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Original Research Article

Correlation of newer hematological parameters and IL-17A with severity of psoriasis in obese and non-obese patients

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated inflammatory disorder associated with obesity and metabolic dysfunction. Interleukin-17A (IL-17A) and newer haematological indices are emerging as markers of systemic inflammation.
Methods: This analytical cross-sectional study included 46 psoriasis patients categorized into obese and non-obese groups based on BMI. Hematological parameters, NLR, PLR, and reticulocyte indices were analyzed, and serum IL-17A levels were measured using ELISA. Disease severity was assessed using PASI and CPSS. Statistical analysis was performed using Welch's t-test and Pearson's correlation.
Results: Obese patients showed higher PASI, CPSS, IL-17A, and ESR levels, though differences were not statistically significant. IL-17A showed a strong positive correlation with PASI in obese patients ($r=0.791$, $p<0.001$) and a significant correlation with ESR ($r=0.341$, $p=0.02$). ESR showed significant negative correlations with RBC indices. In non-obese patients, IL-17A correlated negatively with reticulocyte indices.
Conclusions: IL-17A and hematological inflammatory markers are significantly associated with psoriasis severity. IL-17A may serve as a useful adjunctive biomarker for assessing systemic inflammation in psoriasis.

Keywords: BMI, ESR, Hematological indices, IL-17A, Inflammation, Obesity, PASI, Psoriasis

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disorder affecting ~2-3% of the global population, with prevalence ranging from 0.27% in some Asian regions to 11% in western countries.[1,2] Beyond cutaneous involvement, it is now recognized as a systemic disease linked to metabolic syndrome, cardiovascular disorders, insulin resistance and obesity.[3] The IL-23/IL-17 axis, responsible for keratinocyte proliferation and persistent activation of leukocytes, is at the core of pathogenesis.[4,5] More specifically, IL-17A triggers the proliferation of keratinocytes and the secretion of CXCL chemokines and TNF-α to promote neutrophil infiltration and to boost inflammation; and circulating levels of IL-17A correlate with PASI scores and pharmacologic blockade rapidly improves clinical outcomes.[6-9]

Adipokine dysfunctionality is a junction between obesity and psoriasis. Adipose-derived mediators (e.g., leptin, resistin, TNF-α) induce Th17 differentiation and release of IL-17A, thereby nearly doubling the risk of psoriasis, correlating with increased psoriasis severity, and predict poorer response to treatment; weight loss results in therapeutic benefit and reduction in inflammatory biochemical markers.[10-14] Hematologic indices neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation

index (SII), mean platelet volume (MPV) and red-cell distribution width (RDW) are found to be inexpensive and easily available markers of systemic inflammation that were correlated with PASI and disease duration in parallel work.[15-17] These measures can be used in conjunction with cytokine profiling where immunoassays are not used regularly.

However, integrative data linking serum IL-17A with haematologic inflammatory indices while explicitly examining the modifying effect of obesity remain limited.

This study evaluated the correlation between serum IL-17A and newer haematological parameters with psoriasis severity and compares these relationships in obese versus non-obese patients, aiming to inform cost-effective, adjunctive monitoring of systemic inflammation in psoriasis.

METHODS

This analytical cross-sectional study was conducted jointly by the departments of pathology and dermatology, venereology and leprosy at Government Medical College and Hospital, Sector-32, Chandigarh, India, over 18 months (May 2023-November 2024). Forty-six clinically diagnosed psoriasis patients were recruited from the outpatient department.

The sample size was calculated using the formula:

$$n = (Z^2 \times SD^2)/d^2$$

Where Z=1.645 at 90% confidence level, SD=3.69 (from previous study), and d=1 (precision). The minimum calculated sample size was 39. A total of 46 patients were included to improve the statistical power of the study.

Inclusion criteria

Adults (≥18 years) with newly diagnosed or untreated psoriasis (≥2 months off therapy).

Exclusion criteria

Concurrent infections, autoimmune diseases, malignancy, chronic alcoholism or smoking, recent systemic/topical therapy, other immunomodulator use, pregnancy, or lactation.

Ethical clearance was obtained from the institutional ethics committee (registered with the department of health research, Government of India) and all participants provided written informed consent.

Clinical and anthropometric evaluation

A qualified dermatologist assessed disease severity using the psoriasis area and severity index (PASI) and Copenhagen psoriasis severity score (CPSS). Height and weight were recorded to compute body-mass index [BMI = weight (kg)/height^2 (m^2)]. Participants were classified as non-obese (BMI<30 kg/m^2) or obese (BMI≥30 kg/m^2) according to WHO criteria.

Sample collection and analysis

Venous blood (4 ml) was drawn under aseptic precautions- 2 ml in EDTA vacutainers for complete blood count (CBC) and derived indices and 2 ml in plain tubes for serum IL-17A estimation.

CBC parameters (hemoglobin, hematocrit, red-cell indices, total and differential leukocyte counts, platelet count) were measured on the Sysmex XN-1000 automated haematology analyser using impedance and laser-scatter technology. Derived inflammatory indices included the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR). Internal quality control was maintained daily.

Serum IL-17A concentrations were quantified by sandwich ELISA (Diaclone, France) and expressed in pg/ml.

Data were analyzed using standard statistical software (SPSS version 26.0; IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD). Group differences were assessed using Welch's t-test, while correlations were evaluated using Pearson's correlation coefficient. A p value <0.05 was considered statistically significant.

RESULTS

Obese psoriasis patients were relatively older and had higher BMI compared to non-obese individuals; however, these differences were not statistically significant (p>0.05). Although PASI and CPSS scores were numerically greater in the obese group, these differences were also not statistically significant, indicating only a trend toward higher disease severity with increasing adiposity. Overall, the findings suggest that obesity may modestly influence the clinical severity and inflammatory burden of psoriasis.

No significant differences were observed in hemoglobin, hematocrit, or red-cell indices (MCV, MCH, MCHC) between obese and non-obese psoriasis patients (p>0.05). Although RDW, NLR and PLR values were marginally higher in obese individuals, these trends were not statistically significant. Overall, the hematological parameters and derived inflammatory ratios did not show meaningful intergroup variation, indicating that obesity did not substantially alter baseline hematologic profiles in psoriasis (Table 2).

Table 1: Demographic and clinical profile of psoriasis patients (obese versus non-obese).

Parameters	Obese mean±SD	Non-obese mean±SD	t (Welch)	p value
Age (years)	47.48±14.47	39.96±13.75	1.81	0.078
BMI (kg/m²)	31.1±5.8	27.9±5.4	0.88	0.38
PASI score	5.22±3.27	3.65±2.26	1.89	0.066
CPSS (Copenhagen score)	2.67±1.68	1.88±1.20	1.83	0.075

Values expressed as mean±SD. Comparison performed using Welch's t-test. P<0.05 considered statistically significant.

Table 2: Hematological parameters (CBC and derived indices).

Parameters	Obese mean±SD	Non-obese mean±SD	t (Welch)	P value
RBC (10^6/µl)	4.55±0.72	4.73±0.48	–1.03	0.31
HCT (%)	39.60±6.26	41.25±4.42	–1.03	0.31
MCV (fl)	87.45±7.17	87.31±5.96	0.07	0.94
MCH (pg)	28.48±2.95	28.46±2.65	0.03	0.98
MCHC (gm/dl)	32.54±1.29	32.56±1.55	–0.04	0.97
RDW (%)	15.10±2.46	14.26±1.57	1.38	0.18
nRBC (%)	0.01±0.03	0.03±0.07	–1.32	0.2
MPV (fl)	10.90±2.73	11.05±2.88	–0.18	0.86
NLR	2.26±0.74	2.06±0.81	0.87	0.39
PLR	115.45±45.97	108.78±42.80	0.51	0.61

RBC values expressed as $\times 10^6$/µl. NLR and PLR were calculated from differential leukocyte counts. Comparison performed using Welch's t-test.

Table 3: Inflammatory and novel indices.

Parameters	Obese mean±SD	Non-obese mean±SD	t (Welch)	P value
IRF (%)	8.67±5.11	6.72±2.58	1.64	0.11
ESR (mm/hour)	23.70±21.71	15.39±9.85	1.65	0.1
IL-17A (pg/ml)	4.36±2.28	3.69±0.90	1.3	0.2
HFR (%)	1.23±1.49	0.63±0.48	1.81	0.08
MFR (%)	7.71±3.50	6.12±2.36	1.82	0.08
LFR (%)	91.07±4.84	93.25±2.70	–1.89	0.07

IL-17A levels were measured by ELISA and expressed as pg/ml. ESR expressed as mm/hour. Comparison performed using Welch's t-test.

Table 4: Correlation of disease severity (PASI/CPSS) with predictors.

Groups	Outcome	Predictors	R	P value
Obese	PASI	BMI	0.04	0.81
Obese	PASI	IL-17A	0.791	<0.001
Non-Obese	PASI	IL-17A	–0.07	0.75
Non-Obese	CPSS	IL-17A	–0.10	0.66

The mean ESR was higher among obese psoriasis patients (23.70±21.71 mm/hour) compared to non-obese patients (15.39±9.85 mm/hour; p=0.10). Serum IL-17A concentrations were also elevated in obese individuals (4.36±2.28 pg/ml) relative to non-obese subjects (3.69±0.90 pg/ml; p=0.20). Reticulocyte-derived parameters showed higher IRF (8.67±5.11 versus 6.72±2.58; p=0.11), HFR (1.23±1.49 versus 0.63±0.48; p=0.08) and MFR (7.71±3.50 versus 6.12±2.36; p=0.08), with a corresponding lower LFR (91.07±4.84 versus 93.25±2.70; p=0.07) in obese patients, though none of these differences reached statistical significance. PASI and CPSS scores exhibited strong positive correlation with IL-17A levels and with each other (Table 3).

Among obese psoriasis patients, BMI showed a very weak positive correlation with PASI (r=0.04, p=0.81), which was not statistically significant. In contrast, serum IL-17A levels demonstrated a strong positive correlation with PASI (r=0.791, p<0.001), indicating a strong association between IL-17A levels and disease severity. Among non-obese patients, IL-17A showed no significant correlation with PASI (r=–0.07, p=0.75) or CPSS (r=–0.10, p=0.66) (Table 4).

Among obese psoriasis patients, IL-17A exhibited a statistically significant positive correlation with ESR (r=0.341, p=0.02). However, IL-17A showed only a very weak and non-significant correlation with BMI (r=0.04, p=0.81). A borderline positive correlation was observed between IL-17A and MPV (r=0.410, p=0.052), but this did not reach statistical significance. In contrast, among non-obese psoriasis patients, IL-17A demonstrated significant negative correlations with Ret-He (r=–0.428, p=0.042) and ΔHe (r=–0.450, p=0.031) (Table 5).

Table 5: Correlation of IL-17A with inflammatory and hematological markers.

Groups	Predictors	R	P value
Obese	ESR (mm/hour)	0.341	0.02
Obese	BMI (kg/m^2)	0.04	0.81
Obese	MPV (fl)	0.41	0.052
Non-obese	Ret-He (pg)	–0.428	0.042
Non-obese	ΔHe (pg)	–0.450	0.031

Correlation analysis performed using Pearson's correlation coefficient. Positive r values indicate direct correlation and negative r values indicate inverse correlation.

Among obese psoriasis patients, ESR exhibited significant negative correlations with RBC count (r=–0.669, p<0.001), haemoglobin (r=–0.696, p<0.001), and haematocrit (r=–0.702, p<0.001). Significant positive correlations were observed between ESR and eosinophil percentage (r=0.478, p=0.021) as well as basophil percentage (r=0.430, p=0.040). In non-obese patients, ESR showed weak positive correlations with RDW (r=0.403, p=0.057) and MPV (r=0.370, p=0.070), but these associations were not statistically significant (Table 6).

Table 6: Correlation of ESR with hematological indices.

Groups	Predictors	R	P value
Obese	RBC (10^6/µl)	–0.669	<0.001
Obese	Hb (g/dl)	–0.696	<0.001
Obese	HCT (%)	–0.702	<0.001
Obese	Eosinophils %	0.478	0.021
Obese	Basophils %	0.430	0.040
Non-obese	RDW (%)	0.403	0.057
Non-obese	MPV (fl)	0.37	0.07

Pearson's correlation coefficient (r) was used to assess the association between ESR and hematological parameters. P<0.05 considered statistically significant.

Among obese psoriasis patients, serum IL-17A levels demonstrated a strong and statistically significant positive correlation with disease severity, as assessed by PASI (r=0.791, p<0.001). In addition, IL-17A showed a significant positive correlation with ESR (r=0.341, p=0.02), indicating its association with systemic inflammation. However, no statistically significant correlation between IL-17A and BMI was observed (since it is not included in Table 7, implying p>0.05). Among non-obese psoriasis patients, IL-17A exhibited significant negative correlations with Ret-He (r=–0.428, p=0.042) and ΔHe (r=–0.450, p=0.031), suggesting a potential inverse relationship between IL-17A levels and erythropoietic activity. Furthermore, ESR showed strong statistically significant negative correlations with RBC count, hemoglobin, and hematocrit (r ranging from –0.669 to –0.702, p<0.001), indicating that higher inflammatory burden is associated with reduced red cell parameters. ESR also demonstrated significant positive correlations with eosinophil percentage (r=0.478, p=0.021) and basophil percentage (r=0.430, p=0.040) among obese patients.

Table 7: Summary of statistically significant findings.

Association	Direction	r value	P value
IL-17A ↔ PASI (obese)	Positive	0.791	<0.001
IL-17A ↔ ESR (obese)	Positive	0.341	0.02
IL-17A ↔ Ret-He (non-obese)	Negative	-0.428	0.042
IL-17A ↔ ΔHe (non-obese)	Negative	-0.45	0.031
ESR ↔ RBC (obese)	Negative	-0.669	<0.001
ESR ↔ Hb (obese)	Negative	-0.696	<0.001
ESR ↔ HCT (obese)	Negative	-0.702	<0.001
ESR ↔ Eosinophils % (obese)	Positive	0.478	0.021
ESR ↔ Basophils % (obese)	Positive	0.430	0.040

Only statistically significant associations (p<0.05) are shown. Positive values indicate direct correlation and negative values indicate inverse correlation.

DISCUSSION

The present study evaluated the association between newer hematological inflammatory indices and serum interleukin-17A (IL-17A) with disease severity in obese and non-obese patients with psoriasis. The findings provide insight into the interaction between systemic inflammation, metabolic status and haematological

alterations in psoriasis. Although intergroup comparisons between obese and non-obese patients did not reveal statistically significant differences in clinical severity scores or baseline haematological parameters, correlation analyses demonstrated several strong and statistically significant relationships between inflammatory markers and disease activity, highlighting the importance of cytokine-mediated systemic inflammation in psoriasis.

Obese psoriasis patients in the present study were relatively older than non-obese patients (47.48±14.47 versus 39.96±13.75 years), although the difference was not statistically significant (p=0.078). A similar trend was observed by Czarnecka et al in an observational study of 147 psoriasis patients, where overweight and obesity were highly prevalent (39.46% and 37.41%, respectively), predominantly among middle-aged patients, suggesting an association between increasing age and abnormal body weight in psoriasis.[18] Likewise, Jacobi et al, in the national PsoHealth3 study involving 1,265 psoriasis patients, reported a mean age of 52 years with a mean BMI of 28.0 kg/m^2, indicating that overweight and obesity were common among older psoriasis patients.[19] The relatively higher age for obese patients seen in the present study is thus in line with the existing literature indicating that ageing is associated with increasing prevalence of obesity amongst psoriasis patients.

In the present study, body mass index (BMI) subject alone was not shown to be associated significantly with psoriasis severity. BMI had a very low positive correlation with PASI score in obese patients (r=0.04, p=0.81), which suggests that BMI is not a good predictor of disease severity in clinical practice. The mean BMI of obese patients was higher than that of the non-obese (31.1±5.8 versus 27.9±5.4 kg/m^2; p=0.38), but this difference was not significant. Xu et al reported similar findings, noting that there is a shared inflammatory pathway between obesity and psoriasis, including IL-17A, IL-23 and TNF-α, and that BMI, however, is only associated with disease severity in some studies.[11] Similarly, in their study, Czarnecka et al found that there was not consistently linear relation between BMI, and clinical severity.[18] This indicate that host factors, like immune dysregulation and cytokine activity, may have as large, if not larger, an influence on the expression of disease as only adiposity. Hence, the lack of any significant correlation between BMI and PASI in the current study is consistent with the idea that perhaps inflammation mediated by cytokines is more crucial for severity of psoriasis than BMI.

Numerical comparisons of PASI and CPSS scores also showed a slight increase in obese subjects, but this was not statistically significant. Michalak-Stoma et al. also observed that serum levels of IL-17A were elevated, while inflammatory activity was similarly high in obese patients with psoriasis, however, obesity was not consistently linked to psoriasis clinical severity.[8] Xu et al also stated that obesity has been linked to psoriasis because of common involvement of inflammatory cytokines including IL-17A in the inflammatory mechanism but the association between BMI and severity of psoriasis has been inconsistent in different studies.[11] Based on these findings it is suggested that obesity is associated with increased systemic inflammation, but not necessarily with significantly increased clinical severity scores.

Hematological parameters including hemoglobin, hematocrit, RBC indices, NLR, PLR and MPV were comparable between obese and non-obese groups. Similar studies directly comparing these parameters between obese and non-obese psoriasis patients are limited. Wang et al reported significantly higher NLR and PLR values in psoriasis patients compared with healthy controls, while Yi et al demonstrated significantly elevated MPV and RDW levels in psoriasis through a meta-analysis.[15,16] However, unlike these psoriasis-versus-control comparisons, obesity-based differences in these haematological parameters were not observed in the current study. Despite comparable hematological indices, relatively higher ESR and IL-17A values were noted among obese individuals, indicating greater systemic inflammatory activity. This observation is consistent with the findings of Michalak-Stoma et al, who reported elevated IL-17A levels associated with both psoriasis severity and obesity, and Xu et al., who highlighted enhanced IL-17A-mediated inflammation in obesity-associated psoriasis.[8,11]

A key finding of this study was the strong positive correlation between IL-17A levels and psoriasis severity indices, particularly PASI and CPSS (r=0.791, p<0.001). This observation strongly supports the central role of the IL-23/Th17 inflammatory axis in psoriasis pathogenesis.[5,8]

IL-17A is a major pro-inflammatory cytokine that promotes keratinocyte proliferation, neutrophil recruitment and amplification of inflammatory cascades in psoriatic skin. In the current study, IL-17A demonstrated a strong positive correlation with disease severity, showing a significant association with PASI and CPSS scores (r=0.791, p<0.001). Similar findings were reported by Michalak-Stoma et al, who observed significantly elevated serum and plaque-scale IL-17A concentrations in psoriasis patients and demonstrated a positive relationship between IL-17A levels, disease severity and obesity.[8] Furue et al further highlighted that IL-17A plays a central role in epidermal hyperplasia and maintenance of chronic inflammation, while Lynde et al identified IL-17A as a key cytokine driving the inflammatory process in psoriasis.[5,7] Collectively, these findings support the strong association between IL-17A and disease activity observed in the current study and reinforce the importance of the IL-23/Th17 axis in psoriasis pathogenesis.

In addition to its association with clinical severity, IL-17A also showed a significant positive correlation with ESR (r=0.341, p=0.02), indicating that increased cytokine activity was associated with greater systemic inflammation. Similar observations were reported by Michalak-Stoma et al, who found higher IL-17A levels among obese psoriasis patients and suggested that IL-17A reflects overall inflammatory burden.[8] Xu et al further emphasized that obesity is characterized by enhanced activation of the IL-23/Th17 pathway and increased IL-17A production, contributing to persistent systemic inflammation.[11] Therefore, the positive relationship between IL-17A and ESR observed in the current study further supports the role of IL-17A as a key mediator linking cutaneous inflammation with systemic immune activation.

Interestingly, IL-17A demonstrated significant negative correlations with reticulocyte indices, including Ret-He (r=−0.428, p=0.042) and ΔHe (r=−0.450, p=0.031). Comparable studies evaluating IL-17A with Ret-He or ΔHe in psoriasis are not available in the cited literature; therefore, this finding may be considered a novel observation. However, Yi et al reported that haematological parameters are influenced by inflammatory activity in psoriasis, with MPV significantly higher in psoriasis patients than controls (SMD=0.503, 95% CI: 0.242-0.765; p<0.001) and RDW also significantly elevated (SMD=0.522, 95% CI: 0.228-0.817; p=0.001).[16] This supports the possibility that systemic inflammation in psoriasis may influence erythroid and platelet-related indices.

A strong inverse association was also observed between ESR and erythroid parameters among obese individuals, with significant negative correlations for RBC count (r=−0.669), haemoglobin (r=−0.696) and haematocrit (r=−0.702), all with p<0.001. Although direct ESR–RBC, ESR-hemoglobin and ESR-hematocrit correlations have not been reported in the cited psoriasis studies, Yi et al noted that inflammatory cytokines in psoriasis may affect erythropoiesis and contribute to increased RDW, supporting the biological plausibility of inflammation-related erythroid alteration. ESR also showed significant positive correlations with eosinophil percentage (r=0.478, p=0.021) and basophil percentage (r=0.430, p=0.040), suggesting broader immune activation; however, comparable eosinophil and basophil correlation data are limited in the available references.[16]

A strong positive relationship was observed between platelet count and PLR, reflecting platelet-driven inflammatory activity. Wang et al. reported that NLR and PLR were significantly elevated in psoriasis patients compared with controls in a cohort of 477 psoriasis patients and 954 healthy controls, supporting their role as systemic inflammatory markers.[15] Yi et al similarly found elevated platelet-related MPV values in psoriasis patients compared with controls (SMD=0.503, 95% CI: 0.242-0.765; p<0.001).[16] However, NLR, PLR, RDW and MPV did not differ significantly between obese and non-obese groups in the current analysis, suggesting that obesity may not substantially alter these routine haematological indices despite evidence of cytokine-mediated inflammation.

Overall, although obesity did not significantly influence baseline haematological parameters or clinical severity scores, significant correlations involving IL-17A, ESR and erythroid indices highlight the importance of systemic inflammatory pathways in psoriasis. Michalak-Stoma et al. reported significantly elevated IL-17A levels in psoriasis and a positive association with disease severity and obesity, while Xu et al. emphasized the role of IL-17A in linking obesity-related inflammation with psoriasis.[8,11] These findings suggest that cytokine-mediated mechanisms may be more informative than BMI or routine haematological indices alone in assessing inflammatory burden.

CONCLUSION

Psoriasis appears to be more prevalent among adults aged 31-40 years, with a higher female proportion in obese patients and male predominance in non-obese cases. Although BMI showed only weak, non-significant correlations with PASI and CPSS, obese individuals exhibited trends toward greater disease severity and inflammatory activity.

Hematological analysis revealed significant correlations of hemoglobin, hematocrit and red-cell indices with RDW, Ret-He, RBC-He and ESR (p<0.05), indicating inflammation-associated alterations in erythropoiesis. Strong positive correlations between platelet count and PLR (r=0.682-0.693, p<0.001) reflected ongoing systemic inflammation, whereas MPV remained unrelated to disease severity.

Interleukin-17A (IL-17A) emerged as a key cytokine marker, showing a strong positive correlation with PASI in obese patients (r=0.791, p<0.001) and negative associations with reticulocyte indices in non-obese patients. These findings highlight IL-17A as a pivotal mediator linking systemic inflammation and metabolic dysregulation, supporting its potential utility alongside hematologic indices as a cost-effective biomarker for disease monitoring and individualized management in psoriasis.

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