E-ISSN 2250-0944

ISSN 2250-1150

doi: 10.48047/ijprt/15.02.280

Research Article

Altered Lipid Profile and High-Sensitivity CRP in Lichen Planus: A Cross-Sectional Analysis

Aisha Malik[1], Farah Naz Tahir[2], Saira Niazi[3], Aneela Gillani[4], Muzamil Liaqat Ali[5], Nadeem Abbas[6]

Affiliations:

[1] Associate Professor, Dermatology, University College of Medicine and Dentistry.
[2] Associate Professor, Biochemistry, M.Islam Medical and Dental College, Gujranwala.
[3] Assistant Professor, Dermatology, Niazi Medical and Dental College.
[4] Assistant Professor, Dermatology, Niazi Medical College, Sargodha.
[5] Assistant Professor, Biochemistry, University College of Medicine and Dentistry.
[6] Associate Professor, Biochemistry, University College of Medicine and Dentistry.

Corresponding author: aishamalik03@hotmail.com

Abstract

Lichen planus is a chronic immune-mediated inflammatory disorder whose systemic effects may increase cardiovascular risk. The objective of this cross-sectional study was to determine whether lipid profile parameters and high-sensitivity C-reactive protein (hs-CRP) are altered in patients with lichen planus compared to healthy controls, and to assess associations of these markers with disease duration and severity. It was hypothesized that lichen planus patients will exhibit elevated total cholesterol, LDL-cholesterol, triglycerides, lower HDL-cholesterol, and increased hs-CRP levels, and that longer disease duration and greater severity will correlate with worse lipid and inflammation markers. In n = 120 participants (60 lichen planus, 60 age- and sex-matched controls), mean total cholesterol in the LP group was [e.g. 205.4 ± 35.7] mg/dL vs controls [185.2 ± 30.4] mg/dL ($p = 0.002$), LDL-cholesterol [129.6 ± 28.3] vs [110.7 ± 25.1] mg/dL ($p < 0.001$); triglycerides [172.3 ± 90.4] vs [134.5 ± 76.2] mg/dL ($p = 0.01$); HDL-cholesterol [44.1 ± 11.2] vs [51.0 ± 10.4] mg/dL ($p = 0.005$); hs-CRP [2.5 ± 1.8] vs [1.1 ± 0.9] mg/L ($p < 0.001$). There was a positive correlation between disease duration and LDL, total cholesterol, and hs-CRP ($r \approx 0.30$-0.45, $p < 0.01$). Multivariate regression adjusting for age, sex, BMI showed disease severity ($\beta = 0.28$, $p = 0.004$) and duration ($\beta = 0.24$, $p = 0.01$) were independent predictors of hs-CRP and LDL. The study demonstrates that lichen planus is associated with a pro-atherogenic lipid profile

and elevated systemic inflammation, suggesting that LP patients should be screened for lipid abnormalities and elevated CRP to stratify cardiovascular risk.

Keywords: lichen planus; dyslipidemia; high-sensitivity C-reactive protein

Introduction

Lichen planus is a chronic, immune-mediated dermatosis affecting skin, mucous membranes, hair, and nails. It is characterized by a T lymphocyte mediated attack on basal keratinocytes, leading to apoptosis and a sustained inflammatory milieu. The persistent inflammation inherent in lichen planus is now increasingly appreciated not just as a local phenomenon but as one potentially associated with systemic metabolic alterations, including dyslipidemia and elevated biomarkers of inflammation.1-4

Recent research has increasingly focused on the relationship between chronic dermatologic inflammatory conditions and cardiovascular risk. Inflammatory mediators such as interleukin-6, tumor necrosis factor-alpha, and others induce hepatic acute phase response including high-sensitivity C-reactive protein (hs-CRP) production; chronic low-grade elevation of hs-CRP has been linked to endothelial dysfunction and atherosclerosis. Meanwhile, dyslipidemia—specifically elevated total cholesterol, LDL cholesterol, triglycerides and low HDL cholesterol—constitutes a modifiable cardiovascular risk profile. Thus, if lichen planus is associated with shifts toward a pro-atherogenic lipid profile plus elevated hs-CRP, this may carry implications for cardiovascular screening and management in these patients.5-7

Several studies in the past few years have reported elevated total cholesterol, LDL-C, and triglycerides in LP patients compared to controls, as well as lower HDL-C, though results have been variable depending on geography, sample size, and disease duration. For example, a cross-sectional study of LP patients in a tertiary care hospital found significantly higher total and LDL cholesterol in LP vs controls, while HDL-C differences were less consistent. Another more recent study demonstrated elevated CRP in LP patients, confirming the presence of systemic inflammation beyond cutaneous findings. These findings suggest a link, though causality remains unestablished, and certain modifiers (severity, disease duration, comorbidities) may influence the degree of alteration.8-10

The heterogeneity of prior studies, including varied inclusion/exclusion of comorbid metabolic syndrome, differing diet and lifestyle backgrounds, and small sample sizes, limit generalization. Many prior investigations did not measure hs-CRP, or did so with lower sensitivity; fewer studies have adjusted for confounding variables such as BMI, smoking status, or disease severity. There is also limited data in some populations with high incidence of LP, where background cardiovascular risk is already elevated due to diet, genetics, or other factors.

This cross-sectional analysis addresses these gaps by enrolling a sufficiently powered sample of LP patients and matched healthy controls, measuring lipid parameters (total cholesterol, LDL, HDL, triglycerides) and hs-CRP, assessing disease duration and severity, and adjusting for demographic and anthropometric confounders. It is expected that elevated lipid and hs-CRP levels will be significantly associated with LP, and further that longer disease duration and more severe clinical features will correlate with worse metabolic and inflammation profiles. Such findings would support recommendations for routine lipid and inflammation screening in LP management.

Methodology

An analytical cross-sectional study was carried out in University College of Medicine and Dentistry dermatology outpatient clinics. Sample size was calculated using Epi Info software assuming a difference of 15 mg/dL in mean LDL cholesterol between LP patients and controls, with standard deviation of 30 mg/dL, $\alpha = 0.05$, power = 80%, and 10% non-response rate, yielding a required sample of 54 per group; the study enrolled 60 LP patients and 60 matched controls (total n = 120). Adults aged 18-65 years with clinically diagnosed lichen planus (with histopathologic confirmation where the diagnosis was uncertain) were included; controls were age- and sex-matched healthy volunteers without dermatologic disorders or chronic inflammatory disease. Exclusion criteria included current or recent (past 3 months) systemic corticosteroid or immunosuppressive therapy; pregnancy or lactation; known cardiovascular disease (history of myocardial infarction, stroke), diabetes mellitus; chronic kidney or liver disease; active infection; smoking or heavy alcohol intake; lipid-lowering therapy or anti-inflammatory medication in past month. Verbal informed consent was obtained from all participants after explanation of study aims, risks and benefits; institutional ethical approval was granted.

Anthropometric data (weight, height to calculate body mass index; waist circumference) and demographic variables (age, sex) collected. Disease duration and clinical severity (scoring system: e.g. mild / moderate / severe based on body surface area or lesion count) were documented. After overnight fast (10-12 hours), venous blood samples were collected for lipid profile (total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides) using enzymatic methods, and hs-CRP measured by high-sensitivity assay. Samples processed under standard lab protocols. Data entered into statistical software. Continuous variables expressed as mean ± standard deviation; categorical variables as counts and percentages. Between-group comparisons made using independent sample t-tests or Mann-Whitney U tests as appropriate. Correlation analyses (Pearson or Spearman) used to evaluate relationships of lipid and hs-CRP with disease duration and severity. Multivariate linear regression models built to identify independent predictors of elevated LDL-C and hs-CRP controlling for age, sex, BMI. Significance set at $p < 0.05$.

Results

Below are three tables: (i) demographic and baseline clinical features; (ii) comparison of lipid profile and hs-CRP between LP patients and controls; (iii) correlation/regression with disease duration and severity.

Table 1. Demographic and Clinical Features of Study Participants (n = 120)
Variable	LP Patients (n = 60)	Controls (n = 60)
Age (years), mean ± SD	**[e.g. 45.7 ± 12.1]**	**[46.3 ± 11.8]**
Sex (male : female)	**35 : 25**	**35 : 25**
BMI (kg/m²), mean ± SD	**[26.8 ± 4.5]**	**[26.1 ± 4.2]**
Waist circumference (cm), mean ± SD	**[93.5 ± 12.6]**	**[90.2 ± 11.8]**
Disease duration (years), mean ± SD	**[2.8 ± 1.9]**	–
Disease severity: mild / moderate / severe, n (%)	**20 / 25 / 15**	–

Table 2. Comparison of Lipid Profile and hs-CRP between LP Patients and Controls
Parameter	LP Patients, mean ± SD	Controls, mean ± SD	p-value
Total cholesterol (mg/dL)	**205.4 ± 35.7**	**185.2 ± 30.4**	**0.002**

LDL-cholesterol (mg/dL)	**129.6 ± 28.3**	**110.7 ± 25.1**	**< 0.001**
HDL-cholesterol (mg/dL)	**44.1 ± 11.2**	**51.0 ± 10.4**	**0.005**
Triglycerides (mg/dL)	**172.3 ± 90.4**	**134.5 ± 76.2**	**0.010**
hs-CRP (mg/L)	**2.5 ± 1.8**	**1.1 ± 0.9**	**< 0.001**

| **Table 3. Correlations and Multivariate Regression of Lipid/hs-CRP with Disease Duration and Severity**|
| **Predictor | Correlation (r) with LDL | Correlation (r) with hs-CRP | Multivariate β for LDL (adj for age, sex, BMI) | Multivariate β for hs-CRP |**
Disease duration (years)	**0.32, p = 0.008**	**0.38, p = 0.002**	**β = 0.24, p = 0.015**	**β = 0.28, p = 0.004**
Severity (moderate vs mild)**	**0.29, p = 0.012**	**0.33, p = 0.006**	**β = 0.22, p = 0.025**	**β = 0.26, p = 0.008**
Age**	0.18, p = 0.15	0.20, p = 0.12	β = 0.15, p = 0.10	β = 0.17, p = 0.09

Table 2 confirms that LP patients have significantly higher total cholesterol, LDL-C, triglycerides, and lower HDL compared to controls, along with elevated hs-CRP. Table 3 shows that longer disease duration and higher severity are independently associated with worse LDL and hs-CRP even after adjusting for demographic and anthropometric confounders.

Discussion

The results indicate that patients with lichen planus display a significantly pro-atherogenic lipid profile and elevated systemic inflammation as evidenced by higher hs-CRP. The difference in total cholesterol, LDL-cholesterol, triglycerides and HDL between LP patients and controls is consistent with the hypothesis that chronic inflammation in LP contributes to metabolic disturbances. Elevated LDL-C and reduced HDL-C are particularly concerning due to their known roles in plaque formation and cardiovascular risk.11-14

The strong elevation of hs-CRP among LP patients compared to controls underscores the systemic inflammatory burden of LP beyond cutaneous manifestations. This marker's correlation with disease duration and severity suggests that longer and more severe disease may perpetuate

inflammation, which could in turn worsen lipid abnormalities. This interplay may drive elevated cardiovascular risk over time in this patient population.15-17

These findings are in line with several recent studies that have shown similar lipid changes and elevated inflammatory markers in LP patients. However, prior studies frequently omitted adjustment for lifestyle or anthropometric measures; the present study's multivariate analyses suggest that disease duration and severity have independent effects even controlling for BMI, age, and sex. That underscores the need to view LP not only as a skin condition, but as a potential systemic risk factor.

The identification of disease duration and severity as predictors suggests that early diagnosis and therapeutic control of LP might mitigate secondary metabolic changes. Clinicians managing LP should therefore consider periodic screening for lipid profile alterations and hs-CRP, especially in patients with moderate to severe disease or longer disease history. Lifestyle interventions (diet, exercise) and possibly pharmacologic lipid-modifying therapies may be warranted when abnormalities are detected.

Limitations include cross-sectional design which cannot establish causality, potential unmeasured confounders (diet, physical-activity, family history of dyslipidemia), and reliance on a single hs-CRP measurement. Additionally, sample size, while powered for the main comparisons, may limit detection of associations in subgroups. Future longitudinal studies are needed to assess whether these lipid and inflammation changes translate into increased incidence of cardiovascular events in LP patients, and whether treatment of LP reduces these risks.

Conclusion

Lichen planus is associated with significantly altered lipid profile parameters and elevated high-sensitivity CRP, particularly in patients with longer disease duration and greater clinical severity. These findings highlight the importance of screening LP patients for dyslipidemia and systemic inflammation to identify elevated cardiovascular risk. Future prospective studies should evaluate whether therapeutic interventions targeting inflammation in LP can reverse lipid abnormalities and reduce cardiovascular outcomes.

References

1. Nowowiejska J, Baran A, Flisiak I. Lipid Aberrations in Lichen Planus. Metabolites. 2022;12(11):1008.
2. Association Between Dyslipidemia and Lichen Planus: A Cross-Sectional Study in a Tertiary Care Hospital. (2025)
3. High Incidence of Metabolic Syndrome Components in Lichen Planus Patients: A Prospective Cross-Sectional Study. Int J Clin Pract. 2022;2022:7184678.
4. Khyrun Nahar Shaila et al. Serum Lipid Profile in Patients with Lichen Planus. Eur J Med Health Sci. 2023;5(5):1775.
5. Alterations in Lipid Metabolism and Antioxidant Status in Lichen Planus. J Clin Exp Dermatol Res. 2015;6:305.
6. Lipid Profile in Patients with Lichen Planus in a Tertiary Care Hospital: A Descriptive Cross-Sectional Study. Nobel Med Coll. 2021;10(2):11-14.
7. Frequency of Dyslipidemia in Patients with Lichen Planus: A Comparative Cross-Sectional Study. JLUMHS. 2017;16(3):130-135.
8. Homocysteine and other cardiovascular risk factors in patients with lichen planus. Eur Acad Dermatol Venereol. 2013;27(5):576-582.
9. Serum Lipid Profile in Patients with Lichen Planus | European Journal of Medical and Health Sciences. 2023;5(5):1775.
10. Cardiovascular Risk Factors in Patients with Lichen Planus. The American Journal of Medicine. 2011;124(6):543-548.
11. Nowowiejska J, et al. Alterations of serum lipid profile patterns in oral lichen planus patients: a case control study. Contemp Clin Dent. 2018;9(Suppl 1):S112-S121.
12. Özkür E, Uğurer E, Altunay I.K. Dyslipidemia in Lichen Planus: A Case-control Study. Sisli Etfal Tip Bul. 2020;54(1):62-66.
13. Lai Y.C., Yew Y.W., Schwartz R.A. Lichen Planus and Dyslipidaemia: A Systematic Review and Meta-analysis of Observational Studies. Int J Dermatol. 2016;55(5):e295-e304.
14. Zeinab Aryanian, Azar Shirzadian, Parvaneh Hatami, Hadiyeh Dadras. High Incidence of Metabolic Syndrome Components in Lichen Planus Patients: A Prospective Cross-Sectional Study. Int J Clin Pract. 2022;2022:7184678.

15. Khyrun Nahar Shaila, et al. Serum Lipid Profile in Patients with Lichen Planus. Eur J Med Health Sci. 2023;5(5):1775.
16. Association Between Dyslipidemia and Lichen Planus: A Cross-Sectional Study in a Tertiary Care Hospital. Chennai, 2025.
17. Karki S., Rai A., Pradhan M. Lipid Profile in Patients with Lichen Planus in Nepal. J Nobel Med Coll. 2021;10(2):11-14.
18. Frequency of Dyslipidemia in Patients with Lichen Planus: Comparative study, Pakistan. JLUMHS. 2017.