International Journal Multidisciplinary
Homepage : https://journal.antispublisher.com/index.php/IJMI
Email : admin@antispublisher.com

e-ISSN : 3031-9870
IJMI, Vol. 3, No. 4, November 2026
Page 33-46

Evaluation of Hepatic and Renal Function Alterations Alongside Hematological Indices in Systemic Lupus Erythematosus

Saif A. Hussain[1], Khwam R. Hussein[2], Wissam R. Nayef[3]
[1]Department of Medical Laboratory Techniques, Basrah Health and Medical Technical College, Southern Technical University, Iraq
[2,3]Al-Nasiriyah Technical Institute, Southern Technical University, Iraq

DOI: https://doi.org/10.61796/ijmi.v3i4.622

Sections Info
Article history:
Submitted: July 28, 2026
Final Revised: August 15, 2026
Accepted: August 30, 2026
Published: September 21, 2026
Keywords:
Systemic lupus erythematosus
Hematological indices
Hepatic function
Renal function
Inflammatory markers

ABSTRACT

Objective: *Systemic lupus erythematosus (SLE) is a common chronic autoimmune disease that can affect multiple organ systems plus autoantibody production and chronic inflammation. This study objective is to monitor changes in hepatic and renal function tests, hematologic alterations, and serum levels of autoimmune and inflammatory markers among Iraqi SLE patients.* ***Method***: *A case-control study performed from November 2025 to April 2026 at Al-Karama Hospital and Al-Zahraa Hospital, Wasit Governorate, Iraq. Ninety subjects were included, 45 patients with SLE and 45 age- and sex-matched apparently healthy controls. Hematological parameters (WBC, Hb, PLT) were evaluated as well hepatic markers (ALT, AST), renal markers (blood urea, serum creatinine), autoimmune markers (ANA, anti-dsDNA) and inflammatory markers (CRP, ESR). All statistical analyses were conducted using IBM SPSS version 26.* ***Results***: *Patients with SLE had significantly lower median hemoglobin (10.25 vs. 13.00 g/dL, $P < 0.001$) and platelet counts (240.00 vs. 288.00 × 10⁹/L, $P = 0.001$) than controls, while WBC counts showed no significant difference ($P = 0.119$). ALT, AST, blood urea, and serum creatinine levels were significantly higher in SLE patients compared with controls ($P < 0.001$ for all). Autoimmune (ANA, anti-dsDNA) and inflammatory markers (CRP, ESR) were also significantly elevated in patients ($P < 0.001$). No statistically significant differences were observed across age subgroups within the patient group.* ***Novelty***: *SLE is associated with significant hematological alterations (anemia and reduced platelets), as well as marked hepatic and renal function impairments, alongside elevated autoimmune and inflammatory activity. Simultaneous monitoring of these markers provides essential clinical information for evaluating organ involvement and disease progression.*

INTRODUCTION

Systemic lupus erythematosus (SLE) is a long-lasting, multisystem autoimmune illness that are marked via immunological tolerance loss, autoantibody generation, and persistent inflammation. It can affect various organs, including the hematological system and kidneys. The clinical signs of SLE are quite diverse, ranging from modest generalized complaints to severe organ involvement. As a result, laboratory evaluation is critical for disease assessment, monitoring, and identifying organ involvement [1, 2].

Hematological abnormalities are a frequent symptom of SLE and can arise at any stage of the illness. Patients with SLE typically report anemia, leukopenia, lymphopenia, and thrombocytopenia, which can be caused by immune-mediated death of blood cells, chronic inflammation, bone marrow involvement, or treatment-related causes [3, 4]. These anomalies may also indicate disease activity and contribute to morbidity in patients. As a result, frequent measurement of hematological parameters, notably

hemoglobin (Hb), white blood cell (WBC), and platelet (PLT) counts, offers essential information for the clinical evaluation of SLE [3, 4].

Renal involvement is one of the most serious consequences of SLE and is often known as lupus nephritis [5]. Kidney involvement might vary from minor urine issues to clinically substantial renal failure and progressive kidney disease. Because renal involvement can have a significant impact on long-term morbidity and mortality, early diagnosis and regular monitoring of renal function are critical in individuals with SLE [6, 7]. Biochemical markers such as blood urea and serum creatinine are still commonly utilized laboratory indicators for assessing renal function and recognizing potential renal impairment [6, 7].

Hepatic abnormalities can also be seen in SLE patients. Abnormal liver biochemical tests may be tied to systemic inflammation, autoimmune processes, concurrent illnesses, or medication-related consequences. Elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels may give significant information when evaluating individuals with SLE, particularly when evaluated combined with clinical symptoms and other laboratory studies [8].

Hematological, hepatic, and renal markers provide a more holistic view to evaluate the systemic impact of SLE. The addition of biochemical indicators such as ALT, AST, blood urea, and serum creatinine to the hematological indices may assist in identifying disorders that involve multiple organ systems. Additionally, autoimmune and inflammatory markers (e.g., antinuclear antibodies [ANA], anti–double-stranded DNA [anti-dsDNA], C-reactive protein, and erythrocyte sedimentation rate) provide further information related to the role of immunological and inflammatory characteristics attributable to the disease [1, 6].

SLE is considered a disease of multiple systems, but the nature and degree of hematologic and reparative derangements may vary in different cadre. Studies specific to populations are thus of interest in characterizing laboratory abnormalities among SLE patients as well as for assessing potential clinically meaningful differences when compared with health individuals. Thus, the present study was conducted to assess hematological indices and hepatic and renal function indicators in patients suffering from SLE compared with healthy controls.

RESEARCH METHOD

Study Design and Period

This case-control research was undertaken between November 2025 and April 2026 at Al-Karama Hospital and Al-Zahraa Hospital, Iraq, to investigate hepatic and renal function alterations along with hematological indicators in SLE.

Population source

The study included 90 people, 45 of whom were diagnosed with SLE using recognized clinical diagnostic criteria and 45 who seemed to be healthy and were age and gender matched. The study excluded participants with other autoimmune illnesses,

chronic inflammatory disorders, severe infections, advanced liver disease, chronic renal failure, pregnancy, breastfeeding, or damaged DNA samples.

Data Collection Method

Blood Sample Collection

We aseptically collected around 5 mL of peripheral venous blood from each participant. We transferred 2 mL to EDTA tubes for hematological investigation. The leftover blood was collected in gel tubes, allowed to coagulate, then centrifuged at 3000 rpm for 15 minutes. The separated serum was kept at -20°C until biochemical and immunological analyzes.

Measurement of Hematological Indices

An automated hematology analyzer was used to measure hematological parameters such as WBC count, Hb levels, and PLT count in EDTA-anticoagulated whole blood samples.

Measurement of Liver and Kidney Function Markers

To measure hepatic and renal function, serum samples were examined for ALT, AST, blood urea, and serum creatinine using standard automated clinical chemistry analyzers according to the manufacturer's guidelines.

Measurement of Autoimmune and Inflammatory Markers

Serum ANA and anti-dsDNA antibodies were tested using commercially available ELISA kits, following the manufacturer's instructions. The latex agglutination method was used to assess serum CRP, whilst the usual Westergren method was used to evaluate ESR.

Statistical Analysis

Statistical analyzes were carried out using IBM SPSS Statistics version 26. Continuous variables were reported as mean ± SD or median (interquartile range) based on data distribution. We compared groups using the independent samples t-test or the Mann-Whitney U test. Categorical variables were evaluated using the Chi-squared test or Fisher's exact test, as applicable. A p-value of <0.05 was judged statistically significant [9].

Ethical Considerations

Ethical approval for this study was obtained from “the Basrah Health and Medical Technical College” in Basra and the “Wasit Health Directorate” (Human Development and Training Center) before data collection. After obtaining the necessary administrative clearances, the study was conducted at Wasit governorate hospitals.

Each participant will be interviewed after ensuring their consent to participate in the study, explaining the study's objectives, ensuring that the collected information will be used exclusively for the study, and informing them of its confidentiality.

RESULTS AND DISCUSSION

Results

Socio-demographic Characteristics

The socio-demographic characteristics of the 45 patients and 45 healthy controls are summarized in Table 1. The mean age of the patient group was 32.05 ± 4.72 years and that of the control patients was 34.04 ± 5.20 years, which indicates no statistically significant difference between both groups (P = 0.06). Similarly, when categorizing participants by age groups, no statistically significant difference was noted (P = 0.067). The proportion of patients was most in the age group of 30–39 years (51.1%), followed by <30 years (42.2%).

Table 1. Socio-demographic characteristics among the study groups.

Variable	Category	Patients group (n=45)	Control group (n=45)	p-value
Age (years)	Overall (mean ± SD)	32.05 ± 4.72	34.04 ± 5.20	0.06
	<30 years	19 (42.2%)	10 (22.2%)	
	30-39 years	23 (51.1%)	27 (60.0%)	0.067
	≥40 years	3 (6.7%)	8 (17.8%)	
Sex	Male	3 (6.7%)	7 (15.6%)	0.180
	Female	42 (93.3%)	38 (84.4%)	

Haematological Parameters

Table 2 presents the comparison of hematological parameters between patients with SLE and healthy controls. The median Hb level was significantly lower in SLE patients (10.25 g/dL) than in the control group (13.00 g/dL, P < 0.001). Likewise, median PLT counts were significantly reduced in patients compared with controls (240.00 vs. 288.00, P = 0.001). In contrast, no statistically significant difference was observed in median WBC counts between the two groups (6500.00 vs. 6640.00, P = 0.119).

Table 2. Hematological marker levels between patients and the control group.

Variable	Patients Median (IQR)	Control Median (IQR)	p-value
WBC	6500.00 (5275.00–7800.00)	6640.00 (6040.00–8290.00)	0.119
Hb	10.25 (10.00–11.05)	13.00 (12.00–13.00)	<0.001
PLT	240.00 (167.75–280.00)	288.00 (238.00–356.00)	0.001

Liver and Kidney Function Tests

Table 3 presents the comparison of liver and kidney function markers between patients with SLE and healthy controls. There is a highly significant statistical difference (P < 0.001) across all Liver and kidney function marker variables between the two study groups. The median serum levels of ALT and AST were significantly higher in SLE patients (31.00 and 29.00, respectively) than in the control group (14.00 and 16.00, P <

0.001 for both). Similarly, median blood urea and serum creatinine levels were significantly elevated in patients (40.00 and 1.25, respectively) compared with those in healthy controls (22.00 and 0.80; P < 0.001 for both).

Table 3. Liver and kidney function marker levels between patients and the control group.

Variable	Patients Median (IQR)	Control Median (IQR)	p-value
ALT	31.00 (29.00–32.00)	14.00 (12.00–18.00)	<0.001
AST	29.00 (26.00–31.00)	16.00 (11.00–18.00)	<0.001
Blood urea	40.00 (37.75–42.00)	22.00 (18.00–26.00)	<0.001
Creatinine	1.25 (0.88–1.62)	0.80 (0.70–1.10)	<0.001

Autoimmune and Inflammatory Markers

Table 4 compares autoimmune and inflammatory markers between patients with SLE and healthy controls. The analysis revealed a highly significant statistical difference (P < 0.001) in all autoimmune and inflammatory variables between the two groups. Specifically, the median levels of ANA and anti-dsDNA were considerably higher in SLE patients, with values of 34.00 and 150.00, respectively, compared to the control group, which had median levels of 2.00 and 5.00, respectively. Furthermore, the median levels of inflammatory markers, such as CRP and ESR, were significantly elevated in patients, with values of 13.00 and 35.00, respectively, while healthy controls showed median levels of 3.00 and 5.00, respectively.

Table 4. Autoimmune and inflammatory marker levels between patients and the control group.

Variable	Patients Median (IQR)	Control Median (IQR)	p-value
ANA	34.00 (22.75–81.75)	2.00 (1.00–3.00)	<0.001
anti-dsDNA	150.00 (65.00–242.25)	5.00 (4.00–7.00)	<0.001
CRP	13.00 (10.00–17.00)	3.00 (2.00–5.00)	<0.001
ESR	35.00 (27.75–48.25)	5.00 (3.00–7.00)	<0.001

Distribution of Biomarkers According to Age

Table 5 demonstrated the levels of hematological markers among patients with SLE, categorized by their respective age groups (<30 years, 30–39 years, and ≥40 years). The analysis indicates that there are no statistically significant differences in any of the measured hematological parameters across the different age categories. Specifically, the median WBC counts were 6300.00 for patients under 30 years, 6600.00 for those aged 30–39 years, and 5200.00 for patients aged 40 years and older, showing no significant variation (P = 0.233). Similarly, median Hb levels showed a slight apparent increase with age, recording 10.00, 10.50, and 12.50 across the three age groups, respectively, but this trend was not statistically significant (P = 0.119). Finally, median platelet (PLT) counts

fluctuated across the age groups, recording 245.00 in the <30 years group, 220.00 in the 30–39 years group, and 265.00 in the ≥40 years group, which again demonstrated no significant difference (P = 0.386).

Table 5. Hematological marker levels in the patient group according to age categories.

Marker	<30 years Median (IQR)	30–39 years Median (IQR)	≥40 years Median (IQR)	p-value
WBC	6300.00 (4750.00–7000.00)	6600.00 (5550.00–7900.00)	5200.00 (5150.00–5250.00)	0.233
Hb	10.00 (9.50–11.10)	10.50 (10.00–11.00)	12.50 (12.25–12.75)	0.119
PLT	245.00 (189.00–285.00)	220.00 (163.00–277.50)	265.00 (252.50–277.50)	0.386

Table 6 details the levels of liver and kidney function markers among patients with SLE, categorized by their respective age groups (<30 years, 30–39 years, and ≥40 years). The analysis indicates that there are no statistically significant differences in any of the measured liver or renal parameters across the different age categories. Specifically, the median ALT levels remained remarkably stable across the groups, with values of 31.00 in both the <30 years and 30–39 years groups, and 30.50 in the ≥40 years group (P = 0.974). Similarly, median AST levels showed minimal variation, with values of 28.00 for patients under 30 years, 29.00 for those aged 30–39 years, and 28.00 for patients aged 40 years and older (P = 0.282).

Regarding renal function, median blood urea levels were 41.00 in the <30 years group, 40.00 in the 30–39 years group, and 34.50 in the ≥40 years group, demonstrating no significant difference (P = 0.696). Finally, median serum creatinine levels were 1.20 in the <30 years group, 1.40 in the 30–39 years group, and 1.00 in the ≥40 years group; this fluctuation was also not statistically significant (P = 0.705).

Table 6. Liver and kidney function marker levels in the patient group according to age categories.

Marker	<30 years Median (IQR)	30–39 years Median (IQR)	≥40 years Median (IQR)	p-value
ALT	31.00 (29.00–32.50)	31.00 (29.00–32.00)	30.50 (29.75–31.25)	0.974
AST	28.00 (26.00–30.00)	29.00 (28.00–31.00)	28.00 (27.00–29.00)	0.282
Blood urea	41.00 (35.50–43.50)	40.00 (38.00–42.00)	34.50 (31.25–37.75)	0.696
Creatinine	1.20 (0.90–1.60)	1.40 (0.80–1.65)	1.00 (0.95–1.05)	0.705

Table 7 presents the levels of autoimmune and inflammatory markers among patients with SLE, categorized according to age groups (<30 years, 30–39 years, and ≥40 years). The analysis demonstrates that there were no statistically significant differences in any of the measured autoimmune or inflammatory markers across the different age categories. Specifically, the median antinuclear antibody (ANA) levels were 32.00 in both the <30 years and 30–39 years groups, while a higher median value of 96.00 was observed in the ≥40 years group; however, this difference was not statistically significant (P = 0.502). Likewise, median anti-dsDNA levels were 150.00 in the <30 years group, 146.00 in the 30–39 years group, and 307.50 in the ≥40 years group, with no significant variation among the age groups (P = 0.833).

Regarding the inflammatory markers, median CRP levels were 12.00 in patients aged <30 years, 13.00 in those aged 30–39 years, and 15.00 in patients aged ≥40 years, showing no statistically significant difference (P = 0.470). Similarly, median ESR values were 35.00, 35.00, and 41.00 for the <30 years, 30–39 years, and ≥40 years groups, respectively, with no significant difference observed (P = 0.166).

Table 7. Autoimmune and inflammatory marker levels in the patient group according to age categories.

Marker	<30 years) Median (IQR)	30–39 years Median (IQR)	≥40 years Median (IQR)	p-value
ANA	32.00 (23.00–46.50)	32.00 (18.00–85.50)	96.00 (70.00–122.00)	0.502
anti-dsDNA	150.00 (76.50–211.50)	146.00 (53.50–260.00)	307.50 (183.75–431.25)	0.833
CRP	12.00 (10.00–14.00)	13.00 (10.00–17.00)	15.00 (15.00–15.00)	0.470
ESR	35.00 (26.50–38.00)	35.00 (28.50–66.50)	41.00 (40.50–41.50)	0.166

Discussion

Socio-demographic characteristics among the study groups

The present study included 45 patients with SLE and 45 apparently healthy controls. The mean age of the patients was 32.05 ± 4.72 years, compared with 34.04 ± 5.20 years among controls. This difference was not statistically significant (p = 0.060), indicating that the two groups were generally comparable in terms of mean age. Also, the distribution across age categories did not differ significantly between the groups (p = 0.067). Patients younger than 30 years represented 42.2% of the SLE group compared with 22.2% of controls, whereas participants aged 40 years or older constituted only 6.7% of patients compared with 17.8% of controls. Most participants in both groups were aged 30–39 years. The distribution of SLE cases by age was similar to the well-described epidemiological distribution of SLE. SLE often develops in young adults and during the reproductive years, but can happen at any age. Hormonal, genetic and immunological mechanisms have been suggested to play a role in the age distribution of the onset of SLE,

with large epidemiological studies indicating increased incidence after puberty and highest in women of reproductive age [9, 10]. The higher proportion of patients younger than 30 years in the current study therefore supports previous observations that SLE commonly begins during young adulthood. Females constituted 93.3% of the patient group and 84.4% of the control group, while males represented 6.7% and 15.6%, respectively.

Despite the clear numerical predominance of females among patients, the difference in sex distribution between the groups was not statistically significant ($p = 0.18$). This lack of significance may be related to the relatively small number of male participants rather than an absence of the well-established female predominance of SLE. The marked female predominance agrees with previous studies showing that women account for approximately 85–90% of SLE cases, particularly during reproductive age. Several mechanisms have been proposed, including the immunostimulatory effects of oestrogen, reduced immunosuppressive effects of androgens, incomplete X-chromosome inactivation, increased expression of X-linked immune genes, and sex-related differences in B-cell and type I interferon responses [9, 11]. Thus, although sex distribution was not statistically different between the study groups, the high proportion of female patients remains biologically and epidemiologically consistent with the known characteristics of SLE. The similarity in mean age and the absence of a statistically significant difference in sex distribution indicate reasonable demographic comparability between patients and controls. Nevertheless, the significant difference in categorical age distribution should be considered when interpreting the biochemical and immunological findings. The very small number of patients aged 40 years or older may also reduce the stability of the categorical analysis. Adjustment for age and sex in multivariable analyses would therefore strengthen the conclusion that the observed biomarker differences are independently associated with SLE.

Hematological marker levels between patients and the control group

Haemoglobin was significantly lower among patients, with a median of 10.25 compared with 13.00 among controls. Platelet count was also significantly lower, with medians of 240 and 288, respectively. In contrast, total WBC count did not differ significantly between the groups. Anaemia is one of the most frequent haematological manifestations of SLE and can result from several overlapping mechanisms. These include anaemia of chronic inflammation, iron deficiency, autoimmune haemolysis, chronic kidney disease, nutritional deficiency, medication-related bone-marrow suppression, and blood loss. Bertoli et al. demonstrated that anaemia severity was independently associated with SLE activity and that moderate or severe anaemia predicted greater damage accumulation over time [13]. The median haemoglobin concentration in the patient group suggests clinically relevant anaemia rather than a minor statistical difference. Chen et al. found that more than half of their SLE cohort had anaemia, with anaemia of chronic disease being the most frequent type, followed by autoimmune haemolysis and impaired haematopoiesis [14]. Increased inflammatory cytokines may reduce erythropoietin responsiveness, increase hepcidin production,

restrict iron availability, and shorten erythrocyte survival. The present study did not include red-cell indices, ferritin, transferrin saturation, reticulocyte count, lactate dehydrogenase, bilirubin, haptoglobin, or direct antiglobulin testing. Therefore, the exact cause of the lower haemoglobin cannot be determined. These measurements would help distinguish iron deficiency, inflammatory anaemia, haemolysis, renal anaemia, and medication-related suppression. Although PLT counts remained within the conventional reference range for many patients, the significantly lower median indicates a general downward shift in PLT distribution. This distinction is important because the finding should not be described as thrombocytopenia in all patients. Instead, it may indicate subclinical PLT consumption, increased immune destruction, altered production, or treatment-related effects [15]. Zhao et al. found that thrombocytopenic SLE patients had greater disease activity, more renal involvement, greater organ damage, and higher mortality [16]. Abdel Galil et al. similarly reported an inverse correlation between PLT count and SLEDAI scores [17]. Hanata et al. found that the immature PLT fraction correlated with disease activity and provided information regarding marrow PLT production and response to corticosteroid treatment [18]. PLT reduction in SLE can result from antiPLT antibodies, antiphospholipid-associated consumption, thrombotic microangiopathy, hypersplenism, infection, bone-marrow suppression, or immunosuppressive treatment. Because the present patients had a lower PLT count without severe group-level thrombocytopenia, additional analysis should examine the proportion with PLT counts below 150×10^9/L and below 100×10^9/L. The lack of a significant difference in total WBC count does not exclude SLE-related leucocyte abnormalities. Total WBC may remain within the normal range despite lymphopenia or neutropenia. Differential leukocyte counts would provide more useful information because lymphopenia has greater relevance to SLE classification and disease activity than total WBC alone.

Liver and kidney function marker levels between patients and the control group

ALT and AST were significantly higher in patients than controls. Median ALT was 31 compared with 14, while median AST was 29 compared with 16; both differences were significant at $p < 0.001$. These findings suggest increased hepatocellular stress or hepatic involvement. Nevertheless, the median values may remain within the upper reference range used by some laboratories. The results therefore indicate statistically significant differences but do not necessarily demonstrate severe clinical hepatitis. Piga et al. found persistent liver-enzyme abnormalities in approximately 19% of patients with SLE, while lupus hepatitis accounted for a smaller proportion of these abnormalities. The clinical course was usually mild, subclinical, and fluctuating [19]. Her et al. similarly reported that elevated liver enzymes in SLE may result from medications, herbal preparations, infection, or active systemic disease rather than primary lupus hepatitis alone [20]. Alternative explanations should therefore be considered, including corticosteroid- or immunosuppressant-associated fatty liver disease, viral hepatitis, autoimmune hepatitis, muscle inflammation, medication toxicity, and non-alcoholic fatty liver disease. Measurement of bilirubin, alkaline phosphatase, gamma-glutamyl transferase, albumin,

prothrombin time, creatine kinase, and viral hepatitis markers would improve interpretation. Blood urea and creatinine were significantly higher among patients. Median urea was 40 compared with 22, while creatinine was 1.25 compared with 0.80. Both differences were highly significant. These findings can reflect decreased renal filtration, renal haemodynamic changes, increased protein catabolism, dehydration, treatment effects or renal involvement from lupus. In SLE, Yang et al. showed associations between serum urea and creatinine and various clinical and laboratory characteristics [21]. The disease duration, disease activity, hypertension and renal manifestations were associated with creatinine. This increase in creatinine, urea, anti-dsDNA, oxidative markers, ESR and CRP can be worrisome for renal involvement. But a diagnosis of lupus nephritis cannot be made based solely on serum urea and creatinine. An elevated serum creatinine may represent damage to the kidneys rather than actual inflammation, and normal may be normal at this point but rise as the disease progresses. This work by Julkunen et al. demonstrated that interpretation of renal involvement in SLE should involve more than measuring only serum creatinine, but should be combined with additional laboratory parameters such as complement levels, urinary protein excretion, urinary microscopy, blood pressure, serum albumin and clinical parameters [22].

Autoimmune and inflammatory marker levels between patients and the control group

ANA and anti-dsDNA concentrations were markedly higher in patients than controls. Median ANA was 34 compared with 2, while anti-dsDNA was 150 compared with 5. Both comparisons were highly significant. The pronounced ANA elevation is consistent with its high sensitivity for SLE. Leuchten et al., in a systematic review involving more than 13,000 SLE patients, found that approximately 96% were ANA positive and showed that a titre of 1:80 provided sufficiently high sensitivity for use as an entry criterion [23]. Consequently, the 2019 EULAR/ACR classification criteria require a positive ANA result on at least one occasion before other weighted clinical and immunological criteria are considered [24]. ANA is sensitive but not highly specific. It can occur in other autoimmune disorders, infections, malignancies, medication exposure, and healthy individuals. Li et al. showed that diagnostic specificity increased with higher ANA titres and the presence of multiple specific autoantibodies [25]. Therefore, the large difference between the groups supports the diagnosis but should be interpreted together with clinical findings and specific antibodies. Anti-dsDNA is more specific for SLE and may be associated with immunological activity and lupus nephritis, although its clinical performance depends on the laboratory method. The strong elevation in the present study is therefore biologically and clinically important. Nevertheless, serial anti-dsDNA measurements are generally more informative than a single measurement when evaluating relationships with disease activity. The correlation between ANA and anti-dsDNA shown in the heatmap supports coordinated activation of autoreactive B-cell responses. However, ANA usually remains positive irrespective of disease activity, whereas anti-dsDNA may show greater fluctuation in selected patients. Complement C3 and C4 should be added in future studies to provide a more complete evaluation of

immune-complex activity. Median CRP was 13 among patients compared with 3 among controls, while ESR was 35 compared with 5. Both differences were highly significant. The increased ESR is consistent with systemic inflammatory and immunological activity. Vilá et al.[26], in a large multiethnic longitudinal cohort, found that increasing ESR categories were independently associated with greater disease activity. Moderate and marked ESR elevations were also associated with subsequent damage accumulation. ESR is affected by immunoglobulin levels, fibrinogen, anaemia, age, sex, and erythrocyte characteristics. The lower haemoglobin observed in this study may have contributed partly to the increased ESR. Nevertheless, the substantial difference between patients and controls, together with the increased CRP and autoantibodies, supports genuine inflammatory activation [27]. CRP interpretation in SLE is more complicated. Some patients with active SLE demonstrate only limited CRP elevation because type I interferon activity may suppress hepatic CRP production. Barnes et al. found that high-sensitivity CRP was not consistently associated with global disease activity but was related to conventional cardiovascular risk factors [28]. Rezaieyazdi et al. similarly found higher CRP among SLE patients than controls but no strong association with overall SLE Disease Activity Index (SLEDAI) scores [29]. In contrast, Mok et al. found that high-sensitivity CRP was associated with clinical activity, particularly serositis, musculoskeletal manifestations, and haematological disease [30]. These apparently conflicting results suggest that CRP may reflect particular manifestations rather than global lupus activity in every patient. CRP can also rise substantially during bacterial infection. Schäfer et al. showed that both ESR and CRP may be elevated during SLE flare, infection, or their coexistence. Thus, the increased CRP in the present study indicates inflammation but cannot independently distinguish lupus activity from infection [31]. Clinical assessment, cultures, procalcitonin, complement, anti-dsDNA trends, and the CRP/ESR ratio may help clarify this distinction.

Biomarkers according to age categories

None of the evaluated markers differed significantly among the three patient age categories. This may suggest that oxidative, haematological, biochemical, and immune abnormalities occurred across the age range represented in the study. However, the absence of statistical significance should be interpreted cautiously because only three patients were aged 40 years or older. This subgroup was too small to provide stable estimates or adequate statistical power. The older category also showed numerically higher ANA, anti-dsDNA, CRP, ESR, haemoglobin, and TAC values in some comparisons, but conclusions cannot be drawn from two participants. Age should preferably be analysed as a continuous variable using Spearman correlation or regression rather than divided into highly unequal categories.

CONCLUSION

Fundamental Finding : Systemic lupus erythematosus (SLE) is characterized by significant oxidative imbalance with increased autoimmune and inflammatory activity. Patients showed alterations in hepatic and renal functions, along with a reduced Hb level

and PLT count. **Implication :** Such correlated biomarkers are suggestive of complicated disease interactions. **Limitation :** The issue of the CTLA-4 genetic capture is still to clarify. **Future Research :** The issue of the CTLA-4 genetic capture requires further investigations.

ACKNOWLEDGEMENTS

We would like to thank the Wasit Governorate, particularly the Al-Karama Hospital and Al-Zahraa Hospital, through which the data were collected, and all participants in this study.

REFERENCES

[1] C. H. Siegel and L. R. Sammaritano, "Systemic lupus erythematosus: a review," *Jama*, vol. 331, no. 17, pp. 1480–1491, 2024.

[2] A. Fanouriakis, N. Tziolos, G. Bertsias, and D. T. Boumpas, "Update on the diagnosis and management of systemic lupus erythematosus," *Ann. Rheum. Dis.*, vol. 80, no. 1, pp. 14–25, 2021, doi: https://doi.org/10.1136/annrheumdis-2020-218272.

[3] A. Al-Sheef *et al.*, "Hematologic Manifestations in Systemic Lupus Erythematosus: A Systematic Review of Clinical Patterns, Prognostic Implications, and Epidemiology," *Pharm. Pract. (Granada).*, vol. 23, no. 4, pp. 1–10, 2025.

[4] Z. Yang, Y. Wu, S. Huang, J. Bao, L. Xu, and Y. Fan, "Risk factors associated with thrombocytopenia in systemic lupus erythematosus: a systematic review and meta-analysis," *Autoimmun. Rev.*, vol. 24, no. 2, p. 103721, 2025.

[5] H.-J. Anders, R. Saxena, M. Zhao, I. Parodis, J. E. Salmon, and C. Mohan, "Lupus nephritis," *Nat. Rev. Dis. Prim.*, vol. 6, no. 1, p. 7, 2020, doi: 10.1038/s41572-019-0141-9.

[6] F. K. Alduraibi and G. C. Tsokos, "Lupus nephritis biomarkers: a critical review," *Int. J. Mol. Sci.*, vol. 25, no. 2, p. 805, 2024.

[7] E. Xagas, K. Drouzas, G. Liapis, and S. Lionaki, "Evidence based treatment for lupus nephritis: present perspectives and challenges," *Front. Nephrol.*, vol. 4, p. 1417026, 2024.

[8] L. Zhou, S. Z. Cai, and L. L. Dong, "Recent advances in pathogenesis, diagnosis, and therapeutic approaches for digestive system involvement in systemic lupus erythematosus," *J. Dig. Dis.*, vol. 25, no. 7, pp. 410–423, 2024.

[9] A. S. N. Al-Aboudi, J. T. Ahmed, and W. A.-A. Ali Farid, "Factors Influencing on the Consumption of SugarSweetened Beverages among Adults in Thi-Qar Hospitals, Iraq," *Int. J. Nutr. Sci.*, vol. 11, no. 2, pp. 312–320, 2026.

[10] J. S. Nusbaum *et al.*, "Sex differences in systemic lupus erythematosus: epidemiology, clinical considerations, and disease pathogenesis," in *Mayo Clinic Proceedings*, Elsevier, 2020, pp. 384–394.

[11] M. R. W. Barber *et al.*, "Global epidemiology of systemic lupus erythematosus," *Nat. Rev. Rheumatol.*, vol. 17, no. 9, pp. 515–532, 2021, doi: 10.1038/s41584-021-00668-1.

[12] A. A. Vieira, I. Almada-Correia, J. Inácio, P. Costa-Reis, and S. T. Da Rocha, "Female-bias in systemic lupus erythematosus: How much is the X chromosome to blame?," *Biol. Sex Differ.*, vol. 15, no. 1, p. 76, 2024.

[13] A. M. Bertoli *et al.*, "Systemic lupus erythematosus in a multiethnic US cohort LUMINA LI: Anaemia as a predictor of disease activity and damage accrual," *Rheumatology*, vol. 46, no. 9, pp. 1471–1476, Sep. 2007, doi: 10.1093/rheumatology/kem153.

[14] J. L. Chen *et al.*, "Hematological abnormalities in systemic lupus erythematosus and clinical

significance thereof: comparative analysis of 236 cases," *Zhonghua Yi Xue Za Zhi*, vol. 87, no. 19, pp. 1330–1333, 2007.

[15] A. Mititelu, M.-C. Onisâi, A. Roșca, and A. M. Vlădăreanu, "Current understanding of immune thrombocytopenia: a review of pathogenesis and treatment options," *Int. J. Mol. Sci.*, vol. 25, no. 4, p. 2163, 2024.

[16] H. Zhao, S. Li, and R. Yang, "Thrombocytopenia in patients with systemic lupus erythematosus: significant in the clinical implication and prognosis," *Platelets*, vol. 21, no. 5, pp. 380–385, 2010.

[17] S. M. Abdel Galil, A. M. Edrees, A. K. Ajeeb, G. S. Aldoobi, M. El-Boshy, and W. Hussain, "Prognostic significance of platelet count in SLE patients," *Platelets*, vol. 28, no. 2, pp. 203–207, 2017.

[18] N. Hanata *et al.*, "Immature platelet levels correlate with disease activity and predict treatment response of thrombocytopenia in lupus patients," *Lupus*, vol. 30, no. 11, pp. 1764–1772, 2021.

[19] M. Piga, A. Vacca, G. Porru, A. Cauli, and A. Mathieu, "Liver involvement in systemic lupus erythematosus: incidence, clinical course and outcome of lupus hepatitis," *Clin. Exp. Rheumatol.*, vol. 28, no. 4, p. 504, 2010.

[20] M. Her, Y. Lee, E. Jung, T. Kim, and D. Kim, "Liver enzyme abnormalities in systemic lupus erythematosus: a focus on toxic hepatitis," *Rheumatol. Int.*, vol. 31, no. 1, pp. 79–84, 2011.

[21] Z. Yang, Y. Liang, C. Li, W. Xi, and R. Zhong, "Associations of serum urea, creatinine and uric acid with clinical and laboratory features in patients with systemic lupus erythematosus," *Rheumatol. Int.*, vol. 32, no. 9, pp. 2715–2723, 2012.

[22] H. Julkunen, S. Ekblom-Kullberg, and A. Miettinen, "Nonrenal and renal activity of systemic lupus erythematosus: a comparison of two anti-C1q and five anti-dsDNA assays and complement C3 and C4," *Rheumatol. Int.*, vol. 32, no. 8, pp. 2445–2451, 2012.

[23] N. Leuchten *et al.*, "Performance of Antinuclear Antibodies for Classifying Systemic Lupus Erythematosus: A Systematic Literature Review and Meta-Regression of Diagnostic Data," *Arthritis Care \& Res.*, vol. 70, no. 3, pp. 428–438, 2018, doi: https://doi.org/10.1002/acr.23292.

[24] J. Sota *et al.*, "Anakinra drug retention rate and predictive factors of long-term response in systemic juvenile idiopathic arthritis and adult onset still disease," *Front. Pharmacol.*, vol. 10, p. 918, 2019.

[25] H. Li, Y. Zheng, L. Chen, and S. Lin, "High titers of antinuclear antibody and the presence of multiple autoantibodies are highly suggestive of systemic lupus erythematosus.," *Sci. Rep.*, vol. 12, no. 1, p. 1687, Feb. 2022, doi: 10.1038/s41598-022-05807-6.

[26] L. M. Vilá *et al.*, "Systemic lupus erythematosus in a multiethnic cohort (LUMINA): XXIX. Elevation of erythrocyte sedimentation rate is associated with disease activity and damage accrual.," *J. Rheumatol.*, vol. 32, no. 11, pp. 2150 LP – 2155, Nov. 2005, [Online]. Available: http://www.jrheum.org/content/32/11/2150.abstract

[27] V. Alende-Castro *et al.*, "Factors influencing erythrocyte sedimentation rate in adults: new evidence for an old test," *Medicine (Baltimore).*, vol. 98, no. 34, p. e16816, 2019.

[28] E. V Barnes *et al.*, "High sensitivity C-reactive protein in systemic lupus erythematosus: relation to disease activity, clinical presentation and implications for cardiovascular risk," *Lupus*, vol. 14, no. 8, pp. 576–582, 2005.

[29] Z. Rezaieyazdi *et al.*, "Is there any correlation between high sensitive CRP and disease activity in systemic lupus erythematosus?," *Lupus*, vol. 20, no. 14, pp. 1494–1500, 2011.

[30] C. C. Mok, D. J. Birmingham, L. Y. Ho, L. A. Hebert, and B. H. Rovin, "High-sensitivity C-reactive protein, disease activity, and cardiovascular risk factors in systemic lupus erythematosus," *Arthritis Care Res. (Hoboken).*, vol. 65, no. 3, pp. 441–447, 2013.

[31] V. S. Schäfer, K. Weiß, A. Krause, and W. A. Schmidt, "Does erythrocyte sedimentation rate reflect and discriminate flare from infection in systemic lupus erythematosus? Correlation with clinical and laboratory parameters of disease activity.," *Clin. Rheumatol.*, vol. 37, no. 7, pp. 1835–1844, Jul. 2018, doi: 10.1007/s10067-018-4093-3.

*__Saif A. Hussain (Corresponding Author)__
Department of Medical Laboratory Techniques, Basrah Health and Medical Technical College, Southern Technical University, Iraq
E-mail: saif.hussien.2024@fgs.stu.edu.iq

__Khwam R. Hussein__
Al-Nasiriyah Technical Institute, Southern Technical University, Iraq
E-mail: krhussein@stu.edu.iq

__Wissam R. Nayef__
Al-Nasiriyah Technical Institute, Southern Technical University, Iraq
E-mail: wesamsm@stu.edu.iq