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Clinical characteristics and laboratory findings of Iranian patients with systemic lupus erythematosus

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Abstract

Background: Systemic lupus erythematosus (SLE) is a long-term autoimmune condition characterized by diverse clinical and laboratory manifestations influenced by genetic, environmental, and socioeconomic factors. This study aimed to characterize the clinical and laboratory profiles of SLE in Iranian patients.

Methods: A cross-sectional study was conducted on patients with SLE at a private rheumatology clinic from 2015 to 2023. Data on clinical manifestations and laboratory findings, including immunological indices, systemic inflammation, hematological, metabolic, thyroid, and renal factors, were collected from patient medical records.

Results: Of 313 patients (284 females, 29 males; mean age: 43.68±12.39 years for females, 40.20±10.17 years for males), the most prevalent clinical manifestations were joint pain (61.94%) and skin rash (60%). Renal, vascular, serositis, neuropsychiatric, and cardiac manifestations occurred in 33.33%, 17.15%, 9.39%, 7.44%, and 2.91% of patients, respectively. Leukopenia was observed in 24.10%, thrombocytopenia in 10.13%, and all patients were ANA-positive. Gender differences included higher leukopenia in females (25.63% vs. 10.34%; $P=0.041$) and elevated LDL in males (25% vs. 9.6%; $P=0.014$).

Conclusion: This study delineates the clinical and laboratory characteristics of SLE in Iranian patients, revealing patterns consistent with global cohorts but shaped by regional genetic and environmental factors. Comparisons with international studies highlight the influence of ethnicity on disease presentation, emphasizing the need for tailored management and prospective research to address data gaps, such as histopathological and serological details.

Keywords: Systemic lupus erythematosus, Epidemiology, Skin manifestations, Prevalence, Leukopenia

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Introduction

Systemic lupus erythematosus (SLE) is a chronic, potentially life-threatening, and complex autoimmune disorder marked by immune system dysregulation, resulting in diverse multi-organ manifestations (1, 2). The heterogeneous nature of the disease results in a wide variety of clinical and laboratory findings in each patient (3). Common clinical manifestations include cutaneous rashes (e.g., malar rash), arthritis, fatigue, and renal involvement, with lupus nephritis affecting up to 50% of patients in some populations (4). Laboratory findings frequently reveal autoantibodies such as antinuclear antibodies (ANA) in nearly all patients, as well as low complement levels (C3 and C4), leukopenia, thrombocytopenia, and proteinuria in a significant proportion of cases (5).

While many aspects of SLE pathogenesis remain to be fully understood, it is known that the development of SLE is influenced by a mix of genetic and environmental triggers, as well as immunological and hormonal components (6). SLE is a relatively uncommon disease; however, its incidence is rising. The estimated annual occurrence ranges from 0.3 to 31.5 individuals per 100,000, with a prevalence rate of 50 to 100 individuals per 100,000 (7). The prevalence and incidence of SLE differ by geographic region, influenced by factors such as race, ethnicity, age, and gender (8). For instance, the highest incidence has been reported in North America, while the lowest is observed in Africa and Ukraine (8). Clinical symptoms and severity of SLE also vary due to genetics and ethnic factors (9). Studies have demonstrated that individuals of Black, East Asian, South

Asian, and Hispanic descent with SLE are more prone to developing severe manifestations, such as lupus nephritis, and experience more rapid disease-related damage (9). In the United States, the Black population tends to develop lupus nephritis at an earlier stage and exhibits more severe features compared to the White population (4).

To enhance the quality of healthcare, diagnosis, treatment, and screening for SLE, it is essential to evaluate patients across various regions, races, ethnicities, genetics, and environmental exposures. Conducting epidemiological studies is imperative for determining the frequency and characteristics of SLE manifestations across different populations. These studies offer important insights into the influence of intrinsic and extrinsic factors on the clinical and laboratory manifestations of SLE patients. We aimed to examine the prevalence of clinical presentations and laboratory findings among SLE patients who pursued treatment at a private rheumatology clinic between 2015 and 2023.

Methods

Study Design

This cross-sectional study included all SLE patients fulfilling the American College of Rheumatology (ACR) classification criteria (10). Organ involvement was diagnosed by a rheumatologist through a detailed medical history and comprehensive physical examination. Neurological involvement was identified based on the patient's history of headache, seizures, and psychosis, along with clinical findings suggestive of central nervous system (CNS) involvement, and was confirmed by brain MRI. Vascular involvement was assessed based on the patient's history of Raynaud's phenomenon and clinical evidence of vasculitis. Cardiac involvement was evaluated through the patient's symptoms, such as shortness of breath, chest pain, and palpitations, along with echocardiographic assessment of valvular abnormalities, ejection fraction (EF), and electrocardiogram (ECG) findings. Renal involvement was determined by clinical manifestations, including edema, high blood pressure, or alterations in urinary output. Urinalysis was performed to check for proteinuria, hematuria, and signs of kidney failure. Blood tests were conducted to evaluate kidney function via serum concentration of creatinine and blood urea nitrogen (BUN) levels. If lupus nephritis was suspected, a kidney biopsy was performed to assess the extent of damage. Informed consent was received from all patients before entering the study. The study received approval from the local ethics committee, ensuring compliance with ethical standards and guidelines.

Laboratory Measurements

Laboratory tests conducted in this study encompassed several parameters. Enzyme-linked assays on a multiple-sample analyzer (Parsazmun, Karaj, Iran) were employed to assess total cholesterol, low-density lipoprotein (LDL-C), and high-density lipoprotein (HDL-C). Renal function was assessed through markers such as 24-hour urine protein, creatinine (Cr), urinalysis, and BUN. Renal biopsy specimens were examined using both light microscopy and immunofluorescence microscopy. Immunological indexes, including complement component 3 (C3) and complement component 4 (C4), were evaluated. Complement levels were evaluated via nephelometry. antinuclear antibodies (ANA) and anti-double-stranded DNA (anti-dsDNA) antibodies were assessed as part of routine clinical evaluations for SLE diagnosis using standard immunofluorescence assays, with positivity determined according to the diagnostic criteria.

Markers of systemic inflammation, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) measured in the first hour, were evaluated. Thyroid function was determined by levels of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH). Additionally, hematological factors, including hemoglobin, white blood cell count, and platelet count, as well as metabolic parameters like fasting blood sugar, were recorded. Proteinuria was characterized by the presence of over 500 mg of protein in a 24-hour urine collection. Leukopenia was identified as a white blood cell count below 4500 cells/μL. Thrombocytopenia was characterized as having a platelet count below 150,000 cells/μL.

Statistics

All statistical calculations were conducted using Stata version 18 (Stata Corp., College Station, TX, USA). Descriptive data were reported using percentages with a 95% confidence interval (95% CI). Continuous variables are shown as means and standard deviation (SD) or as medians and interquartile ranges (IQR), as suitable. In addition, the chi-square test (or Fisher's exact test) was employed to compare differences among categorical variables. A *P*-value of 0.05 was utilized to determine statistical significance.

Results

Demographic Characteristics

A comprehensive analysis was performed on a total of 313 SLE patients. Among them, 284 (90.73%) were identified as female, while 29 (9.27%) were male, yielding a female-to-male ratio of 10:1. The mean age for women was 43.68 ± 12.39 years, while the mean age for men was recorded as 40.20 ± 10.17 years.

Clinical Manifestations

The most prevalent manifestations among the patients were joint pain (61.94%) and skin rash (60%). Skin manifestations were more prevalent in females (61.07%) than in males (50.0%), though this difference was not

statistically significant (Table 1).

As shown, 103 (33.33%) out of the total patients exhibited renal manifestations of SLE. Renal involvement was meaningfully more common in males (55.17%) compared to females (31.07%) ($P=0.009$).

Our data shows that 23 patients (7.44%) had neurologic manifestations. Neurologic manifestations were more prevalent in males (10%) than in females (7.14%), though this difference was not statistically significant (Table 1).

Moreover, cardiac involvement was identified in 9 (2.91%) of the patients. Likewise, 17.15% of study participants showed vascular events. In addition, serositis was found among 29 SLE patients (9.39%) of the total population, including 27 (9.64%) females. No significant difference was observed between female and male SLE patients in terms of clinical manifestations, except for kidney involvement ($P=0.01$).

Laboratory Features

Laboratory findings in our cohort of Iranian SLE patients are summarized in Table 2, with gender-based comparisons highlighting significant differences in several parameters. Leukopenia, defined as a white blood cell count ≤ 4,500/μL, was observed in 24.1% of patients, occurring more frequently in women than in men (25.63% vs. 10.34%; $P=0.041$). Thrombocytopenia (platelet count ≤ 150,000/μL) occurred in 10.13% of patients, while proteinuria was detected in 44.34%. Elevated serum creatinine levels (> 1.1 mg/dL for women, > 1.2 mg/dL for men) were noted in 13.71% of patients. Lipid profile abnormalities included elevated total cholesterol levels in 17.14% and elevated LDL in 11.15% of patients, while men exhibited a higher prevalence of elevated LDL compared to women (25% vs. 9.6%; $P=0.014$). Conversely, HDL levels were significantly lower in women than in men (21.43% vs. 40.56%; $P=0.049$).

Complement levels were also affected, with low C3 and C4 levels observed in 15.9% and 15.48% of patients, respectively. Notably, low C3 levels were more frequent in men than in women (35% vs. 14.16%; $P=0.015$). Elevated BUN was more common in men (57.14% vs. 33.09%; $P=0.011$). An elevated ESR (ESR ≥ 30 mm/h) was found in 19.67% of patients, and CRP was positive in 21.31%. All patients tested positive for ANA, and 93.88% were negative for lupus anticoagulant. Abnormal hemoglobin levels were observed in 28.99% of patients, and vitamin D < 30 ng/mL was prevalent in 44.81%, with no significant gender differences. A statistically significant variation in TSH levels was noted between females and males ($P=0.03$). Diabetes was present in 2.45% of patients, with no significant gender association ($P=0.5$).

Table 1. Frequency of Clinical Signs in SLE patients.

Variables	Total, n (%)	Female, n (%)	Male, n (%)	*P*-value*
Joint pain	191 (61.81)	176 (62.86)	15 (51.72)	0.240
Skin rash	186 (60.19)	171 (61.07)	15 (51.72)	0.328
Kidney	103 (33.33)	87 (31.07)	16 (55.17)	0.009
Neurologic	23 (7.44)	20 (7.14)	3 (10.34)	0.532
Vascular	53 (17.15)	49 (17.50)	4 (13.79)	0.614
Serositis	29 (9.39)	27 (9.64)	2 (6.90)	0.629
Cardiac	9 (2.91)	8 (2.86)	1 (3.45)	0.593

Abbreviations: SLE: Systemic lupus erythematosus; n: Number.
*A *P*-value < 0.05 was considered indicative of statistical significance.

Discussion

SLE exhibits significant variability in prevalence, clinical manifestations, and laboratory findings across populations, driven by genetic, environmental, and socioeconomic factors (8).

Our study found that joint pain (61.81%) and skin rash (60.19%) were the most prevalent clinical manifestations, consistent with global reports where mucocutaneous and articular symptoms affect 60-90% and 60-80% of SLE patients, respectively (11). These findings align closely with a study on 300 Turkish SLE patients, also of Caucasian descent, which reported mucocutaneous (72%) and arthritis (66.3%) as leading manifestations (12). The slightly lower prevalence in our cohort may reflect differences in case ascertainment or environmental triggers, such as sunlight exposure, which is a known exacerbating factor for skin rashes in SLE (11, 13). Renal involvement was observed in 33.33% of our patients, lower than the 40.3% reported in the study conducted in Turkey, where Class IV lupus nephritis was predominant (12). This discrepancy may be attributed to genetic predispositions or variations in diagnostic thresholds for renal biopsy, which were not available in our retrospective dataset, as noted in our limitations.

Hematological abnormalities were prevalent in our cohort, with leukopenia (24.10%), thrombocytopenia (10.13%), and abnormal hemoglobin (28.99%). Compared to Tekeoglu et al., who reported a higher prevalence of hematological manifestations (72.6%), our lower rates may reflect differences in disease duration or treatment regimens, as prolonged corticosteroid use can mitigate hematological abnormalities (12). In a retrospective study of 632 patients with SLE, the researchers reported a 58% prevalence of thrombocytopenia at diagnosis (14). In a study from Saudi Arabia, hematological abnormalities were observed in 82.7% of SLE patients (15). Laboratory findings, including low C3 (15.90%) and C4 (15.48%) levels, were consistent with complement activation in SLE (16), though less frequent than in some cohorts, possibly due to milder disease phenotypes in our population.

The epidemiology of SLE demonstrates significant variations across different age and gender categories, as well as unequal distribution among geographical regions (17).

SLE is an autoimmune disorder marked by a diverse

Table 2. Comparison of Laboratory Factors by Gender.

Variables	Total, n (%)	Female, n (%)	Male, n (%)	*P*-value
WBC (/μL)				
<4500	74 (24.10)	71 (25.63)	3 (10.34)	0.041
4500-11000	222 (72.31)	198 (71.48)	23 (79.31)	
>11000	11 (3.58)	8 (2.89)	3 (10.34)	
PLT (/μL)				
<150,000	31 (10.13)	29 (10.47)	2 (6.90)	0.435
150,000-450,000	270 (88.24)	244 (88.09)	26 (89.66)	
>450,000	5 (1.63)	4 (1.44)	1 (3.45)	
HGB (g/dL)				
Normal	218 (71.01)	200 (72.20)	17 (58.62)	0.125
Abnormal	89 (28.99)	77 (27.80)	12 (41.38)	
Lupus Anticoagulant				
Positive	6 (6.12)	6 (6.45)	0 (0.00)	0.724
Negative	92 (93.88)	87 (93.55)	5 (100.00)	
CRP (mg/dL)				
Positive	64 (21.31)	58 (21.32)	6 (21.43)	0.990
Negative	236 (78.67)	214 (78.68)	22 (78.57)	
ESR (mm/hr)				
<30	241 (80.33)	215 (79.34)	26 (89.66)	0.184
≥30	59 (19.67)	56 (20.66)	3 (10.34)	
BUN (mg/dL)				
<24	192 (64.65)	180 (66.91)	12 (42.86)	0.011
≥24	105 (35.35)	89 (33.09)	16 (57.14)	
Creatinine (mg/dL)				
Normal	258 (86.29)	234 (86.67)	24 (82.76)	0.561
Abnormal	41 (13.71)	36 (13.33)	5 (17.24)	
Proteinuria (mg/24 hours)				
Positive	47 (44.34)	38 (41.30)	9 (64.29)	0.093
Negative	59 (55.66)	54 (58.70)	5 (35.71)	
Calcium (mg/dL)				
<8.5	21 (7.29)	18 (6.90)	3 (11.11)	0.533
8.5-10.5	265 (92.01)	241 (92.34)	24 (88.89)	
>10.5	2 (0.69)	2 (0.77)	0 (0.00)	
Phosphorus (mg/dL)				
<2.5	2 (0.69)	1 (0.38)	1 (3.70)	0.228
2.5-4.5	250 (86.81)	227 (86.97)	23 (85.19)	
>4.5	36 (12.50)	33 (12.64)	3 (11.11)	
C3 (mg/dL)				
<80	38 (15.90)	31 (14.16)	7 (35.00)	0.015
≥80	201 (84.10)	188 (85.84)	13 (65.00)	
C4 (mg/dL)				
<10	39 (15.48)	33 (14.35)	6 (27.27)	0.109
≥10	213 (84.52)	197 (85.65)	16 (72.73)	
Total cholesterol (mg/dL)				
<200	232 (82.86)	209 (82.94)	23 (82.14)	0.916
≥200	48 (17.14)	43 (17.06)	5(17.86)	

Table 2. Continued.

Variables	Total, n (%)	Female, n (%)	Male, n (%)	*P*-value
LDL (mg/dL)				
<130	247 (88.85)	226 (90.40)	21 (75.00)	0.014
≥130	31 (11.15)	24 (9.60)	7 (25.00)	
HDL (mg/dL)				
Normal	170 (61.37)	148 (59.44)	22 (78.57)	0.049
Abnormal	107 (38.63)	101 (40.56)	6 (21.43)	
FBS (mg/dL)				
<125	279 (97.55)	252 (97.67)	27 (96.43)	0.518
≥125	7 (2.45)	6 (2.33)	1 (3.57)	
T3 (ng/L)				
<75	41 (39.81)	37 (38.14)	4 (66.67)	0.309
75-195	60 (58.25)	58 (59.79)	2 (33.33)	
>195	2 (1.94)	2 (2.06)	0 (0.00)	
T4 (mg/dL)				
<4.6	8 (7.84)	7 (7.29)	1 (16.67)	0.310
4.6-11.2	83 (81.37)	79 (82.29)	4 (66.67)	
>11.2	11 (10.78)	10 (10.42)	1 (16.67)	
TSH (mU/L)				
<0.5	4 (3.81)	4 (4.04)	0 (0.00)	0.032
0.5-4.5	69 (65.71)	68 (68.69)	1 (16.67)	
>4.5	32 (30.48)	27 (27.27)	5 (83.33)	
Vitamin D (nmol/L)				
<30	121 (44.81)	108 (44.26)	13 (52.00)	0.459
≥30	148 (55.02)	136 (55.74)	12 (48.00)	

Abbreviations: WBC: white blood cell; PLT: platelet; HGB: Hemoglobin; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; BUN: Blood Urea Nitrogen; C3: Complement 3; C4, Complement 4; LDL: Low-Density Lipoproteins; HDL: High-Density Lipoproteins; FBS: Fasting Blood Sugar; T3: Triiodothyronine; T4: Thyroxine; TSH: thyroid-stimulating hormone; n: Number.
*A *P*-value < 0.05 was considered indicative of statistical significance.

range of symptoms and primarily affecting young women. Certain ethnic populations are more susceptible to developing SLE and tend to experience higher rates of morbidity and mortality (18). Gender-based differences in our study revealed that women had a higher prevalence of leukopenia (25.63% vs. 10.34%; $P=0.04$), while men exhibited higher LDL levels (25% vs. 9.6%; $p=0.014$) and lower HDL levels (21.43% vs. 40.56%; $P=0.04$). These findings partially align with Bolla et al. (2024), who reported a female predominance (89.7%) and significant cardiovascular risk factors, including hyperlipidemia (19.8%), in a group of 3401 patients with SLE (19). The higher LDL in men in our study may reflect gender-specific metabolic differences or hormonal influences, which warrant further investigation (20). Cardiovascular disease (CVD) risk is a critical concern in SLE, and our study showed elevated total cholesterol (17.14%) and LDL (11.15%), which is consistent with the 36-60% prevalence of dyslipidemia reported globally (21). The increased CVD risk in SLE, driven by autoantibodies, inflammatory mediators, and endothelial damage (22), underscores the need for routine cardiovascular risk assessment, particularly in younger patients, as highlighted by Bolla et al. (19). Our findings of higher LDL in men compared to women suggest gender-specific risk profiles that may guide targeted interventions.

The prevalence of SLE in Iran appears to be rising, consistent with global trends, especially in nations with low to middle income levels where ethnic diversity and socioeconomic factors influence disease burden (23). Limitations of our study include its retrospective design, which restricted data on chronic skin involvement and kidney biopsy pathology, as well as the lack of longitudinal follow-up to assess disease progression. Future prospective studies in Iranian and other Middle Eastern populations should incorporate detailed dermatological and histopathological data to better characterize SLE manifestations and their prognostic implications.

Conclusion

In conclusion, our study highlights the clinical and laboratory profile of SLE in Iranian patients, with findings broadly consistent with global patterns but nuanced by regional genetic and environmental factors.

Comparisons with Turkish and international cohorts underscore the importance of ethnicity and geography in shaping SLE presentation, guiding personalized care and future research.

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Competing Interests
The authors declare that there is no conflict of interest.

Data Availability Statement
The datasets analyzed during the current study are available from the corresponding author at reasonable request.

Ethical Approval
The study was approved by the Ethics Committee of the Mashhad University of Medical Sciences (Ethical Approval code: IR.MUMS.MEDICAL.REC.1399.786). Informed consent was obtained from the patients before entering the study.

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