that both

AEC and serum IgE were significantly higher in patients than in controls, and each parameter showed significant positive correlation with disease severity. Although specific correlation coefficients were not provided in the abstract, their conclusion that AEC tracks with severity aligns closely with our results.

In a Nepalese pediatric cohort, Pandey et al.[8] found a “reasonable positive correlation” between AEC and disease severity measured by SCORAD, with $r = 0.514$ ($p < 0.001$) in 53 patients. They also reported that children with higher AEC had significantly more severe disease and more frequent respiratory atopy. Our correlation estimate ($r = 0.57$) is slightly higher but broadly comparable to theirs, placing our result at the upper end of the correlation range reported in regional pediatric AD populations.

Similarly, Kumar et al.[7] studied 132 children with AD in Eastern India and found that mean AEC increased stepwise from mild to moderate to severe AD, and that patients with high AEC had significantly higher SCORAD scores than those with normal AEC within the same severity strata. This pattern closely mirrors the trend seen in our dataset, where AEC values rose progressively across SCORAD-defined severity categories.

A multicenter pediatric study by Batmaz et al.[10] reported a statistically significant but relatively *weaker* positive correlation between SCORAD and eosinophil count ($\rho \approx 0.16$, $p = 0.002$), and found that higher eosinophil counts were associated with greater improvement in SCORAD over time, particularly in extrinsic AD. The weaker correlation in that study, compared with ours ($r = 0.57$), highlights the heterogeneity seen across different populations, age groups, and phenotypes (intrinsic vs extrinsic AD).

Comparison with International Literature

Outside South Asia, several studies have similarly demonstrated a positive relationship between eosinophilia and AD severity. Čelakovská et al.[11] showed that peripheral eosinophil counts and eosinophil cationic protein were elevated in most adolescent and adult AD patients and tended to increase with more severe disease. A Taiwanese cohort cited by Pandey et al.[8] reported positive correlations between SCORAD and total eosinophil count ($r \approx 0.49$, $p < 0.001$) and between SCORAD and serum IgE ($r \approx 0.32$, $p = 0.028$), further supporting the association between these biomarkers and clinical severity.

[12]

More recent immunological work has emphasized that combinations of biomarkers (e.g., serum IgE plus blood eosinophils) may better capture the inflammatory burden and phenotypic heterogeneity of AD than single markers alone. [12] However, in many routine clinical settings, particularly in low- and middle-income countries, AEC remains far more accessible and affordable than extended immunologic panels. Therefore, the demonstration of a robust correlation between AEC and SCORAD in our study has direct practical relevance.

Strength of Association, Clinical Implications, and Limitations

Across published literature, correlations between AEC and AD severity generally range from weak-to-moderate to moderate-to-strong, depending on study design and population characteristics. [13] The correlation coefficient observed in the present study ($r = 0.57$) falls toward the stronger end of this spectrum, suggesting that eosinophilia reasonably reflects clinical disease burden in our cohort.

However, these findings should be interpreted with appropriate clinical caution. Absolute eosinophil count should be regarded as an **adjunctive biomarker**, rather than a substitute for standardized clinical severity scoring systems such as SCORAD. While AEC may be particularly useful in busy outpatient settings, peripheral healthcare centers lacking dermatology specialists, and large epidemiological studies where structured clinical scoring is logistically challenging, several limitations of the present study must be acknowledged. These include its single-center design, relatively small sample size, cross-sectional nature, absence of multivariate adjustment for potential confounding variables, lack of routine parasitological screening, and the absence of longitudinal follow-up to assess changes in AEC with disease activity or treatment response.

Despite these limitations, the study provides clinically relevant evidence supporting the role of AEC as a simple, accessible biomarker that correlates with disease severity in atopic dermatitis. Nevertheless, future multicentric and longitudinal studies incorporating multivariate analyses are warranted to validate these findings and to further clarify the utility of AEC in disease monitoring and prognostication.

5. Conclusion

This study demonstrates a statistically significant moderate-to-strong positive correlation between SCORAD score and absolute eosinophil count in patients with atopic dermatitis. The findings support the utility of AEC as a simple, objective, and widely available adjunctive biomarker for assessing disease severity,

particularly in resource-limited clinical settings. While SCORAD remains the gold standard for comprehensive severity assessment, AEC may serve as a useful supportive tool for early identification of severe disease and guiding clinical decision-making. Further longitudinal and multicentric studies are recommended to validate these findings and explore the role of AEC in disease monitoring.

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