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Assessment of the IL-33/sST2 axis in the serological profile of psoriasis patients

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Abstract

Objective: Previous studies have reported conflicting findings regarding the role of the IL-33/sST2 axis and circulating IL-33 and sST2 concentrations in psoriasis. This study aimed to evaluate serum IL-33 and sST2 concentrations in patients with psoriasis and controls.
Materials and methods: The study included 75 patients with psoriasis and 75 age- and sex-matched controls with non-inflammatory dermatological conditions, all aged 18–65 years. Demographic and clinical characteristics and body mass index (BMI) were recorded, and disease severity was assessed using the Psoriasis Area and Severity Index (PASI). Serum IL-33 and sST2 concentrations were measured using ELISA.
Results: Mean serum IL-33 concentrations were significantly lower in patients with psoriasis than in controls (153.9 ± 59.7 vs. 157.8 ± 34.4 pg/mL, p = 0.001). Median serum sST2 concentrations did not differ significantly between patients with psoriasis and controls (9.1 vs. 10.6 ng/mL, p = 0.74). IL-33 concentrations showed a positive correlation with age (r = 0.26, p = 0.007), whereas sST2 concentrations were higher in male than in female participants (11.2 vs. 7.8 ng/mL, p = 0.003).
Conclusion: Lower circulating IL-33 concentrations were observed in patients with psoriasis, whereas sST2 concentrations did not differ significantly from those in controls. These findings suggest that circulating IL-33 may not directly reflect its local activity in psoriatic tissue. Tissue-specific regulation or limited systemic release may contribute to this discrepancy, although this hypothesis requires further investigation.

Keywords: IL-33, psoriasis, serum concentrations, sST2

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Introduction

Interleukin-33 (IL-33) is increasingly recognized as a key molecular link between tissue injury and immune activation. Unlike classical secreted cytokines, IL-33 is mainly retained within the nucleus of structural cells, such as endothelial and epithelial cells, and is released into the extracellular environment primarily in response to cellular stress or damage (1–3). Through this mode of action, IL-33 contributes to the regulation of tissue homeostasis, repair processes, and immune responses.

IL-33 signaling is initiated through binding to its receptor, suppression of tumorigenicity 2 (ST2), which is present in both membrane-bound and soluble forms. Activation of the transmembrane ST2 isoform triggers intracellular signaling cascades involved in immune modulation, whereas the soluble isoform (sST2) limits IL-33 activity by functioning as a decoy receptor and reducing ligand availability (4).

Psoriasis represents a chronic inflammatory disorder driven by immune dysregulation and characterized by excessive keratinocyte proliferation and sustained systemic inflammation. Dysregulated innate and adaptive immune responses, particularly those involving dendritic cells and T helper lymphocyte subsets, promote cytokine release, angiogenesis, and epidermal hyperplasia, ultimately leading to the formation of psoriatic plaques (5).

Although the IL-33/ST2 axis has been associated with various inflammatory and immune-mediated diseases, its contribution to psoriasis has not been fully elucidated. Studies examining circulating IL-33 and sST2 concentrations in psoriasis have yielded inconsistent results (6,7). In this context, the present study sought to further delineate the role of the IL-33/ST2 axis by evaluating serum IL-33 and sST2 concentrations in a relatively large cohort of patients with psoriasis and controls with non-inflammatory dermatological conditions.

Materials and methods

Patients with psoriasis and control participants were prospectively recruited from the institutional dermatology outpatient clinic between March 1, 2021, and June 1, 2022. Approval for the study protocol was granted by the institutional ethics committee before enrollment commenced. All procedures complied with accepted ethical principles for clinical research, and written informed consent was obtained from all participants prior to participation.

Study population

A total of 75 patients with psoriasis, aged between 18 and 65 years, were prospectively enrolled from the institutional dermatology outpatient clinic. Eligible patients had not received any systemic antipsoriatic treatment or phototherapy within the preceding three months. The control group comprised 75 age-matched individuals who presented with non-inflammatory dermatological conditions, including melanocytic nevi, fibromas, and milium cysts. Individuals were excluded if they declined participation or had conditions that could potentially affect systemic

inflammatory status, such as current systemic treatment for psoriasis, known malignancy, acute or chronic infections, renal or hepatic insufficiency, chronic systemic inflammatory diseases, or the presence of any inflammatory skin disorder.

Data collection and measurements

Demographic and clinical characteristics were documented for each participant, including age, sex, smoking and alcohol use, family history, disease duration, presence of psoriatic arthritis, nail involvement, and accompanying systemic conditions. Body mass index was derived from measured height and weight. All participants underwent dermatological evaluation, and disease severity among patients with psoriasis was assessed using the Psoriasis Area Severity Index (PASI). After written informed consent was obtained, venous blood samples (1–2 mL) were collected into gel-containing biochemistry tubes. Samples were processed by centrifugation at 3000 rpm for 15 minutes, and serum aliquots were prepared for analysis. Serum IL-33 and sST2 concentrations were quantified using commercially available human sandwich ELISA kits (Elabscience, USA) according to the manufacturer's instructions. IL-33 was measured using the Human IL-33 ELISA Kit (catalog no. E-EL-H2402; detection range, 15.63–1000 pg/mL; sensitivity, 9.38 pg/mL), and sST2 was measured using the Human sST2 ELISA Kit (catalog no. E-EL-H6082; detection range, 0.31–20 ng/mL; sensitivity, 0.19 ng/mL). According to the manufacturer, both intra-assay and inter-assay coefficients of variation were <10%. All analyses were conducted at the central laboratory of the study institution.

Statistical analysis

All statistical procedures were carried out using SPSS software for Windows (IBM Corp., Armonk, NY, USA; version 21.0). Frequencies and percentages were used to describe categorical variables, which were compared between groups using the Chi-square test or Fisher's exact test when appropriate. Assessment of data distribution for continuous variables was based on histogram evaluation and the Kolmogorov–Smirnov test. Parametric data are reported as mean ± standard deviation and were analyzed using the Student's t-test. Non-parametric variables are presented as median with interquartile range and were compared using the Mann–Whitney U test. Depending on data distribution, Pearson or Spearman correlation coefficients were applied to evaluate associations between numerical variables. Statistical significance was defined as a two-tailed p value below 0.05.

Results

Seventy-five patients with psoriasis and 75 control participants were included in the study. The patient and control groups were comparable in terms of age and sex. The mean age was 43.6 ± 13.7 years in the psoriasis group and 43.7 ± 13.9 years in the control group ($p = 0.98$). The sex distribution was identical in both groups, with 49 male (65.3%) and 26 female (34.7%) participants ($p = 0.99$). Demographic and clinical characteristics of the psoriasis group are presented in Table 1. Most patients had moderate psoriasis severity, and the median PASI score was 8.5 (IQR, 5.4–16.1).

The median disease duration was 10 years (IQR, 3–17 years). Psoriatic arthritis was present in 12 patients (16.4%), and nail involvement was present in 26 patients (35.6%). Twenty-two patients (30.1%) had comorbidities.

Characteristic	Value
Age, mean (SD)	43.6 (13.7)
Sex, n (%)	
Female	26 (34.7%)
Male	49 (65.3%)
PASI, median (IQR)	8.5 (5.4, 16.1)
Disease duration, years, median (IQR)	10 (3, 17)
Age at onset, median (IQR)	30 (23, 42)
Family history, n (%)	
Yes	26 (35.6%)
No	47 (64.4%)
Psoriatic arthritis, n (%)	
Yes	12 (16.4%)
No	61 (83.6%)
Nail involvement, n (%)	
Yes	26 (35.6%)
No	47 (64.4%)
Previous treatments, n (%)	
Topical agents	49 (67.1%)
Systemic agents	20 (27.4%)
Phototherapy	9 (12.3%)
Biologic agents	4 (5.5%)
Smoking status, n (%)	
Active smoker	35 (47.9%)
Never smoked	38 (52.1%)
Smoking amount, pack-years, median (IQR)	10 (5-21.5)
Alcohol use, n (%)	
Never	59 (80.8%)
Social drinker	9 (12.3%)
Regular drinker	5 (6.8%)
Comorbidities, n (%)	
Cardiovascular disease	15 (20.5%)
Diabetes mellitus	7 (9.6%)

SD: Standard deviation, IQR: Interquartile rate, PASI: Psoriasis area and severity index

Serum IL-33 and sST2 concentrations

Patients with psoriasis exhibited lower circulating IL-33 concentrations compared with controls. Mean serum IL-33 concentrations were 153.9 ± 59.7 pg/mL in the psoriasis group and 157.8 ± 34.4 pg/mL in the control group, with a statistically significant difference between groups ($p = 0.001$; Figure 1).

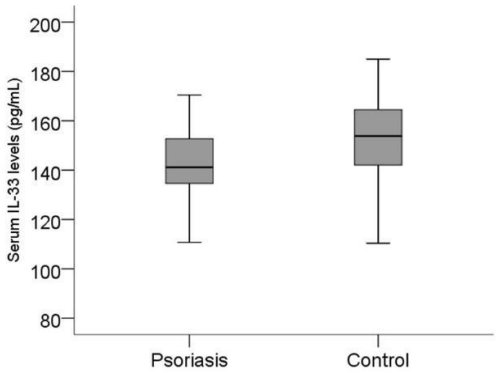

Figure 1: Serum IL-33 concentrations (p = 0.001)

Median serum sST2 concentrations were 9.1 ng/mL (interquartile range [IQR], 4.5–13.7) in patients with psoriasis and 10.6 ng/mL (IQR, 7.1–13.1) in the control group. Comparative analysis did not demonstrate a statistically significant difference between the two groups (p = 0.74; Figure 2).

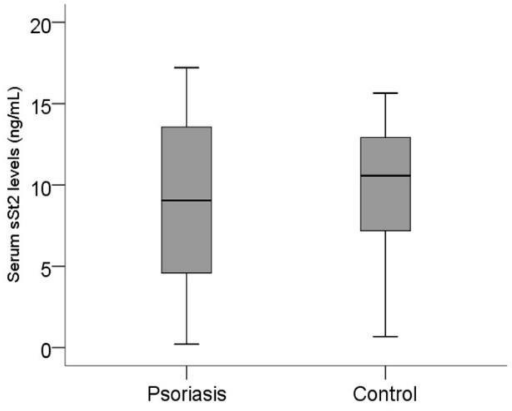

Figure 2: Serum sST2 concentrations (p = 0.74)

Serum IL-33 concentrations demonstrated a weak but statistically significant positive association with age (r = 0.26, p = 0.007), while no sex-related differences were observed (p = 0.19). In contrast, serum sST2 concentrations were higher in male participants than in females (11.2 vs. 7.8 ng/mL, p = 0.003), whereas sST2 concentrations did not correlate with age (r = 0.11, p = 0.20).

Neither serum IL-33 nor sST2 concentrations showed meaningful associations with body mass index, PASI score, disease duration, or age at disease onset (all p > 0.05). In addition, circulating IL-33 and sST2 concentrations remained comparable regardless of family history of psoriasis, smoking status, alcohol consumption, obesity, presence of psoriatic arthritis, nail involvement, or accompanying comorbid conditions (p > 0.05).

Discussion

Reduced circulating IL-33 concentrations were observed in patients with psoriasis relative to controls, while serum sST2 concentrations remained comparable between groups. An age-related increase in IL-33 concentrations was identified, whereas higher sST2 concentrations were confined to male participants. Beyond these associations, circulating IL-33 and sST2 concentrations showed no consistent relationships with additional demographic or clinical parameters.

IL-33 is expressed by keratinocytes and various immune cells within the skin and is increasingly recognized as a cytokine with context-dependent functions. Rather than acting exclusively as a classical proinflammatory mediator, IL-33 may exert immunoregulatory effects depending on the cellular environment and the mode of release. Extracellular IL-33 is typically liberated following cellular stress or necrosis and functions as a damage-associated molecular pattern capable of amplifying Th2-skewed immune responses (8–10). This dual biological behavior may partly explain the heterogeneous findings reported across different inflammatory conditions.

Several studies investigating IL-33 in psoriatic disease have suggested that systemic IL-33 concentrations may not accurately reflect local inflammatory activity. In patients with psoriatic arthritis, IL-33 has been reported to be absent or minimally detectable in serum and synovial fluid, despite demonstrable expression within endothelial cells of affected joints (11). Similarly, in rheumatoid arthritis, increased IL-33 expression has been observed at both tissue and systemic levels; however, local production rather than joint-derived release has been proposed as the primary source of circulating IL-33 (11). These observations support the concept that tissue-specific regulation plays a dominant role in shaping IL-33 dynamics.

Evidence from lesional skin further reinforces this hypothesis. Wierzbicka et al. demonstrated increased IL-33 mRNA expression in psoriatic plaques compared with non-lesional and healthy skin, accompanied by reduced ST2 mRNA expression within lesional tissue (12). The downregulation of ST2 may attenuate local regulatory feedback mechanisms and contribute to enhanced IL-33 signaling within the psoriatic microenvironment. One possible explanation for the reduced circulating IL-33 concentrations observed in our cohort is that IL-33 activity may remain predominantly localized within lesional tissue, although this hypothesis cannot be established from serum measurements alone.

Taken together, our findings raise the possibility that the IL-33/sST2 axis in psoriasis may be regulated differently at the tissue and systemic levels. The dissociation between local expression and serum concentrations underscores the complexity of IL-33 biology in psoriasis and suggests that circulating measurements alone may not fully capture its pathogenic relevance.

ST2 plays a key modulatory role in IL-33 signaling, and reduced ST2 expression within psoriatic lesions may contribute to an attenuated local regulatory response despite increased tissue IL-33 activity. Experimental data suggest that IL-33 released from keratinocytes can act in an autocrine and paracrine manner, stimulating surrounding keratinocytes and immune cells to produce psoriasis-associated proinflammatory cytokines, thereby sustaining a locally inflamed microenvironment (13). Consistent with this concept, increased IL-33 expression has been

demonstrated in psoriatic lesions, where it may promote inflammatory signaling through activation of mast cells, dendritic cells, and macrophages (6,14).

Experimental studies have suggested an association between IL-33 expression and disease severity in murine models of psoriasis; however, comparable relationships have not been consistently confirmed in human investigations. Moreover, available evidence indicates that IL-33 is not essential for the initiation of psoriatic disease, supporting a modulatory rather than a primary pathogenic role in cutaneous inflammation (12,15,16).

Clinical studies assessing circulating IL-33 concentrations in psoriasis have yielded variable results. Elevated serum IL-33 concentrations have been reported in some cohorts, including an observed association with TNF-α levels and a decline following anti-TNF-α therapy, although these findings were derived from relatively small patient populations (15). Other reports have described increased circulating IL-33 and ST2 concentrations in psoriasis; however, methodological limitations, particularly regarding sST2 assessment, limit the interpretability of these observations (17).

In contrast to these reports, our study demonstrated reduced serum IL-33 concentrations in patients with psoriasis, with a gradual increase observed with advancing age. Although circulating IL-33 concentrations were positively correlated with age, the nearly identical age distributions of the psoriasis and control groups make it unlikely that age alone accounts for the observed between-group difference. Nevertheless, the potential influence of age on circulating IL-33 concentrations should be considered when interpreting these findings. Experimental and clinical data have also shown that IL-33 may be sequestered in adipose tissue and that circulating levels are reduced in obesity, suggesting that metabolic factors may influence systemic IL-33 availability (2).

Beyond its inflammatory functions, IL-33 has been implicated in protective mechanisms, including the promotion of Th2-skewed immune responses in Th1-dominant cardiovascular conditions and the regulation of lipid metabolism, with potential protective effects against metabolic syndrome (2). In the present cohort, serum IL-33 concentrations did not differ according to obesity status or cardiovascular comorbidities. The observed age-related increase in circulating IL-33 may therefore reflect a context-dependent, potentially protective role of IL-33 in psoriasis, which could partly contribute to the reported decline in disease prevalence and severity with increasing age.

The biological effects of IL-33 are largely determined by the balance between membrane-bound ST2 and its soluble isoform. While signaling through membrane-bound ST2 promotes downstream inflammatory responses, soluble ST2 functions as a decoy receptor that limits IL-33 bioactivity and exerts anti-inflammatory effects (6,18). In psoriasis, the role of sST2 remains controversial. Some studies suggest a protective function through inhibition of IL-33 signaling, whereas others report associations between elevated sST2 concentrations and disease severity in inflammatory conditions such as systemic lupus erythematosus, Still’s disease, cardiovascular disorders, and asthma (6,19–21). In clinical practice, circulating sST2 has also been proposed as a prognostic biomarker in myocardial infarction (2).

In the present study, serum sST2 concentrations did not differ between patients with psoriasis and controls, suggesting that sST2 may not represent a central systemic mediator in psoriasis or that its effects are highly context-dependent. The higher sST2 concentrations observed in male participants are consistent with previous population-based data demonstrating higher circulating sST2 concentrations in men than in women. Lu et al. reported significantly higher sST2 concentrations in healthy men than in healthy women (24.9 vs. 16.9 ng/mL, $p < 0.0001$). However, as the sex-related difference observed in our study was identified in a secondary analysis, this finding should be interpreted cautiously and requires confirmation in further studies (23).

The contribution of the IL-33/ST2 axis appears to vary substantially across inflammatory diseases. Increased tissue and circulating levels of IL-33 and sST2 have been reported in conditions dominated by type 2 immune responses, such as atopic dermatitis, asthma, and systemic lupus erythematosus (7,11,18,20–22). In contrast, psoriasis is primarily driven by Th1/Th17-mediated pathways, in which type 2 inflammation plays a less prominent role. This immunological distinction may partly explain the reduced circulating IL-33 concentrations observed in our cohort and the absence of significant changes in sST2 concentrations.

Beyond cytokine-based biomarkers, hematological indices also provide evidence of systemic inflammation in psoriasis. Çelik and Aktaş reported significantly higher neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) values in patients with psoriasis vulgaris compared with those with lichen planus, and NLR was also significantly higher in patients with psoriatic arthritis (24). These findings highlight the heterogeneous systemic inflammatory profile of psoriasis and provide additional clinical context for the interpretation of circulating inflammatory biomarkers.

Several limitations of this study should be acknowledged. First, the cross-sectional and single-center design limits the ability to establish temporal or causal relationships and may restrict the generalizability of the findings. Second, only circulating IL-33 and sST2 concentrations were assessed, without simultaneous evaluation of Th1/Th17-related cytokines or other systemic inflammatory markers. Importantly, serum measurements cannot directly reflect IL-33/ST2 activity within psoriatic lesions, and simultaneous tissue measurements were not performed. Future studies integrating paired serum and lesional tissue analyses may provide a more comprehensive understanding of the local and systemic regulation of the IL-33/ST2 axis in psoriasis.

Despite these limitations, our findings add to the growing body of evidence indicating that the IL-33/ST2 axis in psoriasis differs fundamentally from that observed in other inflammatory diseases. The dissociation between local tissue activity and circulating cytokine levels highlights the complexity of IL-33 biology in psoriasis and underscores the need for future studies integrating serum, tissue, and immunological profiling in larger and more diverse patient populations.

Conclusions

Circulating IL-33 and sST2 concentrations were assessed in patients with psoriasis and matched controls. The presence of lower serum IL-33 concentrations in psoriasis, together with unchanged sST2 concentrations, suggests that the IL-33/ST2 axis does not represent a predominant systemic pathway in this Th1/Th17-driven disease. Rather, these findings raise the possibility that circulating IL-33 concentrations may not fully reflect local IL-33 activity within psoriatic tissue. These results support the need for future studies combining serum data with tissue-based analyses and comprehensive immunological profiling to better characterize the role of the IL-33/ST2 axis in psoriasis pathogenesis.

Conflict of interest: The authors declare no conflict interest.
Funding source: This study was financially supported by the Turkish Society of Dermatology for the purchase of ELISA kits used to measure serum IL-33 and sST2 levels.
Ethical approval: The study was approved by the Ankara City Hospital No. 1 Clinical Research Ethics Committee (approval number: E1-20-1318; approval date: February 3, 2021).
Informed consent: Written informed consent was obtained from all participants.
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Peer-review: Externally. Evaluated by independent reviewers working in at least two different institutions appointed by the field editor.
Data availability: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.
Contributions:
Research concept and design: UA, GGA
Data analysis and interpretation: UA, YH
Data collection and/or assembly of data: UA, EFO
Writing the article: UA, BA, AA, GGA, EC, YH, EFO
Critical revision of the article: UA, YH
Final approval of the article: UA, BA, AA, GGA, EC, YH, EFO
All authors read and approved the final version of the manuscript.

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