Open Access

Original Article

# Association of Systemic Lupus Erythematosus with Dyslipidemia

**Muhammad Imran, Hajra Ahmad, Alam Zeb, Najib Ullah, Usman Ali, Almas Khan***

Department of Rheumatology, Khyber Teaching Hospital, Peshawar Pakistan, *Department of Medicine, Khyber Teaching Hospital, Peshawar Pakistan

## ABSTRACT

***Objective***: To find out the association between Systemic Lupus Erythematosus (SLE) disease activity and lipid profile of patients.

***Study Design***: Cross Sectional Study.

***Place and Duration of Study***: Khyber Teaching Hospital, Peshawar Pakistan, from May 2023 to Feb 2024.

***Methodology***: Seventy-eight patients of Systemic Lupus Erythematosus (SLE), were recruited using non-probability consecutive. Apart from patient demographics, SLE disease activity was calculated, and lipid profiles were sent to hospital laboratory sent to hospital laboratory. Correlation between SLE disease activity and different parameters was checked.

***Results***: Our study of 78 patients revealed a predominantly female population 63(80.7%) with moderate disease activity 54(69.2%). Notably, abnormal lipid profiles were observed, with significant positive correlations found between SLE Disease Activity Index (SLEDAI) and Total Cholesterol (r=0.702, *p*=<0.001), Triglycerides (r=0.238, *p*=0.038), and Low-Density Lipoprotein (LDL) (r=0.633, *p*=<0.001). High-Density Lipoprotein (HDL) exhibited a non-significant correlation (r=0.074, *p*=0.519), while duration of disease showed minimal impact (r=-0.045, *p*=0.699).

***Conclusion***: The study demonstrated a significant correlation between SLE Disease Activity Index (SLEDAI) and total cholesterol, low density lipoproteins and triglycerides.

**Keywords**: Cholesterol, High Density Lipoprotein, Lipid Profile, Systemic Lupus Erythematosus.

***How to Cite This Article:*** *Imran M, Ahmad H, Zeb A, Ullah N, Ali U, Khan A. Association of Systemic Lupus Erythematosus with Dyslipidemia. Pak Armed Forces Med J 2026; 76(1): 35-38. DOI: https://doi.org/10.51253/pafmj.v76i1.11665*



## INTRODUCTION

Systemic lupus erythematosus (SLE) is a prevalent and potentially fatal autoimmune disease. The hallmark of SLE is multiorgan damage caused by autoantibodies, which can harm the cutaneous, neurological, cardiovascular, and renal systems.[1] The Center for Rheumatology Research's comprehensive research of rheumatic disorders estimates the prevalence of SLE to be 40 cases per 100,000 persons.[2] Women, with a female to male ratio of 15.5:1, make up most of the cases. SLE can present with a range of symptoms, from skin and joint involvement to potentially fatal consequences like lupus nephritis.[3]

Increases or decreases in the serum lipid fraction (lipoprotein) are indicative of dyslipidemia, a lipid-metabolism condition. It is widely acknowledged that dyslipidemia is a common ailment among SLE patients and that it is a significant risk factor for heart failure, cardiovascular disease (CVD), and renal illness.[4]

In SLE, blood lipids may be significant indicators of disease severity. Among adult SLE patients, dyslipoproteinemia affects 30% to 73% of them.[5,6] There are two patterns of dyslipoproteinemia, according to studies. Triglyceride and high density lipoprotein (HDL) levels are elevated in the first, which is associated with active disease; in contrast, the second is associated with high dosage steroid treatment and is unrelated to active disease.[7,8] Hyperlipidemia according to National Cholesterol Education Program criteria, which defines it as having Total Cholesterol (TC) >200mg/dL, Triglyceride (TG) >150mg/dL, LDL-C >100mg/dL, or HDL-C <40mg/dL.[3] In the International Collaborating Clinics cohort, hypercholesterolemia was seen in 36% of newly diagnosed SLE patients, and the levels increased even more after three years of diagnosis.[9] Dyslipidemia can impact SLE patients' prognosis through events linked to cardiovascular disease.[10]

This study aimed to assess the lipid profile in SLE patients and explore the relationship between dyslipidemia and the severity of the condition.

## METHODOLOGY

This cross-sectional study was conducted at the outpatient department (OPD) of Rheumatology, Khyber Teaching Hospital, Peshawar Pakistan, after approval from Institutional Ethical Review Committee (No.257/DME/KMC) from May 2023 to February 2024.

**Correspondence: Dr Alam Zeb,** Department of Rheumatology, Khyber Teaching Hospital, Peshawar Pakistan

*Received: 06 Mar 2024; revision received: 23 Jul 2024; accepted: 29 Jul 2024*

**Inclusion Criteria**: Patients of either gender aged above 18 years diagnosed with Systemic Lupus Erythematosus (SLE) according to the American College of Rheumatology criteria were included.

**Exclusion Criteria**: Patients with a history of liver or thyroid illness, diabetes mellitus, coronary arterial disease (CAD), essential hypertension, chronic kidney disease (CKD) or familial hyperlipidaemia and those receiving medication to decrease their cholesterol, SLE patients with lupus nephritis, and pregnant or breastfeeding women were excluded.

The total sample size was calculated using the WHO calculator, came to 78 patients, based on minimum of 30% prevalence of dyslipidemia in SLE.[12] The sampling technique was nonprobability consecutive. Informed consent was taken, and data was collected using non-probability consecutive sampling.

Severity of SLE was assessed using SLE Disease Activity Index. A SLEDAI score greater than or equal to 3 points and a greater than or equal to 1-point increase in patient global assessment (PGA) is considered mild/moderate flare (range 0-3). A SLEDAI score greater than or equal to 12 points, and a greater than or equal to 2.5-point increase in PGA is included for severe flare.[10] Dyslipidemia was defined by elevated total cholesterol (> 200 mg/dL), TG (> 200 mg/dL), low-density lipoprotein (LDL; > 100 mg/dL), and decreased HDL (< 40 mg/dL) levels in the serum samples.

Patients were admitted in ward after employing strict inclusion criteria. Data was collected using a checklist that included demographic characteristics, disease duration, medications, and disease activity measured by SLEDAI. Blood samples (10 ml from cubital vein under sterile environment) were collected after overnight fasting, and serum levels of cholesterol, triglyceride, HDL, and LDL were measured in hospital laboratory.

Statistical analysis was done using Statistical Package for Social Sciences version 23. Apart from descriptive statistics, Spearman correlation analysis was used to examine relationship between SLEDAI categories with lipid profile. A *p*-value of ≤0.05 was considered statistically significant.

## RESULTS

Of 78 patients included, the median age of patients 29.0 years with an interquartile range from 21.0 to 37.0 years. Fifteen (19.3%) were male and 63(80.7%) were female. The mean duration of disease was 6.2±4.7 years. Sixty-eight individuals were non-smokers (87.2%), while a minority reported smoking 10(12.8%). Family history was predominantly negative 70(89.7%), with only 8(10.3%) individuals reporting a positive history. Regarding lipid profiles, the majority had normal total cholesterol (n=66, 84.6%) and LDL levels (n=64, 82.1%), while a smaller number showed elevated levels of total cholesterol (n=12, 15.4%) and LDL (n=14, 17.9%). Interestingly, triglyceride levels were raised in 66(84.6%) participants, with only 12(15.4%) individuals showing normal levels. All participants (100%) had low HDL levels. The SLEDAI scores indicated that most participants had moderate disease activity (n=54, 69.2%), while equal numbers showed mild and severe activity 12(15.4%) each, as summarized in Table-I.

**Table-I: Clinicodemographic Variables of Patients (n=78)**

| Variable | Category | n(%) |
|---|---|---|
| Gender | Male | 15(19.3%) |
| | Female | 63(80.7%) |
| Smoking | Yes | 10(12.8%) |
| | No | 68(87.2%) |
| Family History | Positive | 8(10.3%) |
| | Negative | 70(89.7%) |
| Total Cholesterol | Normal (<200 mg/dL) | 66(84.6%) |
| | Raised (>200 mg/dL) | 12(15.4%) |
| LDL | Normal (<100 mg/dL) | 64(82.1%) |
| | Above (>100 mg/dL) | 14(17.9%) |
| Triglyceride | Normal (<150 mg/dL) | 12(15.4%) |
| | Raised (>150 mg/dL) | 66(84.6%) |
| HDL | Low | 78(100%) |
| SLEDAI | Mild | 12(15.4%) |
| | Moderate | 54(69.2%) |
| | Severe | 12(15.4%) |

**LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, SLEDAI: Systemic Lupus Erythematosus Disease Activity Index*

The normality tests for lipid profile parameters, including Total Cholesterol (TC), Triglycerides (TG), LDL Cholesterol (LDL), and HDL Cholesterol (HDL), indicated non-normal distributions, with all *p*-values <0.05 in both Kolmogorov-Smirnov and Shapiro-Wilk tests. The median values for TC, TG, LDL, and HDL were 158.0 mg/dL, 225.0 mg/dL, 78.0 mg/dL, and 28.0 mg/dL, respectively (Summarized in Table-II).

**Table-II: Lipid Profile of Patients (n=78)**

| Parameter | Median | 25th Percentile | 75th Percentile |
|---|---|---|---|
| TC | 158.0 | 121.0 | 198.0 |
| TG | 225.0 | 180.0 | 309.0 |
| LDL | 78.0 | 60.0 | 112.0 |
| HDL | 28.0 | 25.0 | 40.0 |

**TC: Total Cholesterol, TG: Triglycerides, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein*

The relationship between various parameters and the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) was examined. Total Cholesterol showed a significant positive correlation (r=0.702, *p*=0.000), indicating a strong association with disease activity. Triglyceride levels exhibited a significant positive correlation as well (r=0.238, *p*=0.038), suggesting a potential link with SLEDAI. Low-Density Lipid (LDL) demonstrated a significant positive correlation (r=0.633, *p*=0.000), while High-Density Lipid (HDL) exhibited a non-significant correlation (r=0.074, *p*=0.519). Duration of Disease displayed a non-significant negative correlation (r=-0.045, *p*=0.699), implying a minimal impact on disease activity. These findings underscore the significance of lipid parameters in relation to SLE disease activity, with total cholesterol, triglycerides, and LDL showing notable associations. The correlation matrixes are represented in the Figure.

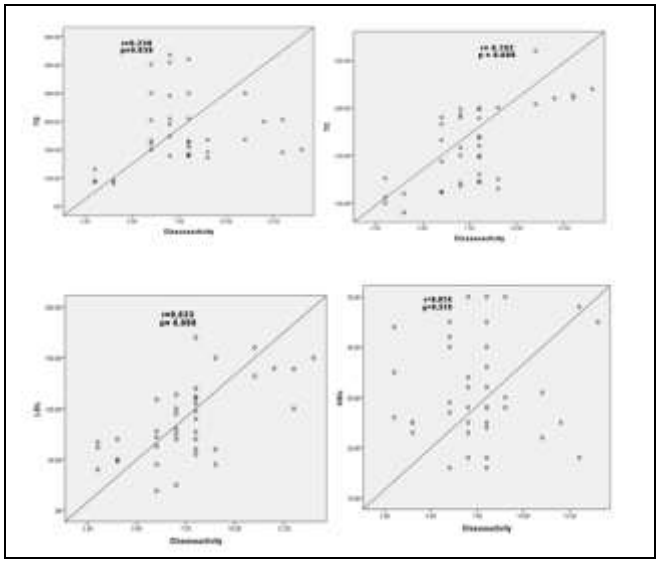

**Figure: Correlation Matrix of Lipid Profile with Systemic Lupus Erythematosus Disease Activity Index (n=78)**

## DISCUSSION

The mean age of patients in our study was 29.89±9.94 years, which aligns with previous research. Haung *et al.,* reported a mean age of 33.03±1.21 years, while Zhou B *et al.,* found that SLE patients with dyslipidemia had a mean age of 27.6±4.2 years.[1,2] In our study, 15(19.3%) patients were male and 63(80.7%) were female, similar to the gender distribution reported by Yaun J *et al.,* who observed 40(87%) female and 6(13%) male patients.[13] The predominance of SLE in young women observed in our study and others is likely attributable to a combination of hormonal influences (particularly estrogen), genetic factors related to the X chromosome, and potential environmental triggers that interact with these biological predispositions during reproductive years.

The mean SLEDAI in our study was 7.56±2.61, which is similar to other studies (13.8±5.9, 12.87±2.81.[4,13]

In our study, the mean lipid profile parameters were: total cholesterol (TC) = 158.2±123.2 mg/dL, low-density lipoprotein (LDL) = 255.7±42.4 mg/dL, triglycerides (TG) = 87.8±37.6 mg/dL, and high-density lipoprotein (HDL) = 31.4±10.1 mg/dL. These results indicate that while total cholesterol was mostly within normal range, LDL and TG levels were elevated, and HDL levels were lower than normal in the majority of patients. One study reported similar findings, except for higher TC levels compared to our study.[14] These lipid profile results are crucial as they highlight the increased cardiovascular risk in SLE patients, emphasizing the need for regular monitoring and early intervention strategies to prevent atherosclerosis and reduce morbidity and mortality in this population.

In our study the most frequent medication used for SLE were immunosuppressants (41%) and C-DMARDS (23.1%). While one study reported that majority of patients were on Hydroxychloroquine (71%).[15] The difference in medication patterns may be due to variations in disease severity, treatment guidelines, drug availability, physician preferences, study timeframes, or patient characteristics between the two study populations.

The study also highlighted significant positive correlations among SLEDAI and Total Cholesterol, Triglycerides, and LDL, indicating their potential relevance as indicators of disease activity. These findings are supported by other studies, which showed a significant correlation between SLEDAI and TC, TG and LDL.[16-18]

## LIMITATION OF STUDY

A small sample size, and being a single-centre study limits the generalizability of our findings.

## CONCLUSION

The study demonstrated a significant correlation between Systemic Lupus Erythematosus disease activity measured through Systemic Lupus Erythematosus Disease Activity Index and Total Cholesterol, Low Density Lipoprotein and Triglycerides.

**Conflict of Interest:** None.

**Funding Source:** None.

**Authors' Contribution**

Following authors have made substantial contributions to the manuscript as under:

MI & HA: Data acquisition, data analysis, critical review, approval of the final version to be published.

AZ & NU: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

UA & AK: Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

## REFERENCES


1. Huang S, Zhang Z, Cui Y, Yao G, Ma X, Zhang H. Dyslipidemia is associated with inflammation and organ involvement in systemic lupus erythematosus. Clin Rheumatol 2023; 1: 1-8. https://doi.org/10.1007/s10067-023-06539-2
2. Rezaeian M, Abbasi A, Abbasifard M. Association between Abnormal Lipid Levels and Systemic Lupus Erythematosus. Rheumatol Res 2021; 6(2): 81–86. https://doi.org/10.22631/rr.2021.69997.1118
3. Sajjad S, Farman S, Ahmed M, Mir N, Azeem B. Frequency of Dyslipidemia in patients with Lupus Nephritis. Pak J Med Sci 2017: 33(2): 358. https://doi.org/10.12669/pjms.332.12410
4. Zhou B, Xia Y, She J. Dysregulated serum lipid profile and its correlation to disease activity in young female adults diagnosed with systemic lupus erythematosus: a cross-sectional study. Lipids Health Dis 2020; 19(1): 40. https://doi.org/10.1186/s12944-020-01232-8
5. Kumarasamy R, Rajendran B. Study on Lupus Pattern of Dyslipidemia in Systemic Erythematosus. Sch Int J Biochem 2019; 2(2): 31-35. https://doi.org/10.36348/sijb.2019.v02i02.004
6. Ding Y, Fan S, Tang Y, He M, Ren M, Shi Y, et al. The Association between Blood Lipids and Systemic Lupus Erythematosus: A Two-Sample Mendelian Randomization. Res Metab 2023; 13(1): 27. https://doi.org/10.3390/metabo13010027
7. Nisar A, Rasheed U, Aziz W, Farooqi AZ. Prevalence of dyslipidemias in autoimmune rheumatic diseases. J Coll Physicians Surg Pak 2012; 22(4): 235–239.
8. Ali M, Mostafa S, Sayed A. Clinical significance of lipid profile in systemic lupus erythematosus patients: Relation to disease activity and therapeutic potential of drugs. Egypt Rheumatol 2017; 39(2): 93–98. https://doi.org/10.1016/j.ejr.2016.08.004
9. Chen W, Wang Q, Zhou B, Zhang L, Zhu H. Lipid Metabolism Profiles in Rheumatic Diseases. Front Pharmacol 2021; 12: 643520. https://doi.org/10.3389/fphar.2021.643520
10. Tselios K, Koumaras C, Gladman DD, Urowitz MB. Dyslipidemia in systemic lupus erythematosus: just comorbidity? Semin Arthritis Rheum 2016; 45(5): 604–610. https://doi.org/10.1016/j.semarthrit.2015.10.010
11. Khalil F, Rafat MN, Beltagy NTE, Gaber HA. Study of Dyslipidemia in Patients with Systemic Lupus Erythematosus and its Correlation to Disease Activity. Egypt J Hosp Med 2018; 73(5): 6586–6595. https://doi.org/10.21608/EJHM.2018.15588
12. Arora S, Isenberg DA, Castrejon I. Measures of Adult Systemic Lupus Erythematosus: Disease Activity and Damage. Arthritis Care Res 2020; 72(S10): 27–46. https://doi.org/10.1002/acr.24221
13. Yuan J, Li LI, Wang Z, Song W, Zhang Z. Dyslipidemia in patients with systemic lupus erythematosus: Association with disease activity and B-type natriuretic peptide levels. Biomed Rep 2016; 4(1): 68-72. https://doi.org/10.3892/br.2015.544
14. Szabó MZ, Szodoray P, Kiss E. (2017). Dyslipidemia in systemic lupus erythematosus. Immunologic Research 2017; 65(2): 543–550. https://doi.org/10.1007/s12026-016-8892-9
15. Wang M, Huang S, Lin X, Wen C, He Z, Huang L. The causal relationship between blood lipids and systemic lupus erythematosus risk: a bidirectional two-sample mendelian randomization study. Front Genet 2022; 13: 858653. https://doi.org/10.3389/fgene.2022.858653
16. Atta AM, Silva JP, Santiago MB, Oliveira IS, Oliveira RC, Sousa Atta ML. Clinical and laboratory aspects of dyslipidaemia in Brazilian women with systemic lupus erythematosus. Clin Rheumatol 2018; 37: 1539-1546. https://doi.org/10.1007/s10067-018-4051-0
17. George J, Harats D, Gilburd B, Levy Y, Langevitz P, Shoenfeld Y. Atherosclerosis-related markers in systemic lupus erythematosus patients: the role of humoral immunity in enhanced atherogenesis. Lupus 1999; 8(3): 220-226. https://doi.org/10.1191/096120399678847597
18. Harmouche H, Ammouri W, Maamar M, Adnaoui M, Cacoub P. Atherosclerosis in systemic lupus erythematosus. Presse Medicale 2014; 43(10Pt1): 1034-1047. https://doi.org/10.1016/j.lpm.2014.01.021