Original Article

Diagnostic Value of NLR, PLR, hsCRP, and MPV for Assessing Disease Severity in Adult Atopic Dermatitis

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Abstract

Aim: Atopic dermatitis (AD) is a chronic inflammatory disease with systemic involvement. Severity is usually assessed using the scoring AD (SCORAD) index, but this method is partly subjective. Blood-derived markers such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), high-sensitivity C-reactive protein (hsCRP), and mean platelet volume (MPV) may provide objective indicators. This study evaluated their diagnostic value in assessing disease severity in adults with AD.

Materials and Methods: A cross-sectional study was conducted among 112 adults with AD at Ho Chi Minh City Hospital of Dermato-Venereology Hospital from March to September 2024. Demographics, clinical features, and SCORAD scores were recorded. Laboratory analyses included NLR, PLR, hsCRP, and MPV. Receiver operating characteristic (ROC) analysis determined the ability of these markers to identify severe AD (SCORAD ≥ 51).

Results: A total of 112 patients were included (62.5% female; mean age 48 years); 20% had severe AD (median SCORAD 30.7). Higher SCORAD was associated with increased neutrophils, NLR, PLR, and hsCRP, and with decreased lymphocytes (all $P < 0.05$). NLR best predicted severe AD, with an area under the receiver operating characteristic curve (AUROC) of 0.805 (cut-off 2.59; sensitivity 77.3%; specificity 80.0%), followed by PLR (AUROC: 0.754) and hsCRP (AUROC: 0.734), whereas MPV showed no predictive value (AUROC: 0.530; $P = 0.663$).

Conclusion: NLR, PLR, and hsCRP are simple, low-cost, and reliable biomarkers that correlate with AD severity, with NLR showing the highest diagnostic accuracy. Incorporating these indices may improve objective assessment and adjunctive tool in clinical practice.

Keywords: Atopic dermatitis, biomarkers, C-reactive protein, lymphocytes, neutrophils, platelets

Introduction

Atopic dermatitis (AD), the most common chronic inflammatory skin disease, imposes a substantial psychosocial burden by affecting appearance and reducing quality of life. Its complex pathogenesis involves immune dysregulation, impaired skin barrier function, and alterations of the commensal microbiota.[1] The disease is not confined to localized skin manifestations but also presents as a systemic inflammatory condition, often associated with other allergic diseases such as asthma and allergic rhinitis.[1,2] The global prevalence of AD is estimated to be as high as 30% in children and 2–10% in adults, and these rates continue to rise annually.[2-4] In addition to cutaneous symptoms, clinical and epidemiological factors including age, sex, age of onset, a history of allergic rhinitis, and personal and family histories of asthma play important roles in disease progression and severity stratification.[5] The integration of epidemiological and clinical characteristics forms the foundation for a more comprehensive and accurate model for assessing disease severity. Evaluating the severity of AD is a key factor in management and treatment selection. The scoring AD (SCORAD) index is a widely used and reliable clinical tool for disease severity assessment.[5] This score comprises three main components: the extent of disease; intensity (redness, swelling, oozing/crusts, scratch marks, lichenification, dryness); and subjective symptoms,

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including itch and sleeplessness, over the last three days. The total SCORAD ranges from 0 to 103, with disease severity classified as mild (< 25), moderate (25–50), or severe (≥ 51).[5-7] However, the method remains subjective as it depends on both physician and patient assessments. Consequently, blood-based biomarkers reflecting systemic inflammation are receiving increasing attention. Parameters such as white blood cell count, neutrophils, lymphocytes, eosinophils, platelet count, mean platelet volume (MPV), and high-sensitivity C-reactive protein (hsCRP) may serve as indicators of inflammatory activity.[2] Furthermore, systemic inflammatory ratios such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), which are readily obtained from a complete blood count, correlate strongly with systemic inflammation in chronic inflammatory diseases. Their low cost and broad applicability make them particularly advantageous.[2,5,8,9] No study in Vietnam has evaluated the NLR, PLR, or their associations with clinical features and disease severity in AD. Establishing threshold values for these indices is essential to enhance objectivity and clinical applicability. Therefore, this study aimed to assess MPV, NLR, PLR, eosinophil-to-lymphocyte ratio (ELR), dNLR, hsCRP, SCORAD scores, and the related demographic and clinical factors in adults with AD, with the aim of developing a more objective clinical tool to assess severity in practice.

Materials and Methods

Participants

Patients aged ≥ 18 years diagnosed with AD who attended Ho Chi Minh City Hospital of Dermato-Venereology Hospital for consultation and treatment between March 2024 and September 2024 were included.

Inclusion criteria were: a confirmed diagnosis of AD according to the Hanifin and Rajka criteria, modified by the American Academy of Dermatology in 2014;[10] age ≥ 18 years; and willingness to participate in the study. Exclusion criteria were: treatment with systemic corticosteroids or cytotoxic drugs within the past month; history of cardiovascular disease, chronic liver disease, end-stage chronic kidney disease, acute or chronic infections, malignancy, or systemic immune-related or immunodeficiency disorders; treatment with medications affecting platelet count or function within the past two weeks; and pregnancy or breastfeeding.

Sample Size and Sampling Method

The sample size was calculated for estimating a single proportion. We used $P = 0.325$ based on the proportion of patients in SCORAD group III (SCORAD ≥ 51) reported by Jiang and Ma,[5] and an acceptable margin of error (d) of 0.065. Using the standard formula for a single proportion ($n = Z^2 \cdot p \cdot (1–p)/d^2$, with Z corresponding to 95% confidence), the required sample size was 97. Allowing for 10% sample loss or unusable samples, the minimum target sample size was increased to 107. A total of 112 participants were recruited for the study. A consecutive convenience sampling method was used to enroll eligible patients until the required sample size was reached.

Data Collection Procedure

Data were recorded on a standardized case-report form that included the following: demographic characteristics (age, sex); personal history (allergic rhinitis, bronchial asthma); family history (bronchial asthma); dermatological examination findings. Clinical examination included the classification of disease stage (acute, subacute, or chronic), the determination of lesion sites and body surface area involvement (BSA%), and the detailed description of lesion morphology (redness, swelling, oozing/crusts, scratch marks, lichenification, and dryness). Photographs of representative lesions were also taken. Disease severity was assessed using the SCORAD index. Venous blood samples were collected into two tubes, one containing EDTA and the other heparin, and transferred to the Medic Hoa Hao Laboratory. Hematological parameters were analyzed using the SIEMENS ADVIA® 2120i automated hematology analyzer, which uses flow cytometry combined with laser fluorescence to identify and classify blood cell lineages, providing counts of red blood cells, white blood cells, and platelets, as well as MPV. Erythrocyte sedimentation rate was measured using the Ves-Matic Easy system based on automated optical detection. hsCRP levels were determined by an immunoturbidimetric assay using the Full Range hsCRP kit (Cat. No. CP3847 or CP3849). The procedure involved mixing serum samples with reagents containing anti-C-reactive protein (CRP) antibodies, measuring optical turbidity, and automatically calculating hsCRP concentration. All laboratory analyses were performed in accordance with standard operating procedures, with both internal and external quality control measures applied regularly to ensure accuracy and reproducibility of results.

Study Variables

Age was calculated as the survey year minus the year of birth (in years). Sex was classified into two categories: male and female. Personal history of allergic rhinitis was recorded as a categorical variable with two values: yes or no. Personal and family history of bronchial asthma were also recorded as categorical variables (yes/no).

Disease severity was assessed using the SCORAD index, a quantitative variable developed by the European Task Force on AD in 1993 and calculated by the formula SCORAD = A/5 + 7B/2 + C, where A represents the percentage of body surface area affected (BSA%), B is the total score of six clinical signs (0–18), and C is the sum of two subjective symptoms, pruritus and sleep loss (0–20). SCORAD severity categories were defined as follows: mild (< 25), moderate (25–50), and severe (≥ 51). This categorical variable thus comprised three levels. Laboratory variables included hematological and inflammatory markers, all treated as quantitative variables: peripheral blood neutrophil count (10^3/mm^3), lymphocyte count (10^3/mm^3), platelet count (K/mL), and MPV (fL). Inflammatory indices were calculated as follows: MPV, NLR, PLR, ELR, and dNLR. hsCRP (mg/L) was also measured.

Table 1. Clinical, demographic characteristics and laboratory results of the participants

	Value	n (%)/median (IQR)/mean ± SD
Age (years)		48 (32–61)
Gender	Male	42 (37.5)
	Female	70 (62.5)
Age of onset (years)		35 (22–49.5)
Age at onset categories	2 years	0 (0)
	2–12 years	12 (10.7)
	12–60 years	91 (81.3)
	> 60 years	9 (8.0)
History of allergic rhinitis	Yes	18 (16.1)
	No	94 (83.9)
History of bronchial asthma	Yes	10 (8.9)
	No	102 (91.1)
Family history of bronchial asthma	Yes	12 (10.7)
	No	100 (89.3)
WBC count, 10^9/L		8.6 (6.9–10.6)
Eosinophils, 10^9/L		0.28 (0.12–0.45)
Neutrophils, 10^9/L		5.0 (4.0–6.2)
Lymphocytes, 10^9/L		2.4 (1.8–2.9)
Platelet count, 10^9/L		295.5 ± 71.6
MPV, fL		9.3 (8.9–9.9)
NLR		2.1 (1.5–3.0)
PLR		114.0 (97.8–153.8)
ELR		0.059 (0.026–0.109)
dNLR		1.49 (1.07–1.92)
hsCRP, mg/L		1.7 (0.8–4.0)
SCORAD score		30.7 (15.1–40.2)
Categorize by SCORAD	Group I SCORAD (0-24)	39 (35)
	Group II SCORAD (25-50)	51 (45)
	Group III SCORAD (51-103)	22 (20)

WBC: White blood cell, NLR: Neutrophils to lymphocytes ratio, PLR: Platelet to lymphocyte ratio, MPV: Mean platelet volume, hsCRP: High-sensitivity C-reactive protein, SCORAD: Scoring atopic dermatitis index, ELR: Eosinophil-to-lymphocyte ratio, SD: Standard deviation, IQR: Interquartile range

Statistical Analysis

Data were managed using Microsoft Excel 365 (Windows OS) and analyzed with SPSS version 20 (IBM Corp., Armonk, NY, USA). The distribution of quantitative variables was assessed using the Shapiro–Wilk test. Variables with normal distribution were compared using one-way ANOVA with Tukey's post-hoc test, whereas non-normally distributed variables were analyzed using the Kruskal–Wallis test followed by Mann–Whitney U tests for pairwise comparisons. Receiver operating characteristic (ROC) curve analysis was used to evaluate the ability of MPV, NLR, PLR, ELR, dNLR, and hsCRP to discriminate patients in group III (SCORAD 51–103). The ROC curve was considered acceptable at ≥ 0.7 and good at ≥ 0.8. Youden's index was applied to calculate sensitivity and specificity and to determine the optimal cut-off values for predicting severe AD. A *P*-value < 0.05 was considered statistically significant.

Ethical Considerations

This study was approved approval from the Ethics Committee in Biomedical Research of Ho Chi Minh City Dermatology Hospital (approval number: 418/CN-BVDL, date: 29.02.2024). Written informed consent was obtained from all adult participants prior to enrollment, consistent with the Declaration of Helsinki (revised 2013).

Results

The study included 112 patients with a mean age of 48 years (range: 32–61). Females predominated, comprising 70 cases (62.5%) compared with 42 males (37.5%) (Table 1). The mean age at symptom onset was 35 years (22–49.5 years), and the majority (81.3%) of patients developed symptoms between 12 and 60 years. No cases of onset were recorded before 2 years of age, and only 8% had onset after 60 years of age. A history of allergy was reported in 18 patients (16.1%). A Personal history of bronchial asthma accounted for only 8.9% (10 cases), whereas a family history of asthma accounted for 10.7% (12 cases). The median white blood cell count was 8.6 × 10^9/L (6.9–10.6), which is within the normal range. Neutrophils were 5.0 × 10^9/L (4.0–6.2), eosinophils were 0.28 × 10^9/L (0.12–0.45), and lymphocytes were 2.4 × 10^9/L (1.8–2.9); all values were within normal limits. The platelet count was 295.5 ± 71.6 × 10^2/L, which is within the normal range. MPV was 9.3 fL (8.9–9.9); the inflammatory ratios included NLR 2.1 (1.5–3.0), PLR 114.0 (97.8–153.8), ELR 0.059 (0.026–

0.109), and dNLR 1.49 (1.07–1.92). The hsCRP level was 1.7 mg/L (0.8–4.0). The median SCORAD score was 30.7 (15.1–40.2). Based on the SCORAD classification, group I (0–24 points) accounted for 35%, group II (25–50 points) for 45%, and group III (51–103 points) for 20%. Most patients belonged to the moderate group, suggesting that the disease generally presented with mild to moderate clinical manifestations.

Comparison of mean neutrophil counts across the three SCORAD-based groups revealed a clear upward trend (Table 2). group III had the highest mean value, 5.82 (4.96–7.40) × 10^9/L, and the overall difference among the three groups was statistically significant ($P = 0.003$). Pairwise analysis revealed that this difference was most pronounced between group I and group III ($P < 0.001$). Lymphocyte counts, in contrast, showed a progressive decline with increasing disease severity, from 2.45 (2.01–2.93) × 10^9/L in group I to 1.70 (1.32–2.34) × 10^9/L in group III. This trend was highly significant ($P < 0.001$). Pairwise comparisons indicated that the largest difference was again observed between group I and group III ($P < 0.001$), and a significant difference was observed between group I and group II ($P < 0.001$). However, no statistically significant difference was observed between group II and group III ($P = 0.851$). Systemic inflammatory indices, including NLR, PLR, and hsCRP, also rose steadily from mild to severe disease, with the highest levels recorded in group III. All showed statistically significant differences among the three groups ($P \leq 0.001$). For NLR in particular, the differences were striking ($P < 0.001$). Pairwise analysis demonstrated that the greatest difference was observed between groups I and III ($P = 0.001$), while a significant difference was also observed between groups I and II ($P = 0.001$). No significant difference was observed between groups II and III ($P = 0.788$).

ELR values showed slight variation among the three groups, but the overall comparison was not statistically significant ($P = 0.057$). Pairwise analyses indicated no meaningful differences among groups I, II, and III ($P = 0.840$ for I vs. II; $P = 0.053$ for I vs. III; $P = 0.118$ for II vs. III).

dNLR values demonstrated a similar pattern, with no significant overall difference across the three groups ($P = 0.063$). Pairwise comparisons showed no significant differences between groups I and II ($P = 0.576$) or between groups II and III ($P = 0.229$); however, groups I and III differed significantly ($P = 0.049$). Similarly, hsCRP levels increased with disease severity, rising from 0.8 (0.4–1.1) mg/L in group I to 3.0 (2.2–4.7) mg/L in group III. The overall difference across the three groups was highly significant ($P < 0.001$). Pairwise comparisons confirmed that the most pronounced difference was between groups I and III ($P < 0.001$), and a significant difference was also observed between groups II and III ($P < 0.001$).

The diagnostic performance of MPV, NLR, PLR, ELR, dNLR, and hsCRP for identifying group III patients (SCORAD ≥ 51) is summarized in Table 3 and illustrated in Figure 1. MPV showed poor diagnostic value, with an AUROC of 0.530 [95% confidence interval (CI): 0.391–0.668; $P = 0.663$]. Moreover, NLR demonstrated the strongest diagnostic performance, with an AUROC of 0.805 (95% CI: 0.707–0.903; $P < 0.001$). At the cut-off value of 2.59, NLR achieved a sensitivity of 77.3% and a specificity of 80.0%. Additionally, PLR demonstrated good predictive ability, with an AUROC of 0.754 (95% CI: 0.621–0.856; $P < 0.001$), a sensitivity of 59.1% and a specificity of 90.0%. ELR demonstrated limited diagnostic value, with an AUROC of 0.589 (95% CI, 0.447–0.731; $P = 0.196$). At the cut-off level of 0.116, ELR provided a sensitivity of

Table 2. Laboratory results of patients according to SCORAD

	Group I (n = 39) SCORAD (0–24)	Group II (n = 51) SCORAD (25–50)	Group III (n = 22) SCORAD (51–103)	P^b	GI-II P^a	GI-III P^a	GII-III P^a
WBC count, 10^9/L	7.89 (6.08–9.33)	8.76 (6.80–19.62)	8.83 (7.51–11.26)	0.089	0.560	0.036	0.095
Neutrophils, 10^9/L	4.26 (3.61–5.24)	5.13 (4.01–6.17)	5.82 (4.96–7.40)	0.003	0.076	0.001	0.044
Lymphocytes, 10^9/L	2.45 (2.01–2.93)	2.51 (1.89–3.20)	1.70 (1.32–2.34)	< 0.001	< 0.001	< 0.001	0.851
Platelet count, 10^9/L	288.46 ± 67.12	291.22 ± 66.20	318.05 ± 88.41	0.209[c]	0.107[d]	0.111[d]	0.804[d]
MPV, fL	9.3 (8.8–9.9)	9.3 (8.8–9.9)	9.5 (8.9–9.9)	0.794	< 0.001	< 0.001	0.139
NLR	1.7 (1.4–2.3)	2.1 (1.5–2.6)	3.4 (2.6–5.0)	< 0.001	0.001	0.001	0.788
PLR	110.3 (99.2–140.0)	108.5 (95.6–140.4)	186.2 (115.1–290.8)	0.001	0.527	0.916	0.622
ELR	0.055 (0.029–0.095)	0.074 (0.018–0.097)	0.079 (0.300–0.140)	0.057	0.840	0.053	0.118
dNLR	1.26 (1.07–1.63)	1.36 (1.00–1.76)	2.09 (1.59–2.38)	0.063	0.576	0.049	0.229
hsCRP, mg/L	0.8 (0.4–1.1)	2.4 (1.1–6.5)	3.0 (2.2–4.7)	< 0.001	< 0.001	< 0.001	0.339

[a]Mann-Whitney U test. [b]Krusal-wallis test. [c]One-way ANOVA test. [d]Tukey's test
WBC: White blood cell, NLR: Neutrophils to lymphocytes ratio, PLR: Platelet to lymphocyte ratio, MPV: Mean platelet volume, hsCRP: High-sensitivity C-reactive protein, SCORAD: Scoring atopic dermatitis index, ELR: Eosinophil-to-lymphocyte ratio

Table 3. Diagnostic accuracy of different formulae with regard to high SCORAD score

	AUROC (95% CI)	*P*-value	Cut-off	Sensitivity, %	Specificity, %
MPV, fL	0.530 (0.391–0.668)	0.663	9.35	59.1	55.6
NLR	0.805 (0.707–0.903)	< 0.001	2.59	77.3	80.0
PLR	0.754 (0.621–0.886)	< 0.001	174.5	59.1	90.0
ELR	0.589 (0.447–0.731)	0.196	0.116	36.4	86.4
dNLR	0.737 (0.610–0.863)	0.001	1.85	72.7	83.0
hsCRP, mg/L	0.734 (0.639–0.828)	< 0.001	1.78	90.9	61.1

NLR: Neutrophils to lymphocytes ratio, PLR: Platelet to lymphocyte ratio, MPV: Mean platelet volume, hsCRP: High-sensitivity C-reactive protein, AUROC: Area under the receiver operating characteristic curve, CI: Confidence interval, SCORAD: Scoring atopic dermatitis index

36.4% and a specificity of 86.4%. dNLR showed moderate diagnostic performance, with an AUROC of 0.703 (95% CI: 0.610–0.863; $P = 0.001$). Using a cut-off value of 1.85, dNLR achieved a sensitivity of 72.7% and a specificity of 83.0%. However, hsCRP demonstrated an AUROC of 0.734 (95% CI: 0.639–0.828; $P < 0.001$), characterized by high sensitivity (90.9%) but lower specificity (61.1%). A comparative analysis using the DeLong test confirmed that NLR was the most reliable predictor of high SCORAD scores, outperforming the other indices.

Discussion

Principal Finding

The study group had a median SCORAD score of 30.7 (15.1–40.2), with 65.2% of patients classified as moderate to severe. Regarding inflammatory indices, the median NLR in adult AD patients was 2.1 (1.5–3.0), and the median serum hsCRP was 1.7 (0.8–4.0) mg/L. Neutrophils, NLR, PLR, and hsCRP increased significantly with increasing SCORAD scores. Among them, NLR and hsCRP were the most reliable indicators of disease severity. NLR, in particular, showed the strongest diagnostic performance, with an AUROC of 0.805 (95% CI: 0.707–0.903; $P < 0.001$), a cut-off of 2.59, sensitivity of 77.3%, and specificity of 80.0%.

Comparing with Previous Studies

In our study, 26.8% of patients were aged ≥ 60 years, consistent with Chan et al.[11] in the United Kingdom, who reported a prevalence of 7.7% in adults and 11.6% among the elderly, with more severe disease in the latter group. This highlights the importance of early detection in older adults. A female predominance was also observed (62.5%; female-to-male ratio = 1.67:1), aligning with findings of Mora et al.[12] in Spain (59.4%; 1.46:1) and Koppes et al.[13] (63.3%). This pattern has been attributed to estradiol and other female sex hormones, which may exacerbate Th2-mediated inflammation in AD. Supporting evidence from animal studies shows that estradiol can directly promote mast cell degranulation and trigger allergic sensitization.[14-16]

Comorbid allergic conditions were common, with 16.1% of patients reporting allergic rhinitis and 8.9% reporting asthma. These rates were lower than those reported in Ravnborg et al.[17] meta-analysis (25.7% for asthma; 21% in physician-diagnosed cases) and in Knudgaard et al.,[18] who found allergic rhinitis in 40.5% of patients with AD compared with

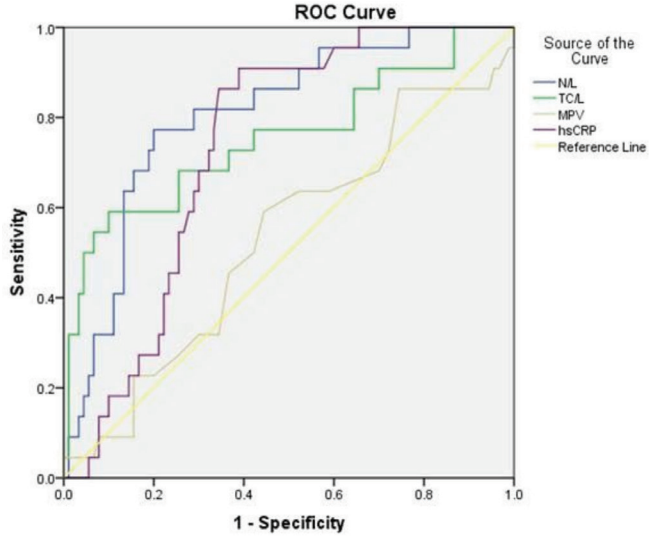

Figure 1. Receiver operating characteristic (ROC) curves showing the sensitivity and specificity of NLR, PLR, MPV, and hsCRP with respect to Group III SCORAD (51–103). NLR (blue line), PLR (green line), MPV (beige line), and hsCRP (purple line); the reference line is shown in yellow

NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, MPV, Mean platelet volume, hsCRP: High-sensitivity C-reactive protein, SCORAD: Scoring atopic dermatitis index

18.0% of controls. Such variability may reflect differences in population characteristics, diagnostic criteria, and study design. The frequent comorbidity of AD, asthma, and allergic rhinitis, often described as the "atopic triad", reflects shared pathogenic mechanisms, including immunoglobulin E (IgE) elevation, Th1/Th2 imbalance, and mast cell activation, supporting the concept of the atopic march.

Our results showed that total leukocyte count and NLR increased significantly with higher SCORAD scores, reflecting systemic inflammation in AD. This trend is consistent with the findings of Sekar et al.,[19] who reported significant increases in both NLR and PLR from mild to severe groups, although the absolute values in our study were lower. In contrast, MPV did not differ significantly across severity groups, which is inconsistent with Sekar et al.,[19] who observed a significant decrease, but consistent with Batmaz,[20] who reported no significant differences in NLR, PLR, or MPV. These discrepancies suggest that NLR may be influenced by population characteristics and study design; nonetheless, recent evidence, particularly from Sekar et al.,[19] reinforces the value of NLR and PLR, especially NLR, markers for disease severity stratification. Similarly, Dogru and Citli[21] demonstrated that, in children, NLR increased with higher SCORAD scores and was positively correlated with disease severity, supporting the presence of systemic inflammation at an early stage. NLR, which reflects the imbalance between innate and adaptive immunity, therefore emerges as a simple, low-cost, and reliable inflammatory marker for severity stratification in both pediatric and adult patients, with particular utility in resource-limited clinical settings. Regarding hsCRP, our findings align with those of Vekaria et al.[22] and Nguyen and Chau,[23] confirming that CRP levels increase with disease severity and can be used as a stratification tool. In contrast, MPV did not show significant differences in our study, consistent with Batmaz,[20] but contrary to Sekar et al.[19] and Bostan Gayret et al.[24] This indicates that MPV is more susceptible to variability related to population differences and laboratory conditions. Taken together, NLR, PLR, and hsCRP, particularly NLR, appear to be reliable inflammatory markers for assessing disease severity and monitoring disease progression; however, MPV currently lacks the stability required for routine clinical application.

Analysis of predictive markers for severe AD (group III) in our study identified NLR as the most prominent indicator. This finding is consistent with Jiang and Ma,[5] who reported an AUC of 0.778 with a lower cut-off value (1.75), yielding a sensitivity above 90% but a specificity of only 58.6%. NLR reflects immune imbalance: neutrophil counts rise as part of the inflammatory response, whereas lymphocytes—particularly regulatory T cells—decline, impairing the regulation of inflammation.[25] Consequently, elevated NLR is typically associated with more severe disease and extensive skin involvement. Supporting this, Inokuchi-Sakata et al.[25] demonstrated a direct relationship between NLR and SCORAD scores. Beyond AD, numerous studies have linked NLR with other allergic and autoimmune diseases, including pediatric allergic rhinitis (Dogru and Citli),[21] asthma, and systemic lupus erythematosus.[26-28] These lines of evidence strengthen the biological rationale for applying NLR as a prognostic marker of disease severity in AD.

From a pathophysiological perspective, interleukin (IL)-17 plays a pivotal role in chronic cutaneous inflammation. It not only promotes neutrophil production but also induces the release of tissue-degrading enzymes (such as metalloproteinases and elastase) and reactive oxygen species, thereby exacerbating skin damage and disease severity.[29] At the same time, reduced regulatory T-cell activity limits the ability to suppress inflammation, sustaining a chronic inflammatory state that is difficult to control.[30] These mechanisms provide a biological explanation for why patients with elevated NLR often present with more severe clinical manifestations, poorer treatment response, and a higher likelihood of persistent disease.

Study Limitations

Our study has several limitations. First, the study was conducted at a single center with a relatively modest sample size; therefore, the findings may not fully capture the diversity of the epidemiological and clinical characteristics of AD patients across regions of Vietnam. Second, the cross-sectional design only allowed assessment of associations at a single time point, without evaluating longitudinal changes in MPV, NLR, PLR, ELR, dNLR, and hsCRP during disease progression or after treatment. Third, we focused exclusively on four peripheral blood inflammatory markers and did not include other immuno-inflammatory markers such as cytokines (IL-4, IL-13, IL-17), serum IgE, or novel molecular biomarkers, all of which could further elucidate disease pathogenesis and enhance predictive value. Fourth, due to limited resources and technical constraints, we were unable to include a well-matched healthy control group stratified by age, sex, and comorbidities, which limits the ability to fully exclude potential confounding factors.

Clinical Implications

The findings of this study suggest that routine, low-cost, and easily implemented blood tests —MPV, NLR, PLR, ELR, dNLR, and hsCRP— may serve as useful adjunctive tools for assessing disease severity in AD. Incorporating these indices into clinical practice could enable physicians to identify patients at higher risk of severe disease at an earlier stage, thereby facilitating closer monitoring and timely adjustment

of therapeutic regimens. In addition, monitoring changes in NLR and hsCRP during treatment may provide valuable information on therapeutic response, helping to guide treatment strategies and improve disease control.

Conclusion

The study demonstrated that total leukocyte count, NLR, PLR, and hsCRP all increased progressively with higher SCORAD scores, reflecting systemic inflammation in patients with AD. Among these markers, NLR showed the strongest diagnostic performance, with a cut-off value of 2.59, suggesting its potential as a simple, low-cost, and easily applicable clinical indicator for risk stratification and treatment decision-making. Multicenter studies with longitudinal designs are warranted to validate and further explore the clinical utility of these indices in routine practice.

Ethics

Ethics Committee Approval: This study was approved approval from the Ethics Committee in Biomedical Research of Ho Chi Minh City Dermatology Hospital (approval number: 418/CN-BVDL, date: 29.02.2024).

Informed Consent: Written informed consent was obtained from all adult participants prior to enrollment, consistent with the Declaration of Helsinki (revised 2013).

Footnotes

Conflict of Interest: The author declared that they have no conflict of interest.

Financial Disclosure: The author declare that this study received no financial support.

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