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Adipokine–Eosinophil Imbalance in Pediatric Atopic Dermatitis: Omentin-to-Eosinophil Ratio as a Novel Composite Biomarker

Serdar HIRA

mdserdarhira@gmail.com

Royal Hospital

Fatih ÇELİKEL

Sultan Abdulhamid Research and Training Hospital

Ömer ÖZCAN

Amsterdam UMC Location VUmc

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Abstract

Atopic dermatitis (AD) is increasingly recognized as a systemic immunometabolic disorder extending beyond cutaneous inflammation. Adipokines such as chemerin and omentin-1 may link metabolic and immune activation; however, pediatric data remain limited. We aimed to evaluate adipokine–eosinophil imbalance and investigate the omentin-to-eosinophil ratio (OER) as a composite biomarker in pediatric AD. This case–control study compared serum chemerin and omentin-1 levels in 40 children with AD and 45 healthy controls and evaluated adipokine–eosinophil composite biomarkers. Serum adipokines were measured by ELISA, and the chemerin-to-eosinophil ratio (CER) and OER were calculated. Children with AD demonstrated significantly higher chemerin levels (91.19±80.74 vs 58.22±47.42 ng/mL, p=0.027) and lower omentin-1 concentrations (5.01±2.57 vs 9.01±5.26 ng/mL, p<0.001) compared with controls. OER values were markedly reduced in the AD group (9.18±4.31 vs 25.18±18.97, p<0.001), whereas CER did not differ significantly. In ROC analyses, OER demonstrated the strongest discriminatory performance for pediatric AD (AUC=0.853), exceeding the performance of omentin-1 (AUC=0.740) and chemerin (AUC=0.638). In multivariable analyses adjusted for age and BMI, lower OER values remained independently associated with pediatric AD. Exploratory analyses additionally demonstrated an inverse correlation between OER and SCORAD scores. These findings demonstrate a distinct adipokine–eosinophil imbalance in pediatric AD and support the concept of AD as a systemic immunometabolic disorder. Composite biomarkers such as OER may provide clinically meaningful insight beyond isolated inflammatory markers alone.

Introduction

Atopic dermatitis (AD) is a chronic relapsing inflammatory skin disease characterized by epidermal barrier dysfunction, immune dysregulation, microbiome alterations, and type 2 inflammatory activation [1]. Although traditionally regarded as a cutaneous disorder, increasing evidence suggests that AD represents a systemic immunometabolic disease associated with persistent low-grade inflammation extending beyond the skin [2, 3]. This systemic inflammatory milieu may contribute to metabolic, vascular, and immune alterations beginning early in life, particularly in pediatric patients [3].

Adipose tissue functions as an active inflammatory and metabolic organ through the secretion of adipokines that regulate inflammatory signaling, endothelial function, and metabolic homeostasis [4]. Among these mediators, chemerin and omentin-1 appear to exert biologically opposing effects. Chemerin promotes leukocyte chemotaxis and pro-inflammatory immune activation through the CMKLR1 pathway, whereas omentin-1 demonstrates predominantly anti-inflammatory and insulin-sensitizing properties, partly via suppression of NF-κB signaling [5, 6]. Dysregulation of these adipokines has been implicated in several chronic inflammatory and metabolic disorders; however, their roles in pediatric AD remain insufficiently characterized [7].

Most previous studies in AD have focused on isolated inflammatory or adipokine markers, whereas the interaction between adipokine signaling and eosinophilic inflammation has received limited attention,

particularly in pediatric populations [8]. Because eosinophils are key effector cells of type 2 inflammation in AD, integrating eosinophilic activity with adipokine-related pathways may yield more biologically relevant biomarkers than assessment of circulating adipokines alone [8, 9]. Eosinophil-adjusted adipokine indices may therefore better reflect the integrated inflammatory and metabolic alterations underlying pediatric AD [9]. Composite indices reflecting multiple inflammatory domains have increasingly emerged as promising tools for improving disease discrimination and clinical interpretation in chronic inflammatory disorders [10]. However, adipokine–eosinophil composite biomarkers have not been adequately investigated in pediatric AD.

Therefore, this study aimed to evaluate serum chemerin and omentin-1 levels in children with AD and to investigate the omentin-to-eosinophil ratio (OER), a novel composite immunometabolic biomarker integrating adipokine signaling with eosinophilic inflammation. To the best of our knowledge, OER has not previously been investigated in pediatric AD or other inflammatory skin diseases. We additionally assessed its discriminatory and potential clinical relevance in pediatric AD.

Methods

Study Design and Participants

This case–control study included 40 children with AD and 45 age-matched healthy controls recruited from Tatvan State Hospital, Bitlis, Türkiye, between December 2015 and May 2016. The diagnosis of AD was established according to the Hanifin and Rajka criteria [11]. Children aged 1–16 years with complete clinical and laboratory data were eligible for inclusion.

Healthy controls were selected among children without allergic, inflammatory, infectious, metabolic, dermatologic, or chronic systemic diseases. Exclusion criteria for all participants included acute or chronic infection, asthma, allergic rhinitis, autoimmune disease, endocrine or metabolic disorders, obesity, malnutrition, malignancy, and recent use of systemic corticosteroids, topical corticosteroids, or antihistamines.

The study was conducted in accordance with the Declaration of Helsinki and approved by the local Ethics Committee (approval number: 971–8582, 09.11.2015). Written informed consent was obtained from the parents or legal guardians of all participants.

Clinical Assessment

Clinical severity was assessed using the Scoring Atopic Dermatitis (SCORAD) index in patients with AD [12]. No patients met criteria for severe AD in the present cohort.

Laboratory Measurements

Venous blood samples were collected in the morning after overnight fasting. Complete blood count analyses were performed using an automated hematology analyzer (Medonic CA-620, Boule Diagnostics AB, Stockholm, Sweden). Absolute eosinophil counts and neutrophil-to-lymphocyte ratio (NLR) values were recorded.

Serum samples were centrifuged and stored at − 40°C until analysis. Serum chemerin and omentin-1 concentrations were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits (Elabscience Biotechnology Co., Ltd., Houston, TX, USA) according to the manufacturer's instructions. Absorbance measurements were obtained at 450 nm using an ELx800 microplate reader (BioTek Instruments, Winooski, VT, USA). The assay ranges were 0.156–10 ng/mL for chemerin and 0.625–40 ng/mL for omentin-1, with detection limits of 0.094 ng/mL and 0.375 ng/mL, respectively. All samples were analyzed in duplicate, and intra-assay coefficients of variation were < 10%. Samples exceeding the upper detection range were appropriately diluted according to the manufacturer's instructions and reanalyzed.

Composite Biomarker Definitions

The chemerin-to-eosinophil ratio (CER) was calculated by dividing serum chemerin levels (ng/mL) by absolute eosinophil count (×10³/μL). The OER was calculated by dividing serum omentin-1 levels (ng/mL) by absolute eosinophil count (×10³/μL).

Statistical Analysis

Statistical analyses were performed using SPSS version 22.0 (IBM Corp., Chicago, IL, USA). Data distribution was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation or median (interquartile range), as appropriate. Comparisons between groups were performed using the independent samples t-test or Mann–Whitney U test according to data distribution.

Correlations between continuous variables were evaluated using Spearman's rank correlation coefficient. Receiver operating characteristic (ROC) curve analyses were performed to assess the discriminatory performance of chemerin, omentin-1, CER, and OER. The area under the curve (AUC), sensitivity, specificity, positive predictive value, negative predictive value, diagnostic accuracy, and optimal cut-off values were calculated.

Multivariable logistic regression analyses adjusted for age and body mass index (BMI) were performed to identify variables independently associated with pediatric AD. Internal model validation was performed using 5-fold cross-validation and bootstrap resampling analyses. Calibration performance was additionally evaluated using calibration plots and Brier scores.

A two-tailed p value < 0.05 was considered statistically significant.

Results

A total of 85 children were included in the study, comprising 40 patients with AD and 45 healthy controls. The groups were comparable with respect to age and BMI (Table 1). Absolute eosinophil counts and NLR values were significantly higher in children with AD compared with controls (both $p < 0.001$). The mean SCORAD value in the AD group was 28.90 ± 6.73, and no patients met criteria for severe disease.

Table 1
Baseline Characteristics and Core Inflammatory Parameters

Variable	AD (n = 40)	Controls (n = 45)	p value
Age (months)	47.8 ± 21.1	53.0 ± 24.1	0.291
BMI (kg/m²)	17.46 ± 1.18	17.57 ± 1.06	0.629
Eosinophil (×10³/μL)	0.56 ± 0.14	0.40 ± 0.15	< 0.001*
NLR	2.66 ± 0.52	1.89 ± 0.93	< 0.001*
SCORAD	28.90 ± 6.73	—	—

BMI: Body mass index; NLR: Neutrophil-to-lymphocyte ratio; SCORAD: Scoring Atopic Dermatitis index.**Statistically significant.*

Serum chemerin levels were significantly higher in the AD group compared with healthy controls (91.19 ± 80.74 vs 58.22 ± 47.42 ng/mL, $p = 0.027$), whereas serum omentin-1 concentrations were significantly lower (5.01 ± 2.57 vs 9.01 ± 5.26 ng/mL, $p < 0.001$) (Table 2). Absolute eosinophil counts were also elevated in the AD group. The OER was markedly reduced in children with AD (9.18 ± 4.31 vs 25.18 ± 18.97, $p < 0.001$), while the CER did not differ significantly between groups ($p = 0.776$).

Table 2
Serum Adipokines and Composite Ratios

Variable	AD (n = 40)	Controls (n = 45)	p value
Chemerin (ng/mL)	91.19 ± 80.74	58.22 ± 47.42	0.027*
Omentin-1 (ng/mL)	5.01 ± 2.57	9.01 ± 5.26	< 0.001*
CER	148.57 ± 93.15	142.39 ± 106.70	0.776
OER	9.18 ± 4.31	25.18 ± 18.97	< 0.001*

Abbreviations: CER: Chemerin-to-eosinophil ratio; OER: Omentin-to-eosinophil ratio.

**Statistically significant.*

ROC curve analyses demonstrated that OER had the highest discriminatory performance for pediatric AD, with an AUC of 0.853, sensitivity of 87.5%, specificity of 77.8%, and an optimal cut-off value of 13.52

(Fig. 1, Table 3). Omentin-1 also demonstrated acceptable discriminatory ability (AUC = 0.740), whereas chemerin showed relatively limited performance (AUC = 0.638). In contrast, CER did not demonstrate meaningful discriminatory value.

Table 3
Diagnostic Performance and Internal Validation of OER-Based Models

Metric	Value
Cross-validated AUC (Model 2)	0.859
Bootstrap mean AUC	0.860
Bootstrap 95% CI lower	0.774
Bootstrap 95% CI upper	0.924
Brier score	0.155
AUC of OER (single marker)	0.853
AUC of Omentin-1 (single marker)	0.740
AUC of Chemerin (single marker)	0.638
AUC of NLR	0.850
Mean AUC difference (OER vs Omentin-1)	0.118
Bootstrap p value for AUC comparison	0.018*
Optimal OER cut-off value	13.52
Sensitivity (%)	87.5
Specificity (%)	77.8
Positive predictive value (%)	77.8
Negative predictive value (%)	87.5
Overall diagnostic accuracy (%)	82.4

In multivariable logistic regression analyses adjusted for age and BMI, elevated chemerin levels and reduced omentin-1 concentrations were independently associated with pediatric AD (Table 4). In the alternative model, lower OER values also remained independently associated with AD after multivariable adjustment. Internal validation analyses demonstrated stable model performance, with consistent cross-validated and bootstrap-derived AUC values and acceptable calibration characteristics. An additional exploratory model including NLR is presented in Supplementary Table 2.

Table 4. Multivariable Logistic Regression Models for Pediatric Atopic Dermatitis

Model 1: Chemerin + Omentin-1 + Age + BMI

Predictor	OR	95% CI	p value
Chemerin (per 1 SD increase)	3.13	1.45–6.76	0.004*
Omentin-1 (per 1 SD increase)	0.17	0.07–0.42	<0.001*
Age (per 1 SD increase)	0.85	0.50–1.43	0.533
BMI (per 1 SD increase)	0.94	0.57–1.56	0.815

Model 2: OER + Age + BMI

Predictor	OR	95% CI	p value
OER (per 1 SD increase)	0.025	0.004–0.144	<0.001*
Age (per 1 SD increase)	0.94	0.53–1.66	0.825
BMI (per 1 SD increase)	0.86	0.50–1.49	0.597

OR: Odds ratio; CI: Confidence interval; BMI: Body mass index; OER: Omentin-to-eosinophil ratio.**Statistically significant*.

Exploratory correlation analyses within the AD group demonstrated a moderate inverse association between SCORAD and OER (Spearman r=−0.612, p < 0.001) (Supplementary Table 1, Supplementary Fig. 1). SCORAD values were inversely correlated with omentin-1 levels and OER values. A modest positive correlation was additionally observed between SCORAD and chemerin levels, whereas associations with eosinophil counts and NLR did not reach statistical significance.

Discussion

The present study demonstrated a distinct adipokine–eosinophil imbalance in pediatric AD characterized by elevated chemerin levels, reduced omentin-1 concentrations, increased eosinophilic inflammation, and markedly decreased OER values. Among the investigated biomarkers, OER demonstrated the strongest discriminatory performance for pediatric AD and remained independently associated with disease status after adjustment for potential confounders. These findings suggest that integrating adipokine signaling with eosinophilic inflammation may provide biologically meaningful insight beyond isolated adipokine measurements alone.

Chemerin is a multifunctional adipokine that integrates metabolic signaling with innate and adaptive immune activation through the CMKLR1 pathway and contributes to leukocyte chemotaxis and inflammatory regulation [13]. In AD, most previous evidence has focused on cutaneous chemerin expression rather than circulating levels. Experimental studies have demonstrated increased chemerin

expression in inflamed skin and its involvement in immune cell recruitment; however, data regarding circulating chemerin concentrations in AD remain limited and inconsistent, particularly in pediatric populations [2, 14]. In the present study, children with AD demonstrated significantly elevated serum chemerin levels compared with healthy controls, supporting the concept that chemerin dysregulation in AD may reflect systemic inflammatory and metabolic activation rather than localized cutaneous inflammation alone. Similar elevations in circulating chemerin have also been reported in several chronic inflammatory disorders, further supporting its potential role as a marker of systemic inflammatory burden [15].

In contrast, omentin-1 is generally regarded as an anti-inflammatory and insulin-sensitizing adipokine that suppresses pro-inflammatory signaling partly through inhibition of NF-κB pathways [6, 16]. Although reduced circulating omentin-1 levels have been reported in several inflammatory and metabolic disorders, data in AD, particularly in pediatric populations, remain limited [6, 16]. The significantly lower omentin-1 concentrations observed in our cohort suggest impaired anti-inflammatory adipokine activity and disrupted metabolic–immune crosstalk in pediatric AD. Interestingly, omentin-1 levels demonstrated an inverse correlation with SCORAD scores, supporting a potential relationship between reduced anti-inflammatory adipokine signaling and increasing disease severity. The absence of strong correlations between omentin-1 and routine hematologic inflammatory indices may further indicate that omentin-related inflammation reflects a more chronic immunometabolic component not fully captured by conventional blood markers.

The most notable finding of the present study was the strong discriminatory performance of OER compared with individual adipokine measurements alone. To the best of our knowledge, this is the first study to propose and evaluate OER as a composite adipokine–eosinophil biomarker in pediatric AD. Unlike isolated biomarkers, OER integrates anti-inflammatory adipokine activity with eosinophilic inflammation, thereby capturing multiple immunologic domains involved in AD pathogenesis [10, 17]. The markedly reduced OER values observed in pediatric AD suggest that progressive eosinophilic activation accompanied by impaired anti-inflammatory adipokine signaling may represent a biologically relevant immunometabolic imbalance in the disease. In contrast, CER did not demonstrate meaningful discriminatory utility, which may indicate that combining eosinophilic inflammation with anti-inflammatory rather than pro-inflammatory adipokine pathways provides greater biological and diagnostic relevance in pediatric AD.

Exploratory severity analyses demonstrated a moderate inverse association between SCORAD scores and OER values, suggesting that worsening clinical severity may be accompanied by progressive adipokine–eosinophil imbalance. Reduced omentin-1 levels were also associated with higher SCORAD scores, further supporting the potential relationship between impaired anti-inflammatory adipokine activity and increasing disease severity. Although these findings should be interpreted cautiously because no patients with severe AD were included in the cohort, they nevertheless suggest that inflammatory and metabolic dysregulation may parallel clinical disease activity in pediatric AD.

Beyond their potential utility as circulating biomarkers, both adipokines may also represent biologically relevant therapeutic pathways. Experimental modulation of the chemerin/CMKLR1 axis, including chemerin-derived peptides such as C15, has been shown to suppress allergic inflammation and promote tissue repair in preclinical models [18, 19]. Similarly, exogenous omentin-1 has demonstrated anti-inflammatory and cytoprotective effects through activation of Nrf2/AMPK signaling and inhibition of NF-κB-mediated pathways [20, 21]. Although direct clinical translation remains limited, these findings further support the biological relevance of adipokine-centered mechanisms in AD.

Although NLR demonstrated discriminatory performance comparable to OER in the present cohort, several biological considerations support the potential added relevance of OER in pediatric AD. Unlike NLR, which primarily reflects nonspecific systemic hematologic inflammation, OER integrates eosinophilic type 2 immune activation with adipokine-related immunometabolic signaling [8, 22]. Therefore, OER may provide greater pathophysiological specificity by simultaneously capturing inflammatory and metabolic components of AD pathogenesis. This distinction may be particularly relevant in pediatric AD, where chronic low-grade immunometabolic dysregulation may not be fully represented by routine hematologic indices alone. Accordingly, the comparable diagnostic performance of OER and NLR in the current study should not necessarily be interpreted as biological equivalence, but rather as evidence that composite adipokine–eosinophil biomarkers may offer mechanistically complementary information beyond conventional inflammatory markers.

Several limitations of the present study should be acknowledged. First, the relatively small sample size and single-center design may limit the generalizability of the findings. Second, the cross-sectional nature of the study precludes causal interpretation of the observed associations. Third, no patients with severe AD were included, which may have limited the interpretation of severity-related analyses. Additionally, cytokines, immunoglobulin E levels, and other adipokines relevant to AD pathophysiology were not evaluated. The study cohort was recruited between 2015 and 2016. However, because adipokine pathways represent biologically stable immunometabolic mechanisms rather than treatment-dependent markers, temporal shifts in therapeutic approaches are unlikely to substantially influence the observed associations. Although internal validation demonstrated stable performance, the relatively limited sample size may still predispose the model to optimism bias, and external multicenter validation is necessary before clinical translation. Finally, external validation and longitudinal follow-up studies are required to confirm the clinical applicability and long-term relevance of OER in pediatric AD. Despite these limitations, the present study provides novel pediatric evidence supporting the role of systemic adipokine–eosinophil imbalance in AD.

In conclusion, pediatric AD is associated with a distinct adipokine–eosinophil imbalance characterized by elevated chemerin levels, reduced omentin-1 concentrations, and markedly decreased OER values, further supporting the concept of AD as a systemic immunometabolic disorder. Among the investigated biomarkers, OER demonstrated the strongest discriminatory performance and remained independently associated with pediatric AD after multivariable adjustment. The observed adipokine alterations, partly independent of routine hematologic inflammatory indices, suggest that adipokine-related pathways may

reflect a chronic inflammatory and metabolic component of inflammation not fully captured by conventional markers. Collectively, these findings support the potential clinical relevance of composite adipokine–eosinophil biomarkers in pediatric AD. Further multicenter and longitudinal studies are warranted to validate the clinical utility of OER.

Declarations

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

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Data Availability Statement: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions: Conceptualization: SH; Methodology: SH, FÇ; Formal analysis: SH; Data collection: FÇ; Writing-original draft: SH; Writing-review and editing: ÖÖ; Supervision: ÖÖ.

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Figures

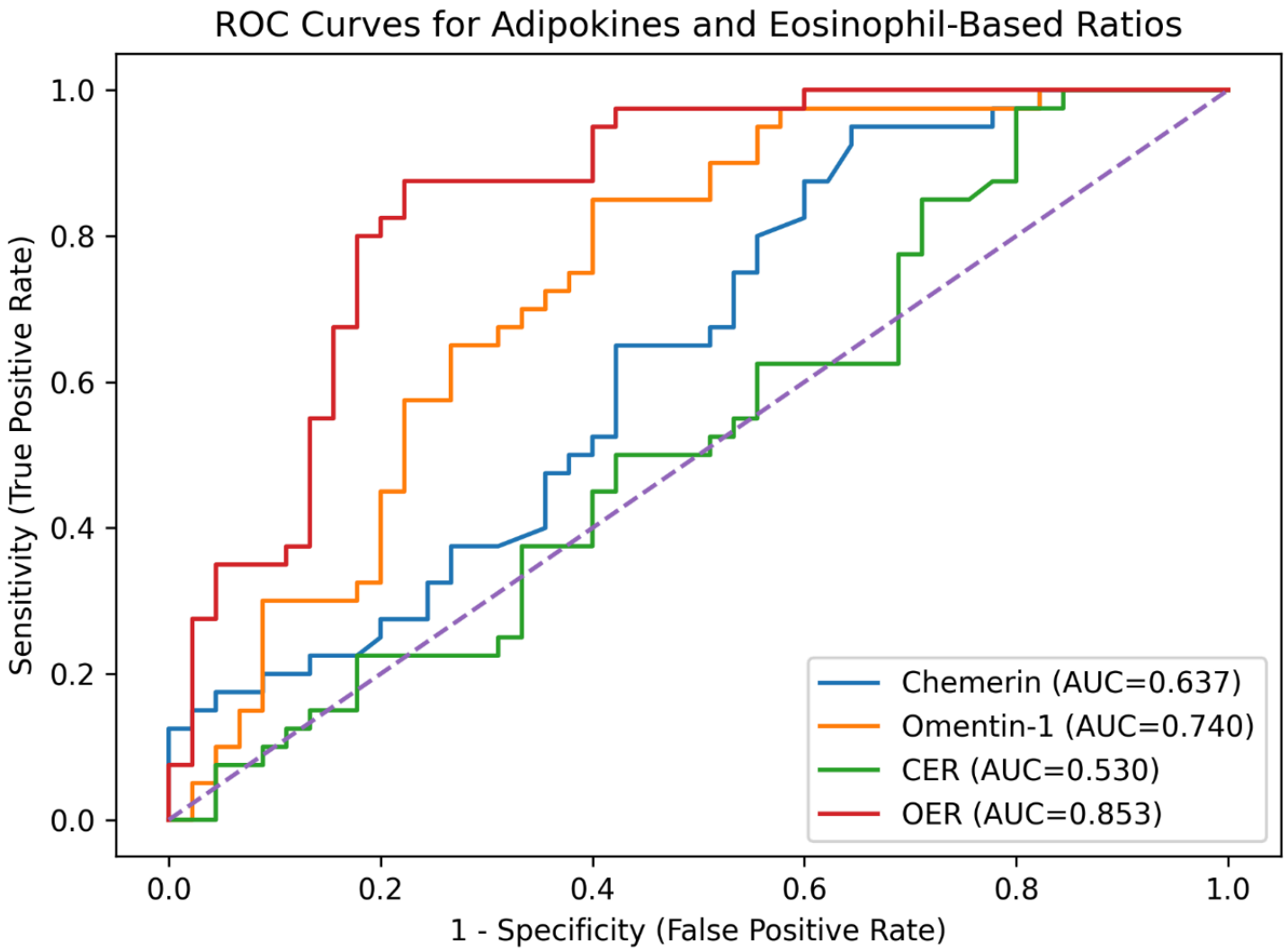


Figure 1

Receiver Operating Characteristic (ROC) Curves for OER, Omentin-1, Chemerin, and CER in Pediatric Atopic Dermatitis

Receiver operating characteristic (ROC) curve analysis demonstrating the discriminatory performance of the omentin-to-eosinophil ratio (OER), omentin-1, and chemerin for distinguishing children with atopic dermatitis from healthy controls. OER demonstrated the highest diagnostic performance (AUC = 0.853), followed by omentin-1 (AUC = 0.740), and chemerin (AUC = 0.638).

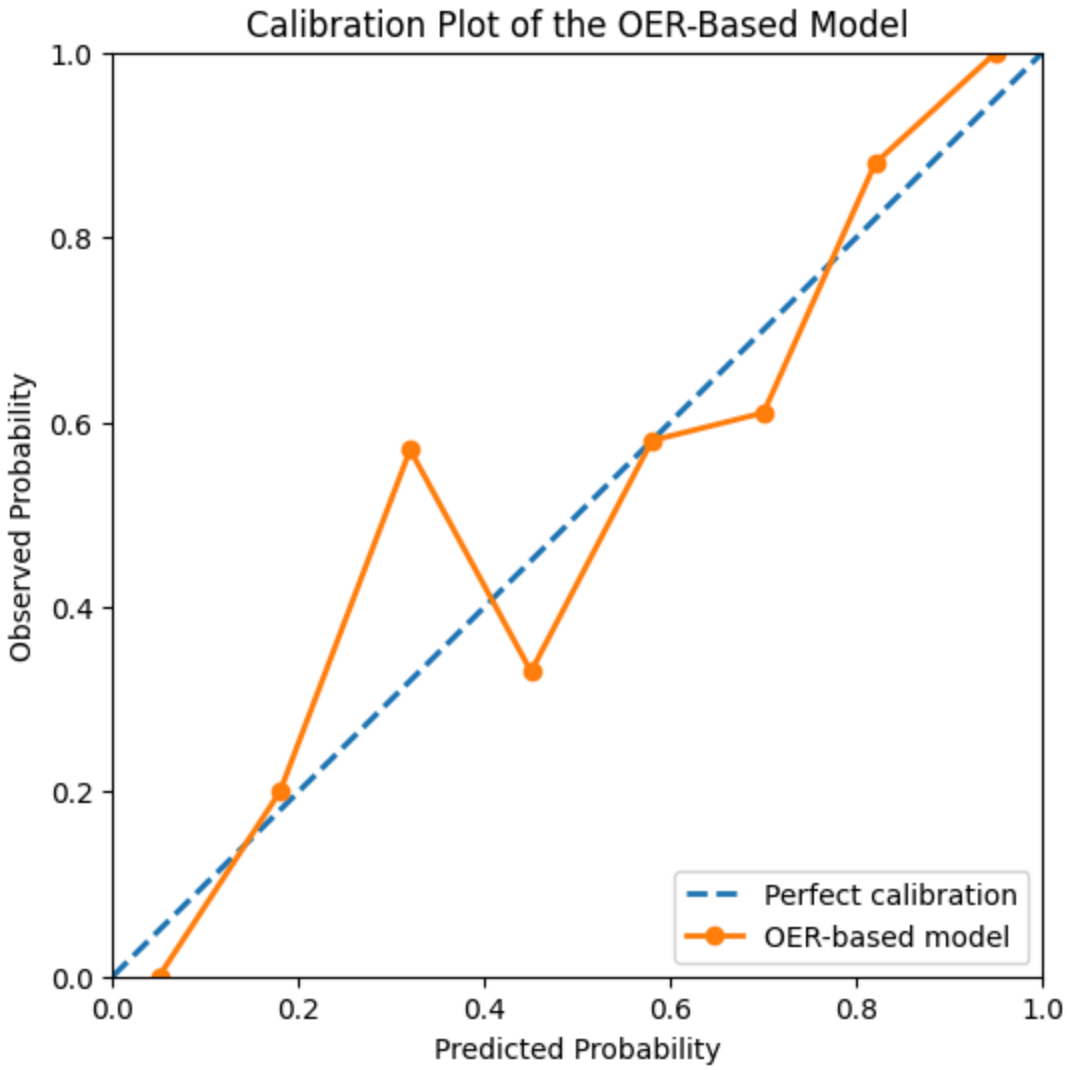


Figure 2

Calibration Plot of the OER-Based Logistic Regression Model

Calibration plot demonstrating the agreement between predicted and observed probabilities for the OER-based logistic regression model in pediatric atopic dermatitis. The dashed diagonal line represents perfect calibration, whereas the solid line indicates the observed model performance. The model demonstrated good calibration across the prediction range, supporting the internal validity of the OER-based classifier.

Supplementary Files

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