

Seborrheic Dermatitis Exhibits Only a Modest Systemic Inflammatory Signature: A Retrospective Case-Control Study

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Abstract

Blood count-derived indices have been proposed as markers of systemic inflammation in inflammatory skin diseases, but data on seborrheic dermatitis are limited. We aimed to compare 9 blood count-derived indices, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) between patients with seborrheic dermatitis and healthy controls and to examine their associations with disease severity and 4 other clinical features. This retrospective case-control study used medical records (2023–2025) from a university dermatology department, including 142 adults with seborrheic dermatitis and 72 healthy adults without comorbidity. We estimated adjusted odds ratios (aORs) per 1-SD increase and areas under the receiver operating characteristic curve (AUCs), with false discovery rate (FDR)-adjusted q values. The pan-immune-inflammation value (PIV; aOR, 1.75; 95% CI, 1.10–2.77; q = .05) and the hemoglobin-to-platelet ratio (HPR; aOR, 0.56; 95% CI, 0.39–0.81; q = .006) differed between groups, but both discriminated poorly (AUC, 0.60 and 0.61), and the other 7 indices did not differ. CRP and ESR discriminated better (AUC, 0.73 and 0.80). Among the 88 patients without comorbidity, PIV (q = .17) and HPR (q = .06) no longer met the FDR threshold, but CRP and ESR remained associated (both q < .001). No marker was associated with disease severity or any other clinical feature (all q $\geq$.06). Blood count-derived indices differed only modestly between patients and controls, the differences were sensitive to the exclusion of patients with comorbidity, and no index was associated with severity. These indices do not appear useful for assessing seborrheic dermatitis severity.

Introduction

Seborrheic dermatitis is a common chronic inflammatory skin disease with a pooled global prevalence of 4.38% (5.64% in adults) [1]. *Malassezia*-derived free fatty acids are thought to activate pattern recognition receptors and the inflammasome in the skin, which leads to the release of interleukin 1 β and other cytokines [2, 3]. Recent reviews place host factors, including immune dysregulation and epidermal barrier dysfunction, at the center of the disease [4, 5], and a large database study linked it to inflammatory diseases of other epithelial barriers [6]. Whether this local inflammation produces a measurable systemic signal is unknown.

Some studies suggest that indices calculated from the complete blood count may serve as simple markers of systemic inflammation. The neutrophil-to-lymphocyte ratio (NLR), the systemic immune-inflammation index (SII) [7], and related indices have been associated with disease severity in psoriasis [8, 9] and hidradenitis suppurativa (HS) [10]. The pan-immune-inflammation value (PIV), which combines neutrophil, platelet, monocyte, and lymphocyte counts, was first shown to be prognostic in metastatic colorectal cancer [11, 12] and has since been linked to psoriasis severity [13], HS severity (area under the curve [AUC], 0.79) [14], systemic involvement in pediatric Behçet disease [15], and rapidly progressive interstitial lung disease in anti-MDA5 dermatomyositis [16]. Elevated NLR has also been associated with in-hospital mortality in adults with dermatomyositis [17].

Evidence in seborrheic dermatitis is limited. The only earlier study of blood count-derived markers that we identified (100 patients and 74 controls) found higher platelet-to-lymphocyte ratio (PLR), mean platelet volume (MPV), and C-reactive protein (CRP) in patients, and NLR, PLR, MPV, and SII did not correlate with disease severity, but red cell distribution width did [18]. A later letter questioned whether MPV reflects inflammation in seborrheic dermatitis [19]. To our knowledge, PIV, the systemic inflammation response index (SIRI), the monocyte-to-lymphocyte ratio (MLR), the derived NLR (dNLR), the neutrophil-to-platelet ratio, and the hemoglobin-to-platelet ratio (HPR) have not been evaluated in this disease. We sought to compare 9 blood count-derived indices, CRP, and erythrocyte sedimentation rate (ESR) between patients with seborrheic dermatitis and healthy controls and to assess their associations with disease severity and other clinical features.

Methods

Study design and participants

We conducted a retrospective, single-center case-control study using medical records from the dermatology department of Istanbul University-Cerrahpaşa, Istanbul, Türkiye, covering January 2023 to December 2025. The study was approved by the institutional ethics committee (see Statements and Declarations) and followed the Declaration of Helsinki and the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline [20].

Patients were adults (aged ≥ 18 years) with a clinical diagnosis of seborrheic dermatitis made by a dermatologist. Controls were adults who attended the same department for cosmetic or other unrelated reasons, had no systemic comorbidity, and had blood test results in the same records. We required controls to be free of comorbidity so that no other condition could alter their blood counts. Patients with comorbidity were not excluded. In our department, blood tests are requested during routine care for some patients without a fixed protocol, and not selectively for patients about to start systemic therapy or another intervention.

Exclusion criteria for both groups were acute infection within 4 weeks, current systemic corticosteroid or immunomodulator therapy, malignancy, autoimmune disease, and pregnancy. Controls were not individually matched to patients, so age and sex were included as covariates in all case-control models. Because controls had no comorbidity, it could not be a covariate in these models; we examined its influence in a sensitivity analysis restricted to patients without comorbidity.

Clinical data

We abstracted age, sex, body mass index (BMI), smoking (pack-years), comorbidity, disease duration, lesion distribution, pruritus, and treatment in the preceding month from the medical record. Comorbidity was defined as diabetes mellitus, thyroid disease, cardiovascular disease, or hypertension. Disease severity was scored at clinic visits with the Seborrheic Dermatitis Area and Severity Index (SDASI), which grades erythema, scaling, and infiltration across affected regions and weights them by the area involved

[18, 21]. Because published SDASI severity bands are not standardized, we used study-defined categories (mild, <12; moderate, 12-24; severe, >24). Extra-scalp involvement was defined as facial or truncal lesions in addition to scalp involvement, and pruritus was recorded as present or absent by patient report.

Laboratory measurements

Complete blood count, CRP, and ESR values were taken from the medical record, and each participant contributed 1 set of results. Complete blood counts were measured on a Sysmex XN-series analyzer, CRP by nephelometry, and ESR by the Westergren method. We calculated 9 indices: NLR, PLR, MLR, SII [7], SIRI [22], PIV [11], dNLR [23], neutrophil-to-platelet ratio, and HPR [24] (formulas in Table S1 in Online Resource 1).

Outcomes

The primary comparison was between patients with seborrheic dermatitis and controls. Within the patient cohort, we examined 6 clinical outcomes: continuous SDASI score; moderate-to-severe (SDASI ≥ 12) vs mild disease; extra-scalp involvement; chronic disease (duration ≥ 60 months; sensitivity threshold ≥ 120 months); treatment refractoriness (moderate or higher severity despite a documented prescription for topical antifungal or corticosteroid therapy in the preceding month; adherence could not be verified); and pruritus. Systemic therapy ($n = 5$) was examined descriptively. The investigators defined the outcomes and thresholds for this analysis, and the analyses were not registered in advance.

Statistical analysis

We compared groups with the Mann-Whitney U test, using Cliff δ as the nonparametric effect size, and with the χ^2 or Fisher exact test, and we used Spearman correlation to relate SDASI to each marker. We standardized each marker (z score) and entered it separately into multivariable models, per 1-SD increase, because the indices share inputs and are highly correlated (Spearman ρ , 0.62-0.92 among NLR, SII, SIRI, PIV, and dNLR). Logistic regression was used for binary outcomes (adjusted odds ratios [aORs]) and linear regression for the continuous SDASI score. Case-control models were adjusted for age, sex, BMI, and pack-years of smoking, which are associated with blood counts and CRP [25, 26]; models within the patient cohort were also adjusted for comorbidity.

Discrimination was quantified as the AUC of the receiver operating characteristic curve, with 95% CIs from 1000 bootstrap resamples, and cutoffs were chosen by maximizing the Youden J statistic [27]. We interpreted an AUC of 0.5 as no discrimination and 0.7 to 0.8 as acceptable discrimination [28]. A composite model that added CRP, ESR, and PIV to clinical covariates was evaluated with 500-replicate bootstrap optimism correction [29] (a correction for overfitting). The false discovery rate (FDR) was controlled with the Benjamini-Hochberg procedure [30] within each outcome, across the markers tested for that outcome, and q values are reported.

Subgroups defined by sex, age category, and comorbidity were examined with formal tests of interaction. Sensitivity analyses used (1) a chronic disease threshold of at least 120 months, (2) restriction to patients without comorbidity, and (3) sex-stratified analyses of hemoglobin- and platelet-containing markers, which we considered exploratory. We did not calculate a sample size and included all eligible patients in the study period. Missing data were rare (<5%), and we used complete-case analyses. Analyses were performed in Python, version 3.12. Two-sided $P < .05$ ($q < .05$ for FDR-adjusted analyses) was considered statistically significant.

Results

Participants

After exclusion of 1 patient with an implausible lymphocyte count, the analysis included 142 patients with seborrheic dermatitis and 72 controls (Table 1). The groups were similar in age (median, 28.0 vs 29.0 years), sex (45.1% vs 43.1% men), BMI, and pack-years of smoking. Comorbidity was present in 54 patients (38.0%) and, by design, in none of the controls. The median SDASI score was 10 (IQR, 8-15); 77 patients (54.2%) had mild disease and 65 (45.8%) had moderate-to-severe disease.

Blood counts, indices, and acute-phase reactants

Patients had higher monocyte counts (median, 0.57 vs $0.50 \times 10^9/L$; $P = .006$), higher platelet counts (276 vs $249 \times 10^9/L$; $P = .02$), and lower hemoglobin concentrations (13.6 vs 14.3 g/dL; $P = .03$) than controls (Table S1 in Online Resource 1; Fig. 1). Neutrophil and lymphocyte counts did not differ, and NLR was nearly identical (1.67 vs 1.79 ; $P = .92$); both medians fell within the published healthy-adult range of 0.78 to 3.53 [31]. Median CRP was 2.1 mg/L (IQR, 0.9 - 5.0) in patients and 0.9 mg/L (IQR, 0.4 - 1.3) in controls, and median ESR was 10 mm/h (IQR, 6 - 18) and 4 mm/h (IQR, 2 - 6), respectively (both $P < .001$).

Among the composite indices, PIV was higher (median, 276 vs 208 ; $P = .02$) and HPR was lower (5.03 vs 5.56 ; $P = .007$) in patients (Table S1). After adjustment for age, sex, BMI, and pack-years of smoking, both remained associated with seborrheic dermatitis (PIV: aOR per 1-SD increase, 1.75 [95% CI, 1.10 - 2.77]; $P = .02$; $q = .05$; HPR: aOR, 0.56 [95% CI, 0.39 - 0.81]; $P = .002$; $q = .006$) (Table 2). The PIV result lay at the FDR threshold. The other 7 indices were not associated with seborrheic dermatitis; their aORs included 1, and all q values were at least $.21$ (Table 2; Fig. S2 in Online Resource 1). The aORs for CRP (28.85 ; 95% CI, 5.55 - 149.93) and ESR (13.11 ; 95% CI, 5.46 - 31.47) were large and imprecise because both markers had narrow distributions among controls (Table 2).

For distinguishing patients from controls, CRP had an AUC of 0.725 (95% CI, 0.658 - 0.791), with an optimal cutoff of 1.5 mg/L (sensitivity, 57.7% ; specificity, 81.9%), and ESR had an AUC of 0.795 (95% CI, 0.734 - 0.852), with a cutoff of 7 mm/h (sensitivity, 71.8% ; specificity, 79.2%) (Fig. 2; Table S2 in Online Resource 1). The AUCs for PIV (0.599 ; 95% CI, 0.515 - 0.673) and HPR (0.613 ; 95% CI, 0.535 - 0.687) indicated poor discrimination (Fig. S1 in Online Resource 1).

Analysis excluding patients with comorbidity

In the analysis restricted to the 88 patients without comorbidity and the 72 controls, the aORs for PIV (1.59; 95% CI, 0.98-2.58; $P = .06$; $q = .17$) and HPR (0.61; 95% CI, 0.42-0.89; $P = .01$; $q = .06$) were similar to those in the primary analysis but less precise, and both fell short of the FDR threshold (Table S6 in Online Resource 1). CRP (aOR, 24.61; 95% CI, 4.51-134.18) and ESR (aOR, 11.85; 95% CI, 4.86-28.91) remained strongly associated with seborrheic dermatitis (both $q < .001$).

Exploratory sex-stratified analyses

The cohort consisted of 95 men (64 patients and 31 controls) and 119 women (78 patients and 41 controls). Among women, HPR was lower in patients than in controls (median, 4.36 vs 5.22; $P < .001$), but it did not differ among men (6.09 vs 6.08; $P = .50$). Conversely, PIV was higher in patients than in controls among men (276 vs 184; $P = .03$) but not among women (281 vs 234; $P = .24$) (Table S7 in Online Resource 1). Hemoglobin was lower in patients among men (15.2 vs 15.9 g/dL; $P = .001$) but not among women (13.0 vs 13.2 g/dL; $P = .13$), and platelet counts were higher in patients among women (290 vs $251 \times 10^9/L$; $P = .002$) but not among men (253 vs $248 \times 10^9/L$; $P = .86$). Formal tests for interaction by sex were not significant (all $P > .10$).

Associations with clinical features

No marker was associated with any clinical feature (Fig. 3; Tables S3 and S4 in Online Resource 1). Correlations between SDASI score and each marker were weak (Spearman ρ , -0.08 for PIV to 0.11 for HPR; Fig. S3 in Online Resource 1), and none was significant after FDR correction (all $q > .80$). In multivariable linear regression, higher NLR, SII, SIRI, and PIV were associated with lower rather than higher SDASI scores before correction for multiple testing (β per 1-SD increase, -0.45 to -0.65 ; $P = .005$ to $.05$), but none remained significant after correction (all $q \geq .06$) (Table S3).

The comparison of moderate-to-severe with mild disease showed the same null pattern: the aORs included 1 for every marker, and AUCs ranged from 0.51 to 0.58. No marker was associated with extra-scalp involvement, chronic disease, treatment refractoriness, or pruritus (all $q > .50$ across 50 marker-outcome comparisons; Fig. 3; Table S4). Results were similar with a chronic disease threshold of 120 months (23 patients; Table S5 in Online Resource 1). Tests for interaction by age category, sex, and comorbidity were not significant (all $P > .10$). Among the 5 patients treated with systemic therapy, no marker differed from the patients who used topical therapy alone (all $P > .27$), although this subgroup was too small for formal inference.

Among patients, a composite model that added CRP, ESR, and PIV to clinical covariates did not improve discrimination: the AUC was 0.668 in the original sample and fell to 0.590 after bootstrap correction for optimism, with no gain over the model with clinical covariates alone (Δ AUC, 0.019; 95% CI, -0.018 to 0.047 ; $P = .22$).

Discussion

In this case-control study of 142 patients with seborrheic dermatitis and 72 healthy controls, 2 of 9 blood count-derived indices, PIV and HPR, differed between the groups, but both discriminated poorly and no longer met the FDR threshold after patients with comorbidity were excluded. The other 7 indices did not differ. CRP and ESR separated the groups more clearly, but no marker was associated with disease severity or any other clinical feature.

Median CRP was 2.1 mg/L in patients and 0.9 mg/L in controls, and median ESR was 10 and 4 mm/h, so the medians in both groups lay within commonly used reference ranges [32, 33]. Seborrheic dermatitis is diagnosed clinically, which means that an AUC here shows how strongly a marker separates the groups, not how useful a blood test would be for diagnosis. By that standard, the blood count-derived indices performed poorly [28], and the large odds ratios for CRP and ESR likewise say little about classification [34].

Metabolic comorbidity has been reported to be more common in seborrheic dermatitis [35], and comparison groups selected for good health can exaggerate case-control differences [36]. Here, controls had no systemic comorbidity, but 38.0% of patients did. When we restricted the analysis to the 88 patients without comorbidity, the point estimates for PIV and HPR changed little, which suggests that comorbidity is unlikely to explain the differences on its own, but the intervals widened and both indices fell short of the FDR threshold, so we regard these 2 findings as tentative. The CRP and ESR findings appeared more stable but also depend on the control group; for example, ESR rises as hemoglobin falls [33], and hemoglobin was lower in patients.

The direction of the blood count differences, with higher monocyte and platelet counts and lower hemoglobin, is compatible with low-grade inflammation [37, 38] and with inflammasome-driven models of seborrheic dermatitis [2, 3]. The pattern is not specific, however. Median hemoglobin stayed within reference ranges in both sexes, and iron status, menstrual blood loss, and diet, each of which can shift hemoglobin and platelet counts, were not recorded. Unmeasured iron deficiency, which can cause reactive thrombocytosis [39], may therefore have contributed, especially to the HPR difference among women, in whom platelet counts were higher in patients (Table S7 in Online Resource 1). We therefore do not interpret the differences as anemia of inflammation [40] or as evidence for a specific inflammatory pathway.

Findings in other inflammatory skin diseases are mixed. In HS, PIV and SII were higher than in controls, and PIV was the marker most strongly associated with severity [14], and a meta-analysis found associations of CRP, ESR, SII, and PIV with severity [10]. In psoriasis, SII and PIV correlated with disease severity [8, 13], but in a meta-analysis NLR and PLR were associated with the presence of psoriasis and not with its severity [41], a pattern that resembles ours.

Psoriasis and HS involve deeper tissue inflammation and, in some patients, systemic manifestations [42, 43]. Seborrheic dermatitis, in contrast, is a superficial dermatosis associated with changes in the surface

microbiome [44] and may generate less systemic inflammation, a hypothesis that our data cannot test. Performance also varies within diseases: PIV separated patients with psoriatic arthritis from those with psoriasis alone only modestly (AUC, 0.63) [45], and NLR and SII were not reproducibly associated with progression from acute to chronic urticaria in a systematic review [46].

In the only earlier study in seborrheic dermatitis that we identified, PLR, MPV, and CRP were higher in patients, and red cell distribution width correlated with SDASI [18]. As in that study, NLR and SII did not differ between patients and controls or correlate with SDASI, and CRP was higher in patients. In contrast, we found no difference in PLR (AUC, 0.50) or MPV (median, 10.1 fL in both groups), and we did not analyze red cell distribution width. Differences in control selection and patient mix may explain these discrepancies, as may differences in laboratory methods [47], and the value of MPV as an inflammatory marker in seborrheic dermatitis has itself been questioned [19], as has its clinical use more generally [48].

Before correction for multiple testing, higher NLR, SII, SIRI, and PIV were associated with lower, not higher, SDASI scores, the opposite of what an inflammatory signal would predict; none of these associations survived correction, and we regard them as likely chance findings. The AUCs for distinguishing moderate-to-severe from mild disease were 0.58 or lower, which argues against a strong association. Two features of the cohort may nonetheless have limited our ability to detect one. Most patients had mild or moderate disease (only 5 [3.5%] had severe disease), and nearly all (93.7%) had used topical antifungal therapy in the preceding month (Table 1), which is effective in seborrheic dermatitis [49] and may have narrowed the range of severity.

HPR differed between patients and controls among women but not among men, and PIV showed the reverse pattern. These sex-stratified findings were exploratory and should not be read as evidence of sex-specific biology: the formal tests for interaction were not significant, the strata were small (31 male and 41 female controls), and a difference between a significant and a nonsignificant subgroup does not establish that the effects differ [50, 51]. The components of HPR also differed by sex (higher platelet counts in female patients and lower hemoglobin in male patients), and because hemoglobin and platelet counts differ physiologically by sex [52, 53], ratios such as HPR would be better evaluated with sex-specific reference values.

These data offer no support for using blood count-derived indices to assess disease severity or to guide management. Future studies would benefit from a prospective design with prespecified analyses, controls who include participants with comparable comorbidity, measurement of iron status, and sex-specific reference values. Direct assays of lesional skin, such as RNA sequencing of skin samples [54] or tape strips [55] and profiling of barrier function and ceramides [56], may be more informative. Longitudinal designs could also show how blood markers change with disease activity or treatment, as NLR and PLR did during biologic therapy in HS [57].

Strengths and limitations

This study has several strengths. It evaluated 9 blood count-derived indices, CRP, and ESR in the same participants, including 6 indices not previously studied in seborrheic dermatitis, controlled the FDR across markers, and tested the main findings after exclusion of patients with comorbidity.

The study also has limitations. Controls could not have comorbidity, but patients could, which would be expected to exaggerate case-control differences, and we could not adjust for comorbidity in the case-control models; the analysis restricted to patients without comorbidity was more comparable but less precise. Blood tests were requested in routine care for only some participants, so inclusion depended on the availability of results, which can introduce selection bias [58]. Because of the retrospective design, we also could not verify the reason for each request, the timing of sampling relative to the clinic visit, or the time of day of sampling, which affects leukocyte counts and hemoglobin [59].

SDASI was scored at clinic visits and was not documented as blinded to the laboratory results. The investigators set the severity categories, chronicity threshold, and definition of treatment refractoriness, and the analyses were not registered in advance, so the within-disease analyses are exploratory despite FDR control. The single-center setting in Türkiye may limit generalizability, treatment adherence could not be verified, and only 5 patients were treated with systemic therapy. Because we did not calculate a sample size, power may have been insufficient to detect small within-disease associations, particularly in the sex strata and among the 5 patients with severe disease. The absence of significant associations should therefore be read as evidence against large effects, not as proof of no effect.

Conclusion

Blood count-derived indices did not appear useful for assessing the severity of seborrheic dermatitis in this retrospective case-control study. PIV and HPR differed only slightly between patients and controls, and CRP and ESR separated the groups more clearly, but these differences may depend on control selection and need confirmation in prospective studies with comorbidity-matched controls. Seborrheic dermatitis appears to carry, at most, a modest systemic inflammatory signature.

Declarations

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Conflicts of Interest: None declared.

Ethical Approval: Reviewed and approved by the Istanbul University-Cerrahpaşa Medical Research Ethics Committee; approval letter No. E-24687260-604.01-1662961.

Ethics statement: Informed consent was not obtained because of the retrospective design of the study; the manuscript contains no individual patient data or images.

Author contribution: **Tuğba Kevser Üstünbaş Uzunçakmak:** Conceptualization, Data curation, Investigation, Methodology, Supervision, Writing (review and editing); **Yusuf Demir:** Conceptualization,

Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing (original draft), Writing (review and editing); **Ömer Faruk Tolun**: Conceptualization, Data curation, Investigation, Methodology, Writing (review and editing); **Muazzez Çiğdem Oba**: Conceptualization, Methodology, Writing (review and editing); **Zekayi Kutlubay**: Conceptualization, Methodology, Supervision, Writing (review and editing); **Burhan Engin**: Conceptualization, Methodology, Supervision, Writing (review and editing).

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Competing interests

The authors have no relevant financial or non-financial interests to disclose.

Author contributions

All authors contributed to the study conception and design. Material preparation and data collection were performed by Tuğba Kevser Üstünbaş Uzunçakmak, Yusuf Demir, and Ömer Faruk Tolun. Data analysis was performed by Yusuf Demir. The first draft of the manuscript was written by Yusuf Demir, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics approval

This study was approved by the Istanbul University-Cerrahpaşa Medical Research Ethics Committee at its meeting of June 16, 2026 (letter No. E-24687260-604.01-1662961, dated June 17, 2026) and was conducted in accordance with the Declaration of Helsinki.

Consent to participate

Informed consent was not obtained because of the retrospective design of the study, which used existing medical records.

Consent to publish

Not applicable. The manuscript contains no individual patient data or images.

Use of artificial intelligence

The authors used Claude (Anthropic) for assistance with manuscript drafting, language editing. All content, results, and conclusions were critically reviewed and verified by the authors, who take full responsibility for the work.

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Tables

Table 1 Characteristics of patients with seborrheic dermatitis and healthy controls

Characteristic	Seborrheic dermatitis (n = 142)	Healthy controls (n = 72)	P value ^a
Demographics			
Age, median (IQR), y	28.0 (21.0-43.8)	29.0 (26.0-35.5)	.21
Male sex, No. (%)	64 (45.1)	31 (43.1)	.89
BMI, median (IQR)	24.8 (22.5-28.1)	24.0 (21.0-27.2)	.13
Current smoker, No. (%)	33 (23.2)	16 (22.2)	>.99
Smoking, median (IQR), pack-years	0.0 (0.0-0.0)	0.0 (0.0-0.0)	.81
Any comorbidity, No. (%) ^b	54 (38.0)	0	<.001
Disease characteristics (patients with seborrheic dermatitis only)			
SDASI score, median (IQR)	10 (8-15)	NA	NA
Mild (SDASI <12), No. (%)	77 (54.2)	NA	NA
Moderate (SDASI 12-24), No. (%)	60 (42.3)	NA	NA
Severe (SDASI >24), No. (%)	5 (3.5)	NA	NA
Disease duration, median (IQR), mo	39 (20-70)	NA	NA
Scalp involvement, No. (%)	140 (98.6)	NA	NA
Facial involvement, No. (%)	62 (43.7)	NA	NA
Truncal involvement, No. (%)	9 (6.3)	NA	NA
Pruritus, No. (%)	109 (76.8)	NA	NA
Treatment in the preceding month (patients with seborrheic dermatitis only)			
Topical antifungal, No. (%)	133 (93.7)	NA	NA
Topical corticosteroid, No. (%)	37 (26.1)	NA	NA
Systemic therapy, No. (%)	5 (3.5)	NA	NA

BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); IQR, interquartile range; NA, not applicable; SDASI, Seborrheic Dermatitis Area and Severity Index

^aContinuous variables were compared with the Mann-Whitney U test and categorical variables with the χ^2 or Fisher exact test

^bDiabetes mellitus, thyroid disease, cardiovascular disease, or hypertension. Controls were required to have no comorbidity (exclusion criterion by design)

Table 2 Association of blood count-derived indices, C-reactive protein, and erythrocyte sedimentation rate with seborrheic dermatitis

Marker (per 1-SD increase)	Unadjusted			Adjusted ^a		
	OR (95% CI)	P value	q value ^b	OR (95% CI)	P value	q value ^b
NLR	0.99 (0.74-1.31)	.92	.92	1.00 (0.76-1.33)	.99	.99
PLR	0.97 (0.73-1.28)	.81	.89	1.00 (0.75-1.34)	.99	.99
MLR	1.22 (0.89-1.68)	.22	.40	1.20 (0.87-1.68)	.27	.46
SII	1.17 (0.85-1.62)	.32	.51	1.19 (0.86-1.64)	.29	.46
SIRI	1.52 (0.96-2.41)	.07	.16	1.49 (0.93-2.36)	.09	.21
PIV	1.79 (1.13-2.82)	.01	.04	1.75 (1.10-2.77)	.02	.05
dNLR	1.13 (0.81-1.57)	.49	.67	1.15 (0.82-1.61)	.42	.57
Neutrophil-to-platelet ratio	1.08 (0.80-1.46)	.61	.74	1.09 (0.80-1.47)	.60	.73
HPR	0.69 (0.51-0.92)	.01	.04	0.56 (0.39-0.81)	.002	.006
CRP	26.08 (5.41-125.64)	<.001	<.001	28.85 (5.55-149.93)	<.001	<.001
ESR	9.02 (4.11-19.78)	<.001	<.001	13.11 (5.46-31.47)	<.001	<.001

CRP, C-reactive protein; dNLR, derived neutrophil-to-lymphocyte ratio; ESR, erythrocyte sedimentation rate; HPR, hemoglobin-to-platelet ratio; MLR, monocyte-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; PIV, pan-immune-inflammation value; PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; SIRI, systemic inflammation response index

^aAdjusted for age, sex, body mass index, and pack-years of smoking. Each marker was modeled separately

^bP value adjusted for the false discovery rate (Benjamini-Hochberg procedure) across the markers shown. The ORs for CRP and ESR are imprecise because both markers have narrow distributions among controls

Figures

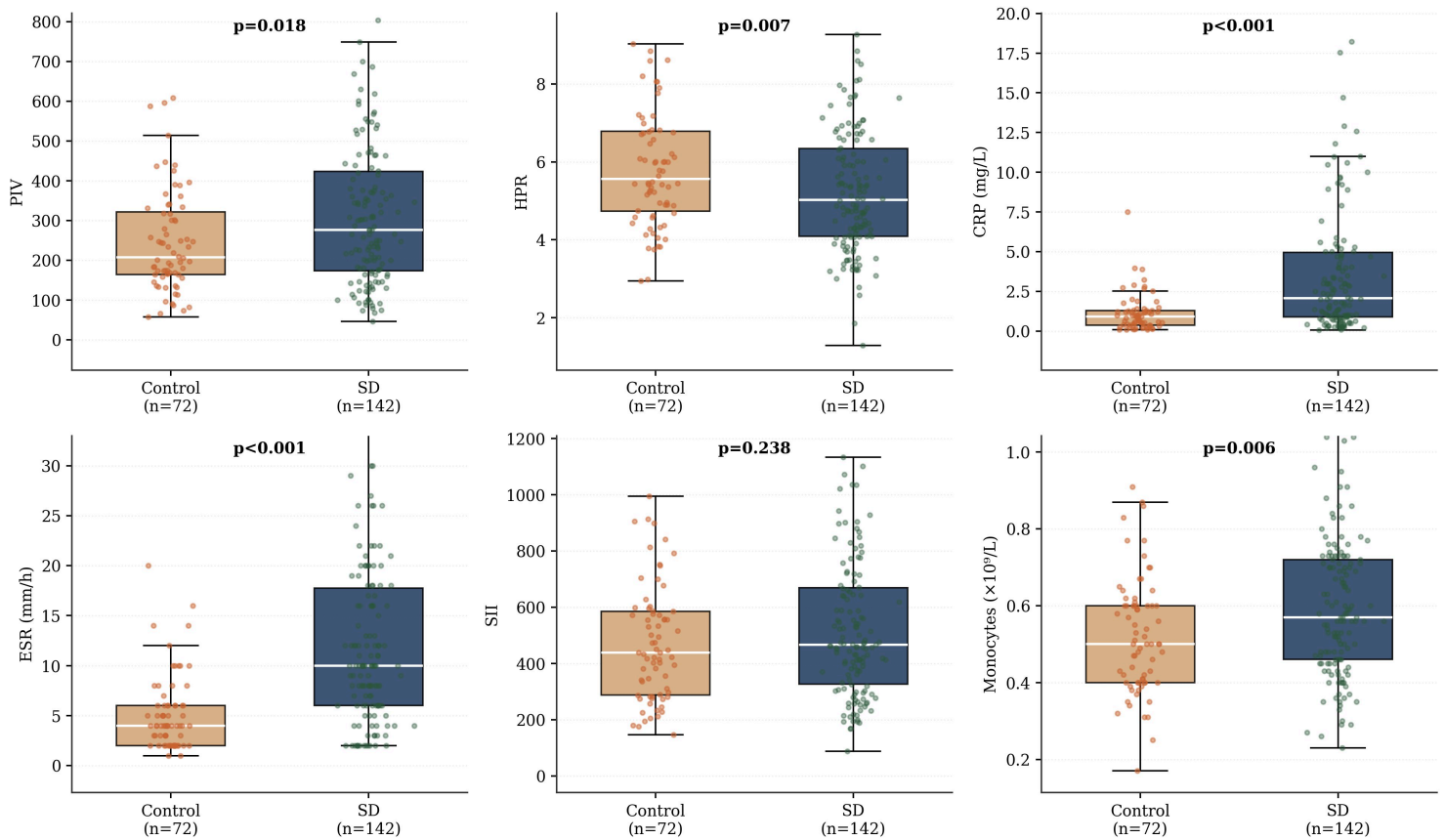


Figure 1

Distribution of selected blood count-derived indices and acute-phase reactants in patients with seborrheic dermatitis and healthy controls. Box plots of pan-immune-inflammation value (PIV), hemoglobin-to-platelet ratio (HPR), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), systemic immune-inflammation index (SII), and monocyte count in patients with seborrheic dermatitis (labeled SD in the figure; n = 142) and healthy controls (n = 72). Boxes show the median and interquartile range (IQR), whiskers extend to 1.5 × IQR, and dots are individual participants. P values are unadjusted (Mann-Whitney U test)

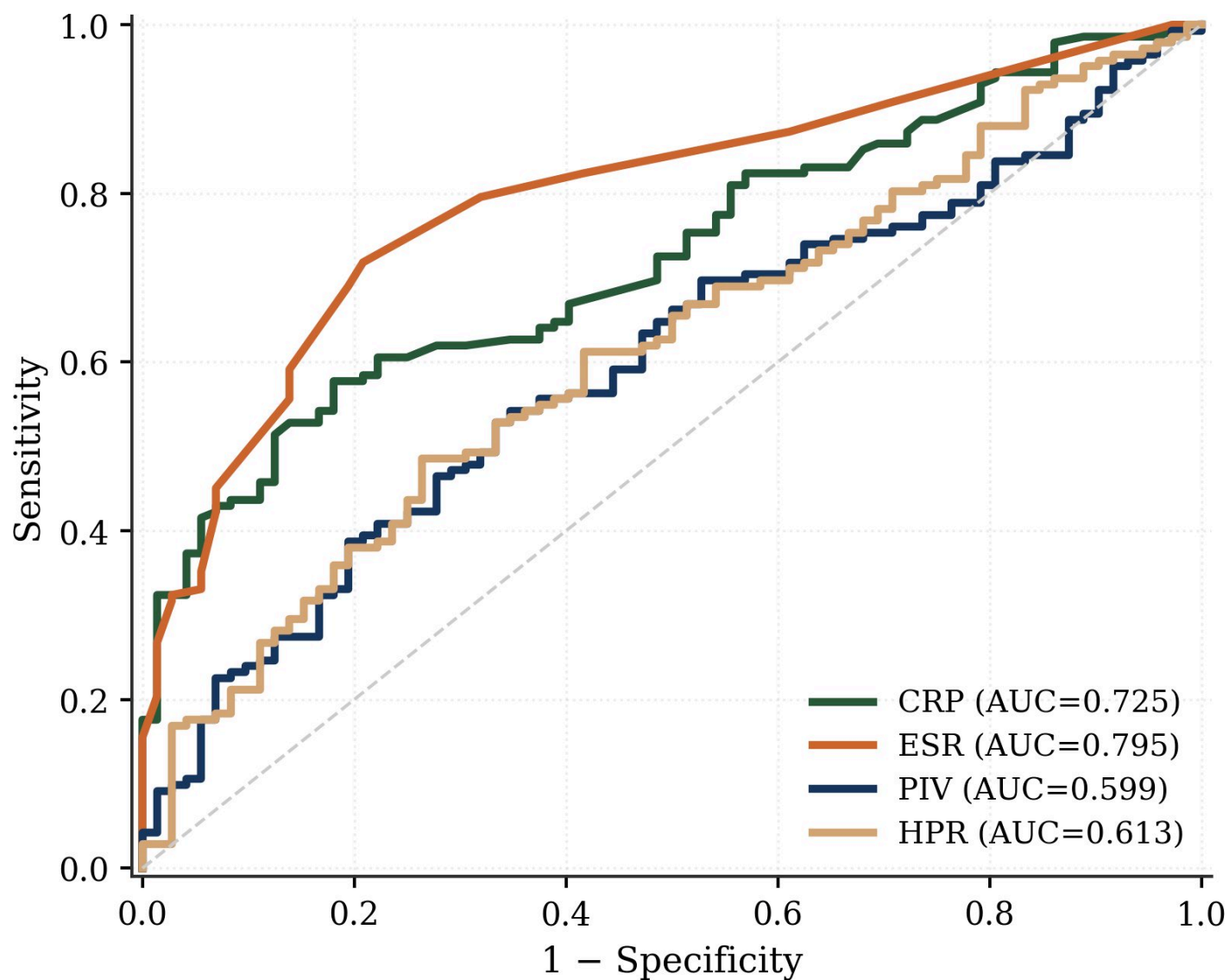


Figure 2

Receiver operating characteristic curves for discriminating patients with seborrheic dermatitis from healthy controls. Areas under the curve: erythrocyte sedimentation rate (ESR), 0.795; C-reactive protein (CRP), 0.725; hemoglobin-to-platelet ratio (HPR), 0.613; pan-immune-inflammation value (PIV), 0.599. The dashed diagonal line indicates chance performance

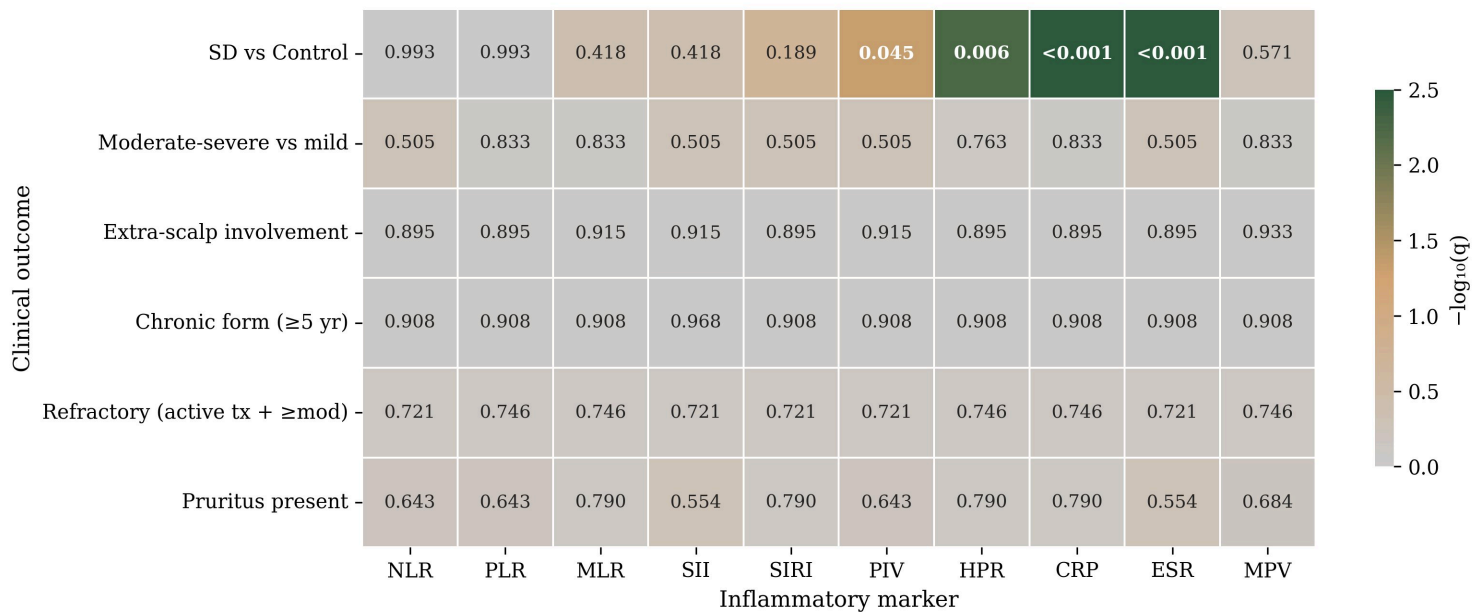


Figure 3

Adjusted q values for the association of each marker with each outcome. Heatmap of q values (Benjamini-Hochberg procedure for the false discovery rate) from separate adjusted models for each marker (columns: NLR, PLR, MLR, SII, SIRI, PIV, HPR, CRP, ESR, and mean platelet volume [MPV]) and each outcome (rows; SD denotes seborrheic dermatitis). Darker cells indicate smaller q values, and white text denotes $q < .05$. Only the case-control comparison (top row) had $q < .05$ (PIV, HPR, CRP, and ESR); no marker was associated with any within-disease outcome shown (all $q > .50$). Chronic form indicates disease duration of at least 5 years; refractory, moderate or higher severity despite active topical treatment. The q values in the figure were adjusted across the 10 markers shown and may differ slightly from those in Table 2. Abbreviations other than MPV are defined in Table 2

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