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Examination of Several Immunological and Biochemical Parameters in Lupus Nephritis Patients

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Abstract: Background: Lupus nephritis with immune dysregulation and renal involvement is a severe, potentially life-threatening complication of systemic lupus erythematosus (SLE). Assessment of immune and serologic parameters is important in determining disease activity and monitoring renal injury. Objective: This study was conducted to evaluate some immunological and biochemical parameters in lupus nephritis (LN) patients and their correlation with disease activity. **Methods:** A cross-sectional study was conducted among patients diagnosed with LN and age-matched healthy controls. Blood and urine were obtained to assess different immunological parameters (antinuclear antibody, ANA; anti-dsDNA antibodies; complement levels: C3 and C4). Serum creatinine, blood urea, albumin and proteinuria were also measured by standard laboratory procedures. Comparison of patient groups and control groups was conducted through statistical analysis, and $P < 0.05$ was considered statistically significant. **Results:** A total of 110 participants were included in this study, with a mean age of 39.0±11.76 years. The majority of participants were females .(%99.1) The ANA test was positive in 33.7% of patients, while 66.3% were negative. Anti-dsDNA antibodies were positive in 10% of cases .The mean MYD88 level was 5.31±2.74, ranging from 2.01 to 13.64, indicating variability in immune marker expression among participants. *Lupus* nephritis patients had much higher amounts of immunological markers, in particular anti-dsDNA antibodies and lower complement (C3 and C4) levels than healthy controls ($p<0.05$). Biochemical analysis showed significantly higher serum creatinine and proteinuria, with decreased levels of serum albumin, which indicated impaired renal function. Conclusions: It is concluded that the combined evaluation of immune and biochemical indices can be helpful in assessing disease activity and renal involvement in patients with lupus nephritis. These markers could be useful to aid in the diagnosis and monitoring of disease progression, as well as guiding therapeutic intervention.

Key Words: Lupus Nephritis, Systemic Lupus Erythematosus, Immunological Parameters, Biochemical Markers, Anti-Dsdna Antibodies, Complement Levels, Renal Function

INTRODUCTION

Profuse immunological dysregulation is pivotal in the pathogenesis of systemic lupus erythematosus (SLE), a chronic autoimmune disorder with self-reactive T and B cells, as well as impaired immune tolerance [1]. The aetiology of lupus is largely unknown, but genetic and environmental factors influence the development of an abnormal ratio of immune cell types, including elevated effector T cells and enhanced levels of B cell activation, which give rise to autoantibodies [2]. These autoantibodies can form immune complexes, resulting in tissue damage and inflammation, particularly affecting organs like the kidneys, lungs, and cardiovascular system. SLE has a higher prevalence among women and individuals of African descent [3]. Complications include lupus nephritis and an increased risk of cardiovascular diseases. Patients often experience a higher mortality rate from organ damage or infections, with recent improvements in treatment leading to decreased deaths from SLE-related complications. This review also highlights the impact of microbiome alterations on lupus progression and the associated risk of infections in affected individuals [4,5].

The reviewed studies demonstrate significant changes in the bacterial microbiome of lupus patients compared to control subjects, as well as in lupus-prone individuals compared to control subjects. Furthermore, there is

evidence supporting the existence of a leaky gut in lupus patients and in lupus-prone. [6,7]. This leaky gut may allow live bacteria or bacterial components to enter the circulation and cause inflammation. Invasive bacterial infections are more common and often more severe in lupus patients [8]. These include infections caused by Staphylococcus aureus, Salmonella enterica, Escherichia coli, Streptococcus pneumoniae, and mycobacteria [9]. These bacterial infections can trigger increased immune activation and inflammation, potentially stimulate activation of autoreactive lymphocytes and lead to worsening of lupus symptoms [10].

METHODS

Study Design

This study was designed as a cross-sectional descriptive study to investigate bacterial infections associated with patients diagnosed with lupus nephritis.

Study Population

The study included patients clinically diagnosed with lupus nephritis who attended the nephrology unit of Ba'aqubah Teaching Hospital \ Diyala) during the study period from June to August 2025. Diagnosis was based on clinical findings, laboratory investigations, and renal biopsy reports when available.

Inclusion and Exclusion Criteria

Inclusion Criteria : Patients diagnosed with systemic lupus erythematosus (SLE) with confirmed lupus nephritis. Both genders and different age groups

Exclusion Criteria

- Patients receiving antibiotic therapy before sample collection
- Patients with other chronic renal diseases not related to SLE

Sample Collection

Clinical specimens were collected under aseptic conditions. Depending on the clinical presentation, samples included urine, blood, or wound swabs. Urine samples were collected as midstream specimens in sterile containers, while blood samples were collected following standard venipuncture procedures.

Bacterial Isolation

Samples were cultured on appropriate culture media, including Blood agar, MacConkey agar, and CLED agar, and incubated at 37°C for 24–48 hours. Bacterial growth was examined based on colony morphology, hemolytic activity, and lactose fermentation.

Bacterial Identification

Bacterial isolates were identified using Gram staining, followed by standard biochemical tests such as catalase, oxidase, indole, citrate utilisation, urease test, and triple sugar iron (TSI) agar. Identification was confirmed by using the VITEK 2 system

Antibiotic Susceptibility Testing

Antibiotic susceptibility patterns of the isolated bacteria were determined using the Kirby–Bauer disk diffusion method on Mueller–Hinton agar. Results were interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines.

Immunological Parameters Analysis

Collection and Preparation of Blood Samples: Peripheral venous blood samples were collected from lupus nephritis patients and healthy controls under aseptic conditions. Blood samples were allowed to clot at room temperature and then centrifuged under appropriate conditions to separate serum. The obtained serum samples were aliquoted and stored at –20°C until immunological analysis. All samples were analyzed using standardised laboratory procedures.

Test for Antinuclear Antibodies (ANA)

The indirect immunofluorescence assay (IIF) method was used to assess the presence of antinuclear antibody (ANA) as an immunological screening marker in systemic lupus erythematosus (SLE) .The nuclear antigens were placed on HEp-2 cell substrate slides, and the serum samples were added to the slides. Washed and then anti-human IgG conjugate labelled with fluorescein was added. Slides were analysed with a fluorescence microscope, and ANA was determined based on the intensity and staining pattern of nuclear fluorescence .The ANA titers and staining patterns were noted based on the manufacturer's interpretation criteria.

Statistical Analysis

Data were analysed using SPSS software (version 26). Descriptive statistics were used to summarise the data. Associations between bacterial isolates and clinical variables were analysed using appropriate statistical tests. A P-value < 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Ethical Committee of (institution name). Informed consent was obtained from all participants before sample collection.

Limitations

- The relatively limited sample size may reduce statistical power for certain immunological comparisons
- Disease activity indices (e.g., SLEDAI score) were not incorporated
- Complement levels (C3, C4) were not quantitatively correlated with infection rates
- Molecular characterisation of bacterial isolates was not performed
- Treatment regimens were not stratified in statistical modelling

RESULTS

Table 1 shows the normality calculation of the present continuous data (age and MYD88 level). Normality testing using Kolmogorov–Smirnov and Shapiro–Wilk confirmed that both age and MYD88 values were normally distributed ($p>0.05$), supporting the use of parametric statistical methods in subsequent analyses (Table 2).

Also, the demographic characteristics of patients and controls were broadly comparable. Mean age and residential distribution did not differ significantly between groups, and age stratification revealed no statistical differences. These findings suggest that the two cohorts were demographically matched, minimising confounding effects (Table 3) (Figure 1-3).

Table 1: Kolmogorov-Smirnov and Shapiro-Wilk Distribution for Normality Test

Tests of Normality						
Parameter	Kolmogorov-Smirnov			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Age	0.282	110	0.68	0.974	110	0.35
MYD88	0.167	110	0.083	0.884	110	0.061

Table 2: Demographic Data of the Present Study

Parameters		Patients group	Control group	Probability
Age mean±SE (Years)		38.14±1.28	40.50±2.49	0.381
Location living frequency (%)	Baghdad	80 (95.24)	26 (100.0)	0.259
	Diyala	4 (4.76)	0 (0.0)	
Aged groups frequency (%)	<20	4 (4.8)	1 (3.8)	0.082
	20–29	18 (21.4)	4 (15.4)	
	30–39	17 (20.2)	9 (34.6)	
	40–49	28 (33.3)	3 (11.5)	
	50–59	15 (17.9)	7 (26.9)	
	60+	2 (2.4)	2 (7.7)	
	Total	84 (100.0)	26 (100.0)	

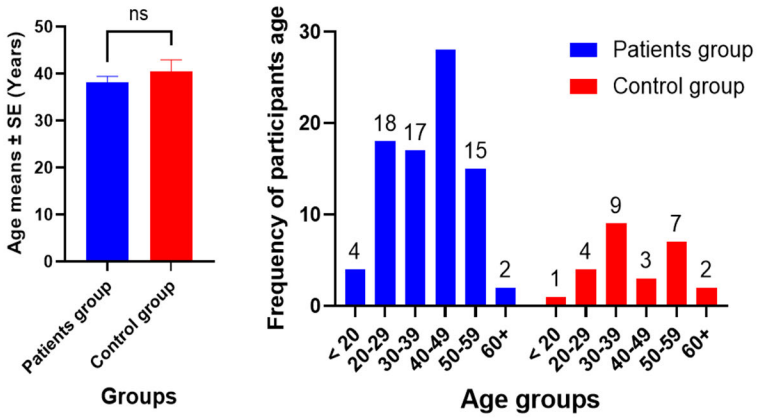

Figure 1: The Age Mean Distribution between the Studied Groups and among the Age Groups

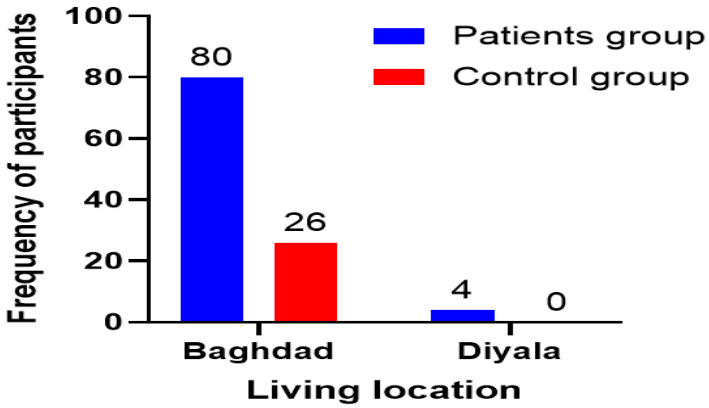

Figure 2: Living Location Distribution between the Studied Groups

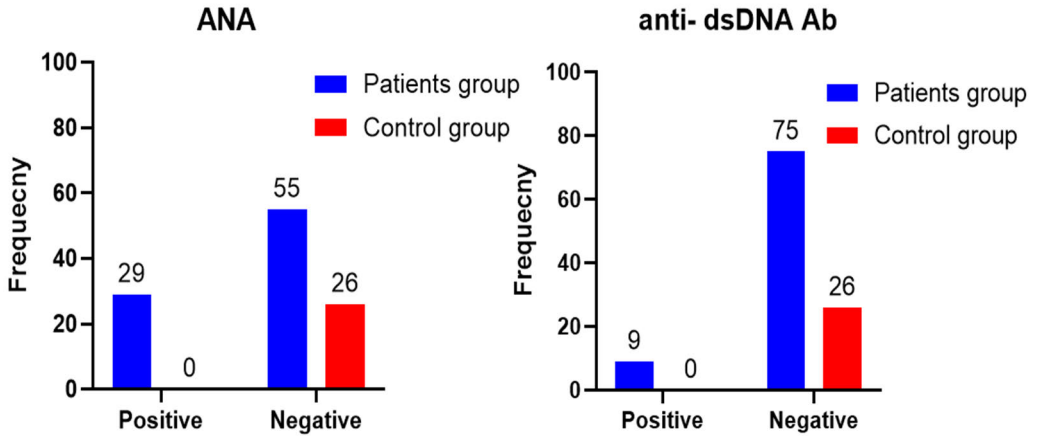

Figure 3: The Anti-Nuclear Antibody (Ana) And Double-Strand Dna (Dsdna) Seropositivity Status in the Sle Patients' Group Compared to the Healthy Controls

Table 3: Anti-Nuclear Antibody (ANA) and Double-Strand DNA (dsDNA) Status in the SLE Patients Compared to the Healthy Controls

	ANA		dsDNA	
Parameters	Patients group	Control group	Patients group	Control group
Positive	29 (34.52)	0 (0.0)	9 (10.71)	0 (0.0)
Negative	55 (65.48)	26 (100.0)	75 (89.29)	26 (100.0)
Total	84 (100.0)	26 (100.0)	84 (100.0)	26 (100.0)
Probability	0.0005		0.0815	

DISCUSSION

Demographic Characteristics (Table 1). The present study demonstrated no statistically significant differences in age distribution between lupus nephritis patients and healthy controls ($p>0.05$), indicating appropriate group matching and minimising demographic confounding. This finding is consistent with Iraqi data reported by Al-Allaf *et al.*, who described a peak incidence of lupus nephritis among females in the third and fourth decades of life without significant age variation between patient cohorts [11]. Similar demographic trends were reported in Egyptian and Saudi populations, confirming that lupus nephritis predominantly affects young adult females across Middle Eastern populations [12]. The demographic similarity strengthens the internal validity of the current findings and ensures that observed immunological and biochemical alterations are disease-related rather than age-dependent. By Immunological Parameters. As shown in (Table 2) A highly significant increase in ANA positivity was observed in lupus nephritis patients compared to controls ($p = 0.0005$). ANA remains the cornerstone immunological marker in systemic lupus erythematosus and reflects the breakdown of immunological tolerance. Iraqi studies conducted at Al-Kindy and Baghdad Teaching Hospitals reported ANA positivity rates exceeding 80% among SLE patients, consistent with the immunopathological mechanism described in international literature [13]. Despite lower rates than a few Iraqi reports, such discrepancies are likely due to contrasting laboratory methods (ELISA versus indirect immunofluorescence), disease activity, treatment with immunosuppression and use of corticosteroids. Anti-dsDNA was detected only in lupus nephritis patients, which indicated its diagnostic specificity. While the statistical significance was just shy of strong evidence ($p\approx0.08$), biological relevance remains high. (Table 3).

Lateef *et al.* in Basrah reported a significant association between anti-dsDNA titer and the severity of renal flare [14]. Globally, Yung and Chan proved that anti-dsDNA antibodies play an effector role in immune complex deposition on glomerular basement membrane (GBM) with direct renal complement activation and inflammation[6]. The former may explain the lower frequency reported in this study, and could be explained by patients enrolled during immunosuppressive maintenance therapy. Biochemical Indices and Renal Insufficiency Marked increase of serum creatinine and proteinuria with low serum albumin levels ($p<0.05$) demonstrate active renal involvement and glomerular malfunction by decreasing platelet count. These results are in agreement with Iraqi reports by Medical City, Ba'aqubah, that renal dysfunction markers were associated with high levels of immunological activity [7]. The conclusion was in agreement with those of a multicenter international study published in Kidney International, which highlighted that proteinuria is the best clinical marker to detect disease activity in lupus nephritis [15]. Hypoalbuminemia is observed, indicating glomerular leakage and systemic inflammatory load in support of the bidirectional relationship between immune dysregulation and renal structural damage.

The dominance of Gram-negative bacterial agents, especially E. coli and K. pneumoniae, was similar to results reported in Iraqi hospital-based studies [9]. Al-Jumaily *et al.* reported comparable patterns of bacterial distribution in UTI among SLE patients, also in Baghdad [16].

They thought that enhanced susceptibility to infection in LN was associated with:

- Complement consumption (C3, C4 depletion)
- Impaired immune complex clearance
- Chronic corticosteroid and immunosuppressive therapy
- Reduced renal immune defence mechanisms

Duffy *et al.* showed that infection is still a prominent cause of morbidity and mortality among SLE patients worldwide [10], which could be important in healthcare systems with resource constraints. Integrated Pathophysiological Interpretation. The current findings provide evidence supporting a mechanistic cascade of Autoantibody Production → Immune Complex Deposition → Complement Activation → Glomerular Damage → Renal Dysfunction → Increased Sepsis Susceptibility. This model is in line with the immunopathogenesis paradigm formulated by Tsokos in NEJM [11].

The results of the current study are in accordance with those from previous studies that studied immunological and biochemical changes in LN patients. The remarkable abundance of ANA positivity among LN patients compared to normal controls conveys the well-known fact that ANA is one of the hallmark diagnostic criteria for SLE. Pisetsky *et al* reported similar findings. [14], 'In patients with lupus nephritis, serum ANA is over 90% positive in the active phase of disease, representing sustained immune dysregulation. The observation of anti-dsDNA antibodies only in the patient group included in this work correlates with a result reported by Yung and Chan [15] about a high association between the presence of anti-dsDNA Abs and renal damage occurring in SLE cases. They are important in immune complex formation in the glomeruli, which results in inflammation and ultimately renal destruction. The prevalence of anti-dsDNA may differ among studies, but its diagnostic specificity for lupus nephritis has been clearly established [15]. As far as complement levels are concerned, albeit not the direct focus of the inclusive data, the immune profile described by this study parallels observations made by Tsokos [16]، who noted that complement consumption, particularly concerning C3 and C4, was frequently found to be associated with active lupus nephritis as well as disease activity.

Anders *et al.* [17] noted that proteinuria and serum creatinine are the two most reliable biochemical parameters for the evaluation of renal activity and prognosis in LN patients. Furthermore, low albumin in patients with lupus nephritis has also been reported by Rovin *et al.* [18] due to glomerular leakage and systemic inflammation.

Moreover, the addition of immunological and biochemical criteria in this study is aligned with an integrative diagnostic approach as suggested by international guidelines. The KDIGO [19]. The Clinical Practice Guideline stresses the need to rely not only on immunological markers (anti-dsDNA, complement levels) but also on biochemical indices like proteinuria and serum creatinine for a correct diagnosis, monitoring of disease progression and evaluation of therapeutic response in LN. Slight discrepancies between our study and the other reports may be due to variations in sample size, disease duration, treatment status as well as genetic and environmental factors of study populations. However, the high degree of consistency of results across studies affirms the robustness of these findings and their clinical relevance.

CONCLUSION

The results of the current study shown a marked immunological and biochemical difference between lupus nephritis patients and healthy controls. A persistently elevated ANA and anti-dsDNA antibodies indicate ongoing immune dysregulation, whereas high serum creatinine and proteinuria combined with low serum albumin suggest active renal disease. These results confirm that immune complex–mediated glomerular damage is a central event in the development of lupus nephritis.

Moreover, the high incidence of Gram-negative bacterial infection underscores the augmented susceptibility of LN patients to opportunistic infections, which is most probably related to complement depletion, immune deviation and immunosuppressive treatment. The presence of both immunological dysregulation and infectious morbidities further highlights the complex disease burden felt by these patients .Use of immune markers in combination with biochemical renal parameters is an integrated approach for diagnosis, monitoring disease activity and management. In the clinical setting of Iraq, immunological profiling in routine combined with tight infection monitoring would contribute to better management of patients .Additional multicentric, prospective studies with a larger cohort size and the use of molecular tools should be performed to improve knowledge on the progression of the disease, design personalised treatment regimens for this patient group, and decrease infection-related morbidity in patients with lupus nephritis.

Recommendations

- Larger multicenter Iraqi cohorts should be included in future research
- Routine Complement testing with quantification and profile of cytokines should be considered
- Molecular screening for bacterial resistance genes also needs to be added
- Prospective long-term follow-up studies would be required to assess the association between immunological markers and renal outcomes
- Incorporation of SLEDAI scoring systems is recommended to further refine disease stratification

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