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Serum Interleukin-38 Depletion in Psoriasis and Its Association with Cytomegalovirus Seropositivity: A Case-Control Study in Baghdad, Iraq

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Abstract

Introduction: Interleukin-38 (IL-38) is an anti-inflammatory cytokine that may restrain psoriatic inflammation. Whether cytomegalovirus (CMV) seropositivity contributes to IL-38 depletion and greater disease severity remains unclear. Aim of this study was to compare serum IL-38 concentrations between patients with psoriasis and healthy controls and assess their associations with CMV serostatus and clinical severity.

Methods: This hospital-based case-control study enrolled 45 patients with psoriasis vulgaris and 45 age- and sex-matched healthy controls at Medical City, Baghdad, from January to December 2024. Serum IL-38 was quantified by enzyme-linked immunosorbent assay, CMV immunoglobulin G by chemiluminescence immunoassay, and psoriasis severity by the Psoriasis Area and Severity Index (PASI). Data were analyzed using group comparisons, Pearson correlation, and multiple linear regression.

Results: Mean serum IL-38 was lower in patients than controls (37.78 ± 6.15 vs 118.6 ± 25.8 pg/mL; $P<0.001$). Among patients, 36 (80.0%) were CMV-seropositive and had lower IL-38 concentrations than seronegative patients (31.2 ± 5.5 vs 64.1 ± 6.8 pg/mL; $P<0.001$). IL-38 correlated inversely with PASI ($r=-0.72$; $P<0.001$). CMV seropositivity ($\beta=-0.58$; $P<0.001$) and PASI ($\beta=-0.35$; $P=0.001$) were independently associated with lower IL-38, whereas age was not.

Conclusion: Reduced IL-38 may represent an immunoregulatory deficit linking CMV seropositivity to greater psoriasis severity. IL-38 and CMV serostatus warrant longitudinal evaluation as complementary markers for risk stratification.

Keywords: Skin diseases, Papulosquamous, Cytokines, Virus latency, Severity of illness index

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Introduction

An inflammatory reaction mediated by the immune system is the hallmark of psoriasis, leading to the abnormal growth and maturation of keratinocytes, accompanied by significant immune system irregularities (1). The prevalence of psoriasis is considered to have increased worldwide in the recent 30 years and it has been estimated that nearly 43 million people suffered from psoriasis in the world by 2021, indicating that the global burden of psoriasis had almost doubled since 1990 and putting psoriasis as a major public health problem(2). While the interplay between innate and adaptive immune cells is significantly impacted by the disturbance of the interleukin (IL)-23/IL-17 axis, which is widely acknowledged as a critical component in the immunopathogenesis of psoriatic illness(3), recent immunological developments have focused more attention on the potential regulatory role of the IL-1 cytokine family, particularly Interleukin-38 (IL-38), in inflammation-related processes.(4).

IL-38 is a protein composed of 152 amino acids and represents a novel anti-inflammatory addition to the IL-1 family of cytokines. It functions as a potent receptor antagonist by binding to the Interleukin-36 receptor (IL-36R) and the Interleukin-1 receptor accessory protein (IL-1RAP), exhibiting biological functions similar to those of the IL-36 receptor antagonist (IL-36Ra)(5). When IL-38 occupies these receptors, it acts as a crucial immunological "molecular brake," dramatically reducing the generation of pro-inflammatory cytokines linked to Th17 and the subsequent signaling cascades of pro-inflammatory mediators like IL-36(6). Recent research has demonstrated that L-38 directly reduces inflammation in psoriasis, and that it is positively connected with responsiveness to anti-IL-17 biological agent therapy and inversely correlated with the severity of the illness(7). Reduction of this immunoregulatory cytokine could therefore be a significant mechanism that exacerbates the condition.

Psoriasis is influenced by a number of factors, including intricate relationships between genetic factors and environmental influences, with particular focus on the role of latent viral infections (8). These viral agents can modulate host immunity, dysregulate antiviral immune responses, and disrupt regulatory gatekeepers, potentially exacerbating chronic systemic inflammation(9). Human Cytomegalovirus (HCMV), a ubiquitous beta-herpesvirus, establishes lifelong persistence within the host, particularly in myeloid progenitor cells. Chronic, latent CMV infection has been implicated in driving a state of persistent low-

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grade inflammation and profound "immune exhaustion," characterized by T-cell senescence, altered cytokine profiles, and impaired immunoregulatory capacity(10). In the Middle Eastern region, including the Iraqi population, the seroprevalence of CMV is notably high, with epidemiological studies reporting rates exceeding 90% in certain demographic groups (11). However, the synergistic interaction between this highly prevalent latent viral infection and novel regulatory cytokines, such as IL-38, in the context of psoriasis remains largely unexplored.

Considering the known anti-inflammatory role of IL-38 and the immune-exhausting characteristics of latent CMV infection, we hypothesized that psoriatic patients have significantly lower serum IL-38 levels than healthy controls. Additionally, we suggested that latent CMV infection is independently linked to further reductions in IL-38 and heightened clinical disease severity, regardless of age. Thus, this study aims to measure serum IL-38 levels in Iraqi patients with psoriasis vulgaris in comparison to healthy controls. It aims to determine the correlation between clinical disease severity as measured by the Psoriasis Area and Severity Index (PASI) and IL-38 levels. Furthermore, it will assess the precise impact of latent CMV infection on IL-38 depletion, differentiating this viral-induced immune exhaustion from the aging process.

Methods

Study Design

A case-control study in a hospital will be conducted between January 1, 2024, and December 31, 2024. Patients were clinically evaluated and enrolled in the study at the visit to the Dermatology Consultant Clinic of the Medical City Hospital, a tertiary center in Baghdad – Iraq. Further serological and immunological investigations were conducted in the Central Clinical Laboratory of Medical City. The protocol of the study was developed and is reported following rigorously the STROBE statement for case-control studies (12).

Participant Selection and Criteria

A total of 90 subjects were included in the study. Using G*Power software (version 3.1.9.7), the sample size was determined a priori(13). A minimum of 45 patients per group were needed since we calculated a medium effect size (Cohen's d = 0.6) for the key outcome parameter (serum IL-38 levels), an α error probability of 0.05, and a statistical power of 85%.

Patients and controls Group A: Psoriasis patients n = 45) Patients who were between the ages of 18 and 65 and had active, clinically diagnosed Psoriasis Vulgaris, as assessed by a senior consultant dermatologist. Clinical disease severity was measured quantitatively by the PASI score, a validated, composite index of erythema, induration and desquamation, which also assesses the body surface area involved by psoriatic plaques(14). Patients were systematically excluded if they had received systemic immunosuppressive treatment (eg, methotrexate, cyclosporine), biological therapies, or phototherapy within 3 months prior to enrollment. Other exclusion criteria were coexisting autoimmune connective tlis s (e.g. Rheumatoid arthritis), known malignancy or acute bacterial or viral infection. Group B: Healthy Controls (n = 45) the control group consisted of healthy control participants without personal or family history of psoriasis, other dermatological illnesses, or systemic inflammatory diseases. To avoid demographic bias, this cohort was closely matched to the patient group by mean chronological age (38.95 ± 10.2 years) and sex (male: 27, female: 18)

Sample Collection and Processing

The samples were taken following a minimum of eight hours of fasting overnight. A simple gel-activator vacutainer tube was filled with a 5 mL sample of whole blood, which was then allowed to coagulate for 20 to 30 minutes at room temperature (20 to 25°C). Centrifugation at 3,500 rpm for 10 minutes at 4°C was then used to separate the serum. To maintain protein integrity and prevent degradation due to successive freeze-thaw cycles, prior to the biochemical examination, the serum was immediately divided into three sterile, endotoxin-free Eppendorf tubes and frozen at about 80°C.

Laboratory Assays

Measurement of Serum IL-38 Human IL-38 ELISA The human IL-38 levels in serum samples were measured using a quantitative sandwich ELISA kit with very high sensitivity (Elabscience; Cat. No. E-EL-H6136), following the instruction of the manufacturer's protocol (15). The assay sensitivity for IL-38 was determined as 10 pg/mL with intra- and inter-assay CV < 10%.

Virological Screen for Latent CMV The serological latent CMV status was determined by assaying specific Immunoglobulin G (IgG) antibodies to CMV by an automated Chemiluminescence Immunoassay (CLIA) system (Abbott Architect i2000SR, Abbott Park, IL, USA) (16). The results were quantitatively reported as Arbitrary Units per milliliter (AU/mL). Following the clinical cut-off values established by the manufacturer, serum samples with values > 6.0 AU/mL were considered to be seropositive indicating previous contact and acquisition of latent CMV

Statistical Analysis

The Statistical Package for the Social Sciences, version 26.0 (SPSS Inc., Chicago, IL), was used for all analyses. The continuous variables' normality of distribution was evaluated using the Shapiro-Wilk test (all continuous variables, $P > 0.05$). Thus, parametric tests were applied. The inferential statistical analyses for two independent groups (eg, patients vs controls; CMV + vs. A one-way ANOVA was used to compare more than two groups (such as age groups), and Tukey's post-hoc test was used to correct for multiple testing. The Cohen's d was used to assess the extent of the differences. Bivariate associations between two continuous variables (IL-38 concentration, PASI scores and CMV IgG titers) were analyzed by Pearson's r coefficient. To identify independent predictors of serum IL-

38 depletion and adjust for possible confounders a Multiple Linear Regression model was built, using age, sex, CMV serostatus and PASI score as predictors. A result of less than 0.05 was regarded as significant.

Results

In this study, 90 subjects were recruited, including 45 with diagnosed psoriasis vulgaris (Group A) and 45 healthy controls matched for age and sex (Group B). The patients' mean age was 39.2 ± 11.4 years, which was comparable to the control group's age of 38.9 ± 10.8 years ($P = 0.89$).The gender distribution was the same for both groups (male:27; female:18), further indicating that the pairing method was effective. "In psoriasis patients, mean PASI was 16.5 ± 6.7, revealing a mainly moderate-to-severe disease phenotype in the study population (Table 1 and Figure 1).

Psoriasis patients had considerably lower serum levels of IL-38 (37.78 ± 6.15 pg/mL) compared to healthy individuals (118.6 ± 25.8 pg/mL). This was extremely significant (t = -18.2, $P < 0.001$), meaning that the sick group's levels of circulating IL-38 were 68.1% lower than those of the controls.

Figure 2 indicates that seropositive patients for CMV have consistently significantly lower levels of IL-38 at all age ranges than those seronegative for CMV, implying that the virus-associated decrease in IL-38 is not influenced by the aging in years. Results among the 45 patients with psoriasis, 36 individuals (80.0%) were positive for CMV IgG, and 9 individuals (20.0%) were seronegative. CMV-positive patients had significantly lower levels of serum IL-38 than CMV-negative patients. A detailed stratification of IL-38 levels according to age group and CMV serostatus is presented in Table 2. Notably One-way ANOVA of IL-38 levels in the three age groups within the CMV-seropositive group revealed no significant difference ($F(2, 33) = 1.45$, $P = 0.24$).

Bivariate correlation results showed a substantial and inverse relationship between serum IL-38 levels and PASI scores (r = -0.72, $P < 0.001$). Nevertheless, there was a modest and non-significant correlation (r = -0.18, $P = 0.08$) between IL-38 levels and chronological age. CMV IgG antibody titers and serum IL-38 concentrations had an unfavorable relationship (r = -0.61, $P < 0.01$). Figure 3 shows the correlation between clinical severity of illness and serum IL-38. It was discovered that the correlation was significantly inverse linear (r = –0.72, $P < 0.001$).

The model explained 74% of the variation in serum IL-38 (Adjusted R2 = 0.74) and was significant ($F(4, 40) = 28.5$, $P < 0.001$). Table 3 presents the regression coefficients for every predictor variable. Regression analysis revealed that the most powerful independent predictor of decreased serum IL-38 ($\beta = -0.58$, $P < 0.001$) and PASI score ($\beta = -0.35$, $P = 0.001$) was CMV seropositivity. On the other hand, sex ($\beta = 0.06$, $P = 0.54$) and chronological age ($\beta = -0.12$, $P = 0.21$) were not statistically significant independent predictors.

Discussion

Three main conclusions were drawn from the current study: First, compared to age- and sex-matched healthy

Table 1. The study population's baseline clinical and demographic characteristics

Parameter	Psoriatic Patients (n = 45)	Healthy Controls (n = 45)	*P*-value
Age (years), Mean ± SD	39.2 ± 11.4	38.9 ± 10.8	0.89
Sex (Male/Female)	27/18	27/18	1.00
PASI Score, Mean ± SD	16.5 ± 6.7	N/A	—
Serum IL-38 (pg/mL), Mean ± SD	37.78 ± 6.15	118.6 ± 25.8	<0.001*
CMV IgG Seropositivity, n (%)	36 (80.0%)	—	—

* Statistically significant at $P < 0.05$.

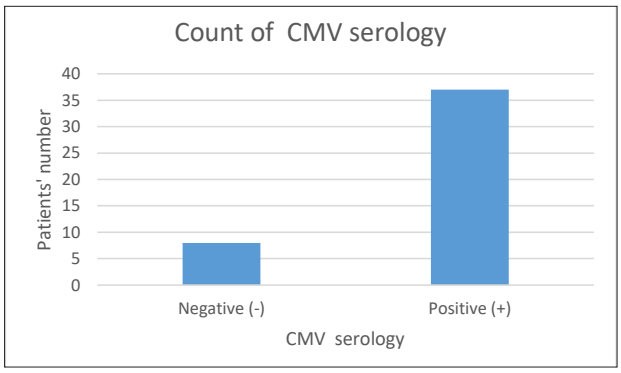

Figure 2. Chart Illustrating the Distribution of Psoriatic Patients by Cytomegalovirus IgG Serostatus

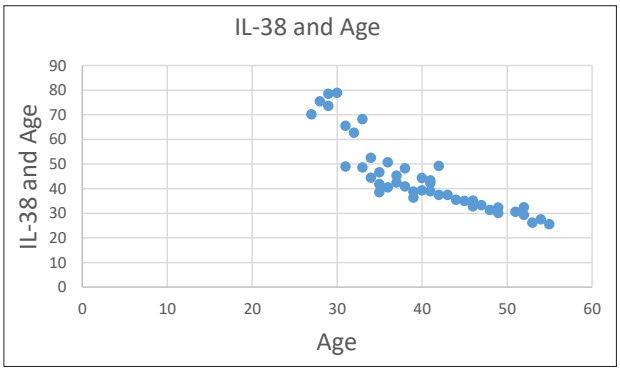

Figure 1. Bar Graph Illustrating Age-Stratified Serum IL-38 Concentrations in Psoriatic Patients According to Cytomegalovirus Serostatus

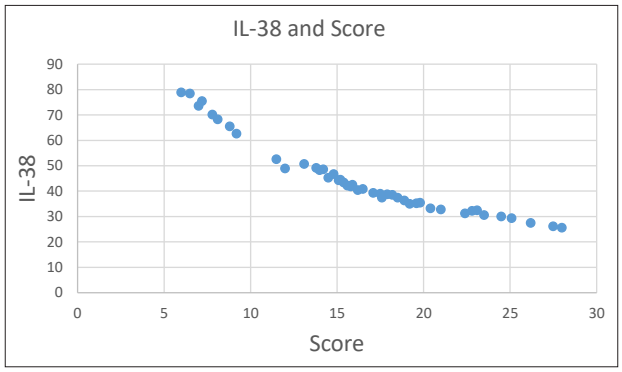

Figure 3. Serum IL-38 Levels and PASI Score in an Inverse Correlation Scatter Plot

Table 2. Serum IL-38 Levels Stratified by Age Group and CMV Serostatus in Psoriatic Patients

Age Group (Years)	n	CMV + n (%)	IL-38 (CMV +) Mean ± SD	IL-38 (CMV–) Mean ± SD	*P*-value	Cohen's d
Young (20–35)	14	10 (71.4%)	38.5 ± 5.2	70.2 ± 6.4	<0.001*	5.4
Middle (36–50)	22	19 (86.4%)	30.2 ± 4.1	62.5 ± 5.8	<0.001*	6.3
Older (>50)	9	7 (77.8%)	28.5 ± 3.9	58.1 ± 4.5	<0.001*	7.1
Total	45	36 (80.0%)	31.2 ± 5.5	64.1 ± 6.8	<0.001*	5.2

CMV + = seropositive; CMV– = seronegative. * At $P < 0.05$, statistically significant.

Table 3. Independent Predictors of Serum IL-38 in a Multiple Linear Regression Model

Variable	Unstandardized β (B)	Standard Error	Standardized β	t-value	*P*-value
(Constant)	128.5	12.2	—	10.53	<0.001
CMV Serostatus (Positive)	-32.4	6.1	-0.58	-5.31	<0.001*
PASI Score	-2.8	0.8	-0.35	-3.50	0.001*
Age (Years)	-0.15	0.12	-0.12	-1.25	0.21
Gender (Male)	2.1	3.4	0.06	0.62	0.54

* Statistically significant at $P < 0.05$. Dependent variable: Serum IL-38 (pg/mL). Adjusted $R^2 = 0.74$.

controls, patients with psoriasis vulgaris had significantly lower circulating IL-38 concentrations. Second, lower IL-38 levels were associated with greater clinical severity, as demonstrated by the strong inverse correlation with PASI. Third, within the psoriasis group, CMV-IgG-seropositive patients had lower IL-38 concentrations than seronegative patients, and both CMV serostatus and PASI remained independently associated with IL-38 in the multivariable model, whereas age and sex did not. Taken together, these findings support the involvement of impaired IL-38-associated counter-regulation in psoriasis and identify prior CMV exposure as a potential correlate of dysregulation. However, because the study was observational and CMV status was defined serologically, the findings establish an association rather than a causal effect of CMV on IL-38 production.

Our study's findings of decreased serum IL-38 in the psoriasis group are consistent with those of Mercurio et al, who reported decreased IL-38 in psoriatic skin and circulation. Additionally, they found that the serum ratio of the IL-36γ agonist to the IL-38 antagonist was moved in favor of IL-36γ and linked with the severity of the disease; IL-38 also increased after secukinumab therapy in conjunction with clinical improvement (7). More recently, Mohamed et al. found an inverse relationship between IL-38 and PASI and lower circulating levels of IL-38 linked to psoriasis as compared to healthy controls. Additionally, their research showed that patients with metabolic syndrome had significantly lower levels of IL-38(17). The interpretation that lower IL-38 levels are connected to the inflammatory burden in psoriasis is strengthened by the consensus of these investigations. Albeit, absolute concentrations should not be directly compared between studies considering the types of specimen, the ELISA plate form, the tool of calibration, the way of handling samples, the phenotype of disease, exposure to treatment and the characteristics of population.

The inverse correlation between IL-38 and PASI is biologically plausible within the context of the IL-36/IL-23/IL-17 inflammatory axis. IL-38 can counteract certain IL-36-mediated effects and inhibit the release of mediators involved in keratinocyte activation and leukocyte migration(5–7). Han et al. showed that in experimental psoriasis, IL-38 deficiency increased γδ T cell production of IL-17 and delayed the resolution of imiquimod (IMQ)-induced skin inflammation, whereas treatment with mature IL-38 suppressed this response(18). These observations are consistent with a model of insufficient IL-38 function, allowing IL-17-dominant inflammation to persist. However, the action of IL-38 should not be reduced to a general anti-inflammatory brake. Keratinocyte-specific IL-38 overexpression improved a subset of inflammatory and desquamation-related indicators, but it did not lessen the overall severity of imiquimod-induced murine skin inflammation, according to a recent study by Huard et al. (19). Therefore, IL-38 effects could be dependent on tissue, cell, dose, and context, and low serum values could be an indicator of disrupted inflammation rather than the initiator.

The first novel finding of the current study is that IL-38 levels are decreased in CMV-seropositive individuals. Previous studies on psoriasis have indicated a potential interaction between CMV and active cutaneous inflammation; however, there is a dearth of information on this topic. According to Asadullah et al, only 12% of healthy laboratory personnel and 6% of blood donors had CMV antigenaemia, compared to 43% of patients with moderate-to-severe plaque psoriasis. Antigenaemia declined during antipsoriatic therapy and correlated with circulating tumor necrosis factor (TNF)-α; in contrast to this, serological examination did not reveal an increased rate of former CMV infections in patients with psoriasis and CMV DNA was only sporadically found in lesions(20). Then Weitz et al. describe higher psoriasis severity in CMV-seropositive patients, a positive correlation of severity with CMV antigenaemia, and a decrease of circulating CMV-specific T-cells in seropositive patients as compared to seropositive controls(21). Our results are in agreement with these

reports; however, they enhance these reports by showing a link between CMV IgG serostatus and a regulatory IL-1-family cytokine.

A few differences between the current study and previous studies on CMV are important for interpretation. CMV IgG seropositivity reflects past exposure and is consistent with lifelong viral persistence, but does not indicate whether there is active replication, recent reactivation, the size of the latent viral reservoir, or activity of CMV in psoriatic skin. Previous psoriasis studies have employed measurements of antigenaemia, viral DNA, or CMV-specific T cells, which cannot be extrapolated from IgG positivity(20,21). Clinical case reports on equivocal CMV serology also stress the benefit of CMV PCR and, if suitable, IgG avidity testing in separating active or recent infection from remote exposure when differentiation is needed for management purposes(22). Therefore, the diminished IL-38 levels in seropositive individuals should be considered an association with prior CMV exposure and not as confirmation of CMV-mediated IL-38 'exhaustion.

However, a mechanistic association is still possible and merits direct investigation. Chronic CMV infection can significantly affect the remodeling of the T-cell compartment, including the preferential expansion of highly differentiated effector-memory subsets and other alterations associated with immunosenescence(10,23,24). The immune remodeling described may synergize with the IL-23/IL-17 axis or IL-36-dependent signalling in psoriasis. On the other hand, systemic inflammation observed in patients with active psoriasis could enhance subclinical CMV activation, as evidenced by the link between TNF-α and antigenaemia in the study by Asadullah et al.(20). Therefore, a two-way model is more admissible than a single causal model: inflammation in psoriasis may affect the control of CMV, while CMV-driven immune remodeling could influence the inflammatory phenotype. The present cross-sectional data do not shed light on which of these processes leads to the other and cannot establish a direct molecular pathway linking CMV to IL-38 expression.

Multivariate analysis provided evidence that the associations between CMV serostatus and PASI with IL-38 were not explained by age or sex within this sample. The larger standardized coefficient for CMV serostatus than for PASI suggests that CMV status contributed substantial, independent statistical information to the model. However, this comparison should be interpreted cautiously. The unstandardized coefficient for the binary CMV variable and the coefficient for age measured in years have different units and should not be compared as a ratio. Moreover, lack of a statistically significant age effect does not demonstrate that aging has no biological influence on IL-38; the restricted age range, modest sample size, and small CMV-seronegative subgroup may have reduced the ability to detect age-related effects. The very large subgroup effect sizes may also be unstable because only nine patients were CMV-seronegative, and the age-stratified seronegative cells contained very few participants.

These findings have potential biomarker relevance but do not yet justify changes in clinical management. Serum IL-38 may be useful as a research marker of inflammatory activity, particularly if longitudinal changes track treatment response, as previously reported(7). Similarly, CMV serostatus may help define biologically distinct subgroups for future investigations. Nevertheless, the current evidence is insufficient to recommend routine CMV screening, intensified immunomodulatory therapy based on CMV IgG alone, antiviral treatment, or IL-38 supplementation. Before clinical application, the incremental value of IL-38 and CMV measures must be demonstrated beyond established clinical indices, and clinically useful thresholds, reproducibility, sensitivity to change, and associations with treatment outcomes must be established in independent cohorts.

The strengths of our study are the age- and sex-matched controls, the use of standardized clinical assessment with PASI, the quantitative determination of IL-38 and CMV IgG, and the multivariable analysis of the key associations. It is important to acknowledge the limitations of this study. The study's observational, single-center design makes it difficult to determine temporal correlations and extrapolate the results. The a priori determined sample of patients was small, particularly in the CMV-seronegative stratum. CMV IgG was not supported by CMV DNA, antigenaemia, IgM/avidity, or CMV-specific cell-mediated immunity; hence, active or subclinical reactivation could not be assessed. IL-38 was assessed only in serum and not in lesional and non-lesional skin at the transcript level or in biologically active IL-38 isoforms. Residual confounding may also exist as body mass index, metabolic syndrome, smoking, socioeconomic status, disease duration, topical treatment, and other chronic infections were not fully adjusted for in the model. This is of particular importance in light of the recent association between metabolic syndrome and decreased IL-38 levels in psoriasis(17). Finally, the viability of the reported subgroup means and test statistics should be checked against the raw data prior to publication.

In the future, studies should implement multicenter longitudinal designs, with larger CMV-seronegative control groups and serial assessments of IL-38, PASI, and treatment response. The combination of CMV IgG with quantitative PCR, antigenaemia, avidity testing where applicable and CMV-specific T-cell phenotyping might aid in the differentiation between remote exposure and viral activity and immune remodeling. The simultaneous analysis of IL-38 in serum and in paired lesional and non-lesional skin together with IL-36, IL-17, IL-23, TNF-α, and other cellular mediators may also help elucidate whether the association detected in our study is due to increased/decreased production, consumption, processing, or redistribution of IL-38. Such studies are required to establish whether CMV represents a causal alterer of IL-38 biology in psorasis or a marker of generalized inflammatory and demographic exposures (25-27).

Conclusion

In comparison to matched healthy controls, the Iraqi

psoriasis vulgaris patients in this study exhibited significantly lower serum IL-38 concentrations, and lower IL-38 was linked to higher PASI-defined disease severity. Among the patients, CMV IgG seropositivity was independently associated with lower IL-38 levels after adjusting for age, sex, and PASI. These findings support IL-38 as a candidate biomarker of psoriatic inflammatory activity and identify prior CMV exposure as a hypothesis-generating correlate of impaired cytokine regulation in psoriasis. However, they did not demonstrate active CMV infection, CMV-mediated IL-38 depletion, or a therapeutic indication for antiviral treatment or IL-38 replacement. Validation in larger longitudinal cohorts incorporating direct virological and tissue-level measurements is required before clinical implementation.

Competing Interests

None.

Data Availability Statement

Upon reasonable request, the corresponding author will provide the data used to support the study's conclusions.

Ethical Approval

The study protocol received approval from the IRB of the Iraqi Ministry of Health (No: MOH/IRB/2024-112, date 5/1/2024). This work was conducted in alignment with the ethical standards established in the Declaration of Independence of Helsinki for medical research involving human subjects.

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