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Original Article

Relationship Between Interleukin-23 Serum Level, Disease Activity, and Different Disease Parameters in Systemic Lupus Erythematosus Patients

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Abstract

Background: Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disorder marked by the overproduction of antibodies that attack the body's tissues, especially focusing on double-stranded DNA (dsDNA) and small nuclear RNA-associated proteins like Sm, Ro, La, and nuclear RNP. The manifestations of SLE often involve the integumentary, muscular and skeletal, renal, and blood-forming systems. Research has correlated increased serum concentrations of interleukin-23 (IL-23) with the development of several autoimmune diseases. The objective of the current investigation was to evaluate the serum concentration of IL-23 in patients with SLE and to investigate its potential correlation with various clinical parameters, including disease activity. **Methods:** This research investigation enrolled 50 individuals with SLE, identified according to the 2019 EULAR/ACR classification criteria, alongside a control group of 50 healthy subjects matched for age and sex. The SLE Disease Activity Index (SLEDAI) served to quantify disease activity among the SLE patients. Laboratory evaluations included ESR, anti-nuclear antibody (ANA), anti-double stranded DNA (anti-dsDNA) antibody, complement components 3 and 4 (C3 and C4), and CRP. Interleukin-23 (IL-23) concentrations in serum were quantified in both the patient and control groups employing an ELISA technique. **Results:** The study revealed a notable elevation in serum IL-23 levels in individuals diagnosed with SLE, exhibiting a mean concentration of 82.1 ± 75 mg/dL, in contrast to the healthy control group, which presented a mean of 44.9 ± 10.6 mg/dL (P-value < 0.001). **Conclusions:** The levels of IL-23 are significantly higher in individuals with SLE than in those without the condition.

Keywords:

Interleukin, Systemic lupus erythematosus, Systemic Lupus Erythematosus Disease Activity Index.

Introduction

SLE represents a multifaceted autoimmune disease affecting multiple organ systems, defined by its heterogeneous clinical presentation, fluctuating disease trajectory, and common recurrences. The etiology of SLE stems from aberrant T and B cell activation, impaired programmed cell death, and insufficient removal of immune complexes. Consequently, excessive autoantibody production occurs, leading to the formation of

immune complexes through binding to chromatin and the subsequent triggering of inflammation (1).

Most of autoimmune diseases are related to dysregulation of inflammatory cytokines. IL-23 (heterodimeric cytokine,) is produced by macrophages, keratinocytes, dendritic cells, and other antigen-presenting cells. IL-23 belongs to the IL-12 family, which plays an important role in the inflammatory process through interaction with the IL-23 receptors, including the induction of Th17 cell (2).

IL-23 plays a pivotal role in driving the expansion and activation of Th17 cells, a subset of CD4+ T cells that secrete the pro-inflammatory cytokine IL-17. In turn, IL-17 stimulates the release of additional inflammatory mediators, including IL-1, IL-6, TNF-α, nitric oxide synthase-2, and various chemokines. This inflammatory cascade triggers the activation of macrophages and granulocytes, leading to tissue damage, sustained inflammation, and the emergence of clinical features typical of autoimmune diseases (3)

IL-23 promotes the proliferation of a subtype of CD4+ T cells (Th17) that produce IL-17 (a proinflammatory cytokine) which induces the release of IL-1, IL-6, tumor necrosis factor-α, nitric oxide synthase-2, and other chemokines. These pathogenic mediators foster the rapid stimulation of macrophages and granulocytes to cause tissue damage that induces chronic inflammation and the development of clinical symptoms (3).

IL-17 is more potent as a predictor of SLE disease activity than other cytokines (IL-4 & INF-γ). The IL-23 IL-17 axis is a regulatory system that is crucial in connecting both arms of the immune system; innate and adaptive. It also plays an important role in the evolution of inflammatory autoimmune diseases. Theoretically, all antibodies directed against the IL-23/IL-17 axis that are approved for the treatment of other inflammatory rheumatic diseases could be tested in patients with SLE. The treatment of SLE is currently facing major challenges. The clinical heterogeneity of SLE makes it clear that processes are responsible for the disease manifestations in defined subgroups of patients must be targeted by biologics (4).

Aim of the study

Assessment of serum level of IL-23 in SLE patients and evaluation of its relation to disease manifestations and activity.

Subjects and Methods

Subjects

This case-control, cross-sectional study was conducted at Fayoum University Hospital, involving 50 patients who satisfied the 2019 European League Against Rheumatism (EULAR)/American College of Rheumatology (ACR) Classification Criteria for SLE (3). In addition, 50 healthy controls, matched for age and sex, were included, we selected them from patients who sought medical advice for non autoimmune disorders, e.g. osteoarthritis patients and some hospital workers. The SLE patients were recruited from the Rheumatology and Rehabilitation Department at Fayoum University Hospital between July 2022 and January 2023.

Inclusion criteria

1.Age of patients: more than 16 years.
2.Sex: both sexes.
3.All Patients must be fulfilling the 2019 EULAR/ACR Classification Criteria for SLE (5)

Exclusion criteria

1.Individuals with other rheumatic diseases.
2.Individuals with malignant tumors.
3.Individuals with infections

Study design

A comprehensive approach was employed for all patient participants, involving detailed medical history acquisition, meticulous clinical evaluations, and a battery of laboratory investigations. The laboratory and immunological evaluation included a complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), 24-hour urinary protein measurement, antinuclear antibody (ANA) testing, anti-double stranded DNA (anti-dsDNA) antibody levels, and serum complement (C3 and C4) levels. Disease activity was quantified using the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), while the extent of accumulated organ damage was assessed using the Systemic

Lupus International Collaborating Clinics/American College of Rheumatology (SLICC/ACR) Damage Index (SDI).

Serum levels of IL-23 in all participants were quantified using ELISA with specific cytokine kits, following the manufacturer's guidelines (catalog number 74 HU). All collected data were systematically organized and analyzed to derive the study's findings.

The disease activity for all participants was evaluated utlizing the SLEDAI scoring system, a validated instrument developed to quantify the severity and course of SLE (6).

The severity of SLE was evaluated using the Systemic Lupus International Collaborative Clinics/American College of Rheumatology (SLICC/ACR) Damage Index (SDI), a validated tool designed to assess cumulative organ damage caused by the disease over time (6).

The SLICC/ACR Damage Index (SDI) is a tool used to quantify cumulative, irreversible organ damage in SLE. It captures non-reversible changes that have occurred since the onset of the disease, as determined through clinical evaluation, and excludes damage attributable to active inflammation.

Serum IL-23 concentrations in patients and controls were quantified using ELISA method, which operates on a sandwich immunoassay principle. The assay utilized a microplate pre-coated with a monoclonal antibody specific to Human IL-23, allowing IL-23 in serum samples to bind to the immobilized antibodies. A biotinylated antibody specific to IL-23 was then added, forming a complex with the captured IL-23, followed by Streptavidin conjugated to horseradish peroxidase (HRP) to bind the biotinylated antibody. After washing away unbound HRP, a chromogenic substrate was introduced, resulting in a color change proportional to the IL-23 concentration. The reaction was stopped with an acidic solution, and absorbance was measured at 450 nm using a microplate reader. All reagents, standards, and samples were prepared and handled according to the manufacturer's guidelines, with the assay performed entirely at room temperature to ensure accuracy.

Statistical analysis:

Data analysis were conducted with SPSS software, version 25. The dataset's characteristics were outlined through descriptive statistics, where categorical data were expressed in numbers and percentages, and continuous data were represented by their mean, standard deviation, median, Interquartile range (IQR), and minimum, maximum values. Group comparisons of percentage distributions were evaluated via chi-squared test. Mean comparisons for normally distributed continuous data between two groups were performed using the independent t-test. Alternative non-parametric methods were employed for continuous data that did not conform to a normal distribution; specifically, the Mann-Whitney U test was used to compare two groups, while the Kruskal-Wallis test was applied when comparing more than two groups.

Statistical significance was determined using the following thresholds: $P > 0.05$ (not significant), $P < 0.05$ (significant), and $P < 0.01$ (highly significant).

Furthermore, the performance of diagnostic tests was evaluated using Youden's J statistic, a metric that integrates sensitivity and specificity. Youden's J was computed using the formula:

$J = \text{sensitivity} + \text{specificity} - 1.$

This index offers a holistic assessment of a diagnostic test's capacity to accurately classify both true positive and true negative results.

Results

A cross-sectional case-control study design was implemented, encompassing a total of 100 individuals. The cohort was bifurcated into two groups: 50 patients diagnosed with SLE and 50 healthy control subjects, matched for age and sex. The participants' ages spanned from 18 to 57 years, with a mean age of 34.1 ± 9.9 years. The SLE patient group comprised 2 males (4.0%) and 48 females (96%).

Statistical analysis revealed the distribution of age and gender did not differ notably between people with SLE and the healthy control group (P-value > 0.05). Within the cohort of SLE patients, the duration of the disease ranged from

1 to 20 years, with a mean duration of 5.6 years and a standard deviation of 4.3 years.

Clinical data of SLE patients: among our fifty patients there were: forty-eight patients (98.0%) had photosensitivity, arthritis was found in 46 patients (92%), forty-five patients (90%) had malar rash, alopecia was found in 45 patients (90%), 32 patients (64%) had oral ulcers, Fever was found in 29 patients (58%).

Serositis in the form of pleurisy, pericarditis and/or pericardial effusion was found in 14 patients (28%).13 patients (26%) have renal affection, 8 patients (16%) had discoid rash. Hematologic disorders in the form of leucopenia and/or thrombocytopenia were found in 3 patients (6%). Figure 1

Immunosuppressive Medications:

All patients included in our study were on prednisolone and hydroxychloroquine . Steroid dose ranged from (5) to (60) milligram (mg) per day at the time of sample collection. Hydroxychloroquine dose ranged from 200 to 400 mg/day. Immunosupressive was prescribed for 24 cases (48%) among the studied SLE patients.

Activity and chronicity indices:

SLE disease activity and chronicity were judged by SLEDAI score and SLICC/ACR SDI respectively. SLEDAI score for all cases have disease activity (moderate to very high activity) ranged from 7 to 29 with a mean of 16±5.7. The SDI score showed no damage in 48 cases (96%) and damage only in 2 cases (4%). Table 1

Comparison between cases and controls regarding serum level of interleukin 23

IL-23 levels were pointedly elevated in SLE people than healthy controls, with mean values of 82.1 mg/dL in SLE cases and 44.9 mg/dL in healthy individuals, demonstrating a statistically substantial variant (p-value = 0.001). Table 2, Figure2

IL 23 level in unlike SLE associated clinical indicators and laboratory investigations: No detected substantial variance in the mean concentration of interleukin 23 was found related to the clinical manifestations, immunosuppressive use, investigations or disease activity and chronicity indices (P-value>0.05). This present study found that interleukin 23 had a significant role in prediction of the presence of SLE at a cut off 55.5 with sensitivity 50% and specificity 94% as shown in table (3).

Table 1: Activity and chronicity indices

		Mean (SD)	**Median (IQR)**
SLEDAI		16 (5.7)	13.5 (12-20)
		N	**%**
SLEDAI3	Moderate activity	6	12.0%
	High activity	30	60.0%
	Very high activity	14	28.0%
SLICC	Without damage	48	96.0%
	With damage	2	4.0%

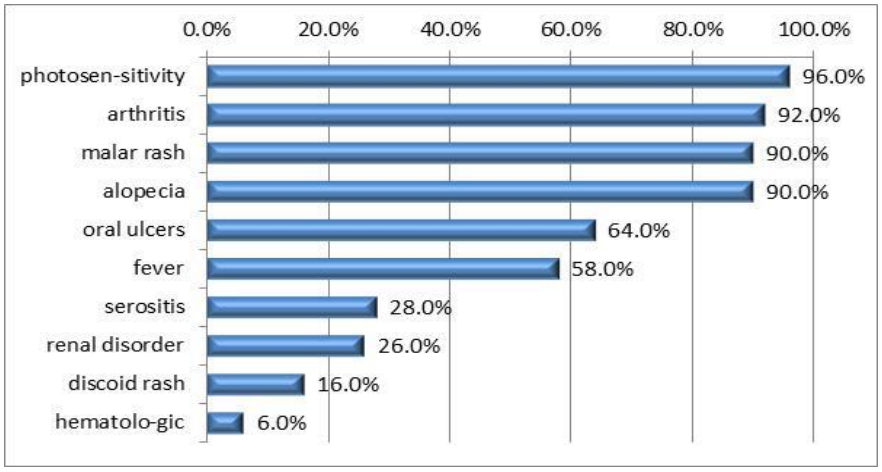

Figure 1: Clinical presentations among SLE cases

Table 2: Difference between cases and controls regarding IL23

SLE	Mean (SD)	Median (IQR)	P-value#
Cases (N=50)	82.1 (75)	55 (42-93)	**0.001***
Controls (N=50)	44.9 (10.6)	44 (40-50)	

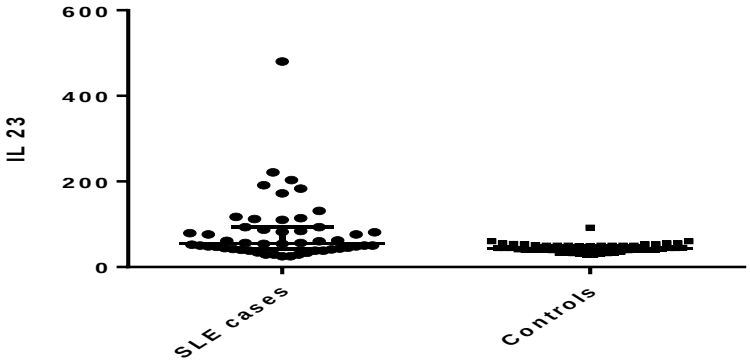

Figure 2: Difference between cases and controls regarding IL23

Table 3: Validity of IL23 in differentiating cases from controls

	AUC (95% CI)	P-value	Cut-off point	Sensitivity	Specificity
IL23	0.695 (0.588–0.803)	0.001*	51.5	56%	80%
			53.5	54%	86%
			55.5#	**50%**	**94%**
			60.5	44%	98%

Cut-off point with maximum Youdin index * statistically significant

Discussion

SLE represents a multifaceted autoimmune disease affecting multiple organ systems and is defined by its heterogeneous clinical presentation, fluctuating disease trajectory, and common recurrences. The pathogenesis of SLE involves aberrant T and B cell activation, impaired programmed cell death, and insufficient removal of immune complexes. These irregularities lead to excessive autoantibody production, resulting in immune complex formation through binding to chromatin and the subsequent triggering of inflammatory responses.

A hallmark of autoimmune disorders, including SLE, is the aberrant regulation of inflammatory cytokines. Interleukin-23 (IL-23), a heterodimeric cytokine, is synthesized by macrophages, keratinocytes, dendritic cells, and other antigen-presenting cells. As a member of the IL-12 family, IL-23 is pivotal in driving inflammation through its interaction with IL-23 receptors. A principal function of IL-23 is the promotion and maintenance of Th17 cells, a subset of T helper cells implicated in the inflammatory cascade characteristic of autoimmune diseases (2).

The IL-23/IL-17 axis serves as a critical regulatory pathway connecting innate and adaptive immunity, and it significantly contributes to the pathogenesis and advancement of inflammatory autoimmune disorders. This axis is especially relevant in the context of SLE. Hypothetically, therapeutic antibodies designed to inhibit the IL-23/IL-17 pathway, currently approved for the management of other inflammatory rheumatic conditions, could be investigated as prospective therapies for SLE (Li H and Toskos, 2021). The involvement of IL-23 in the pathogenesis of SLE is strongly suggested due to its role in the promotion of inflammation and autoimmune responses (7).

The identification of new biomarkers for evaluating disease activity in SLE continues to be a crucial research focus. With a growing number of therapeutic agents progressing to phase II and III clinical trials for SLE and lupus nephritis, there is an urgent requirement for dependable biomarkers capable of predicting therapeutic effectiveness. This need is underscored by the significant heterogeneity of SLE and the incomplete understanding of its underlying etiopathogenesis. The discovery of such biomarkers would enable more tailored treatment approaches, refine clinical trial design, and ultimately improve patient outcomes (8).

The objective of the present study was to assess serum IL-23 concentrations in individuals with SLE and to explore their correlation with various disease characteristics, including disease activity. This case-control study comprised 50 SLE patients and 50 healthy control subjects matched for age and sex. A thorough evaluation of the SLE patients included detailed medical history acquisition, clinical examinations, laboratory tests, and interdisciplinary consultations with other medical specialties as needed. Serum IL-23 levels were measured in both SLE patients and controls using ELISA method.

Of the 50 SLE patients participating in the study, 48 were female and 2 were male, yielding a female-to-male ratio of 24:1. This observed gender distribution is consistent with the established prevalence of SLE among females.

The finding of Female predominance among our SLE patients is in accordance with many other studies and clinical trials, and is supported by the well documented characteristic feature of SLE to be more common in females due to many etiopathogenetic factors. While the variable female to male ratios, can be explained by different patients' demographic and racial factors in different studies. Female predominance was found with a ratio of 6.8:1. (9)

Again, Katkam et al., study was conducted on 212 SLE south Indian patients (194 females and 18 males) and female to male ratio was about 11:1. (10)

Regarding clinical data of SLE patients, forty-eight patients (98.0%) had photosensitivity, Arthritis was found in 46 patients (92%), forty-five patients (90%) had malar rash, alopecia was found in 45 patients (90%), 32 patients (64%) had oral ulcers, fever was found in 29 patients (58%) and serositis in the form of pleurisy, pericarditis and/or pericardial effusion was found in 14 patients (28%).

The number of patients with renal affection was 13 patients (26%), 8 patients (16%) had discoid

rash and hematologic disorders in the form of leucopenia and/or thrombocytopenia were found in 3 patients (6%).

In a 2014 investigation conducted by Skare et al., the clinical presentations of patients with SLE exhibited considerable variability, underscoring the multifaceted nature of the disease. The most frequently observed symptoms were photosensitivity (67.1%), arthritis (60.1%), oral ulcers (43.3%), and malar rash, also known as butterfly rash (39.8%). Additional significant findings encompassed glomerulonephritis (36.3%), leukopenia (32.1%), and a lower frequency of pericarditis (9.0%), hemolytic anemia (8.3%), seizures (8.3%), discoid lesions (6.9%), pleuritis (5.5%), thrombocytopenia (6.2%), and psychosis (3.4%). These findings underscore the diverse range of clinical manifestations seen in SLE, emphasizing the difficulties encountered in both diagnosis and treatment.

Oke et al., 2017 study which conducted on a group of SLE patients photosensitivity in 69%, malar rash was found in 52%, discoid rash in 20%, oral ulceration in 34%, arthritis in 84%, pleuritis in 34%, pericarditis in 17.5%, nephritis in 42%, leukopenia in 49%, lymphopenia in 50%, thrombocytopenia in 20%, hemolytic anemia in 5%, and neuropsychiatric lupus in 11.5% of patients included in that study.

By comparing our results with results of other studies, it can be seen that there exists some variation of the frequencies of different organs involvement in patients with SLE depending on an association of environmental together with genetic factors, in addition to different patients' characteristics and demographics.

All patients included in our study were on prednisolone and hydroxychloroquine. Steroid dose ranged from (5) to (60) milligram (mg) per day at the time of sample collection. Hydroxychloroquine dose ranged from 200 to 400 mg/day.

Immunosupressive was prescribed for 24 cases (48%) among the studied SLE patients.

Our case-control study demonstrated a highly statistically significant disparity in serum interleukin-23 (IL-23) concentrations between individuals with systemic lupus erythematosus (SLE) and healthy control subjects. The average serum IL-23 level among SLE patients was 82.1 ± 75 mg/dL, markedly higher than the 44.9 ± 10.6 mg/dL found in healthy controls ($P < 0.001$), implying a potential role of IL-23 to SLE pathogenesis. These results are consistent with prior research, including investigations by Wong et al., Du et al., Hegab et al., and Hassan et al., all of which documented considerably raised IL-23 levels in SLE people than controls (11, 12, 13,14).

Similarly, Fischer et al. informed notably increased serum IL-23 readings in SLE people ($P = 0.0015$) (9).

In contrast, Mok et al. observed no substantial variance in serum IL-23 levels between Asian SLE persons and healthy individuals ($P = 0.87$), suggesting possible ethnic or regional differences in IL-23 expression in SLE (15).

The discrepancy observed may be attributed to variations in racial backgrounds, extended disease duration, and reduced disease activity including the participants in the findings conducted by Mok et al.

The current study didn't reveal a substantial variation in the mean concentration of interleukin 23 related to the clinical manifestations, immunosuppressive use, investigations or disease activity and chronicity indices (P-value>0.05).

Yanti et al., study results partially agreed with ours. In that search, the level of IL-23 was greater in SLE persons than in controls, but no correlation was found involving the cytokine level and disease activity. (16)

Genc Cetin et al., study also couldn't detect relationship between SLEDAI score and IL-23 ($p=0.476$). (17)

Mok et al. concluded in their research that IL-23 levels (30.3 vs. 31.6 pg/ml, $P=0.11$) did not substantially vary concerning active and inactive people. Additionally, no association was acquired relating to SLEDAI scores and serum IL-23 levels ($r= -0.10$, $P=0.42$) (15). They suggested that this absence of correlation might be due to the acute IL-23 response typically being followed by a Th1 response, which could explain why IL-23 levels did not align with SLEDAI scores in their patient cohort.

In contrast to these findings, Hegab et al. (2014) reported a statistically significant positive

association relating serum IL-23 levels and SLE disease activity as determined by SLEDAI scores (r=0.912, p=0.001). Likewise, Vukelic et al. also observed a substantial positive association concerning SLEDAI scores and IL-23 levels in a study involving 56 SLE people (r=0.47, p<0.001) (8,13).

These conflicting results may stem from several factors, including differences in sample size, patient demographics, clinical characteristics, or treatment regimens. Furthermore, the use of qualitative data with high variability in SLE activity indices across studies may also contribute to these discrepancies.

Conclusion

This study identified elevated levels of IL-23 in the serum of SLE patients compared to the control group, suggesting a potential role for IL-23 in the pathogenesis of SLE. Notably, therapies targeting IL-23 may represent a promising approach for managing SLE. Nevertheless, additional research involving larger sample sizes is required to confirm these findings.

Ethical approval and consent to participate:

The Ethics Committee of the Faculty of Medicine at Fayoum University grante ethical approval for this study under reference number 95, on June 12, 2022.

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