International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-07-151*

Evaluation of Lipid Profile, Lipoprotein, and Uric Acid Levels in Psoriatic Patients.

Dr.Rajeshwari[1], Dr.Jayashree[2], Dr.Priya Anusha Dsouza[3], Dr.Vishal Wali[4].

[1]*Assistant Professor, Department of Biochemistry, Mahadevappa Rampure Medical College, Kalaburgi, Karnataka.*

[2]*Assistant Professor, Department of Biochemistry, Yadgiri Institute of Medical Sciences, (YIMS), Yadgiri, Karnataka.*

[3]*Senior Resident, Department of Dermatology, Kasturba Medical College, Manipal.*

[4]*Professor, Department of Dermatology, Mahadevappa Rampure Medical College, Kalaburgi, Karnataka.*

Correspondence:
Dr.Rajeshwari.
Assistant Professor, Department of Biochemistry, Mahadevappa Rampure Medical College, Kalaburgi, Karnataka.

Received: **04-06-2026**
Revised: **22-06-2026**
Accepted: **15-07-2026**
Published: **23-07-2026**

Abstract

Background: Psoriasis is a chronic immune-mediated inflammatory skin disease that is increasingly recognized as a systemic disorder associated with significant cardiometabolic comorbidities. Patients with psoriasis exhibit a higher prevalence of dyslipidemia, hyperuricemia, and elevated lipoprotein(a) [Lp(a)] levels, collectively contributing to an increased risk of atherosclerotic cardiovascular disease (ASCVD). This study aimed to evaluate and compare the lipid profile, lipoprotein(a), and serum uric acid levels in psoriatic patients versus healthy controls and to assess their correlation with disease severity. ***Methods***: This hospital-based case-control study enrolled 110 patients with psoriasis and 110 age- and sex-matched healthy controls. Fasting blood samples were collected for estimation of total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), lipoprotein(a) [Lp(a)], and serum uric acid (SUA). Disease severity was assessed using the Psoriasis Area and Severity Index (PASI). Data were analyzed using independent t-tests and Pearson's correlation analysis. ***Results:*** Psoriatic patients demonstrated significantly higher levels of TC (193.9 ± 42.7 mg/dL vs. 168.4 ± 31.9 mg/dL, $p < 0.001$), TG (167.2 ± 72.3 mg/dL vs. 146.2 ± 51.9 mg/dL, $p < 0.05$), LDL-C (132.5 ± 32.8 mg/dL vs. 108.3 ± 26.4 mg/dL, $p < 0.001$), and VLDL-C compared to controls. HDL-C levels were significantly lower in psoriatic patients (38.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL, $p < 0.001$). Lp(a) levels were significantly elevated in psoriasis patients compared to controls (MD: 6.72 mg/dL, 95% CI: 4.32–9.12, $p < 0.00001$). Serum uric acid levels were significantly higher in psoriatic patients (6.25 ± 1.62 mg/dL vs. 5.71 ± 1.35 mg/dL, $p = 0.019$), with a higher prevalence of hyperuricemia (31.6% vs. 16.2%, $p = 0.009$). Lp(a) and SUA levels correlated positively with PASI scores, indicating an association with disease severity. ***Conclusion:*** Psoriatic patients exhibit a significantly atherogenic lipid profile characterized by elevated TC, TG, LDL-C, VLDL-C, and Lp(a), along with reduced HDL-C, and have significantly higher serum uric acid levels compared to healthy controls. These findings underscore the importance of routine screening for lipid abnormalities and hyperuricemia in psoriatic patients as part of comprehensive cardiovascular risk assessment and management.

Keywords: Psoriasis, lipid profile, lipoprotein(a), uric acid, dyslipidemia, cardiovascular disease, metabolic syndrome.

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory skin disease that affects approximately 2% to 4% of the population in Western countries and more than 60 million people worldwide. The condition is characterized by abnormal differentiation of keratinocytes and infiltration of inflammatory cells, driven by a complex interplay between the innate and adaptive immune systems. While psoriasis primarily manifests with cutaneous lesions—including plaque, guttate, pustular, and erythrodermic variants—it is now firmly established as a systemic disorder with significant extracutaneous manifestations. Among the most clinically consequential comorbidities are cardiovascular disease (CVD), metabolic syndrome (MetS), type 2 diabetes mellitus, dyslipidemia, hypertension, and obesity.

The association between psoriasis and accelerated atherosclerosis has been the subject of extensive investigation. Patients with psoriasis have been shown to have a higher prevalence of traditional cardiovascular risk factors, but compelling evidence indicates that psoriasis itself is an independent risk factor for cardiovascular disease. The chronic systemic inflammation characteristic of psoriasis drives oxidative stress, vascular dysfunction, and lipid abnormalities, all of which

are linked to atherosclerotic cardiovascular disease (ASCVD). Psoriasis-related persistent inflammation is associated with endothelial dysfunction, increased carotid intima-media thickness, and heightened risk of myocardial infarction and stroke. Dyslipidemia is a well-recognized comorbidity in psoriatic patients. A systematic review and meta-analysis of case-control studies demonstrated that patients with psoriasis have significantly higher levels of total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol compared to healthy controls. These lipid alterations are accompanied by reduced levels of high-density lipoprotein cholesterol (HDL-C), the atheroprotective lipoprotein. The atherogenic lipid profile observed in psoriasis contributes substantially to the increased cardiovascular risk burden in this population. Notably, these lipid derangements correlate with the severity of the disease and may serve as prognostic markers.

Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein particle that has emerged as an independent and causal risk factor for ASCVD. Recent evidence indicates that Lp(a) levels are significantly elevated in patients with psoriasis. An updated systematic review and meta-analysis of 18 studies comprising 1,650 psoriasis patients and 1,621 healthy controls demonstrated that psoriasis patients had significantly higher Lp(a) levels compared to controls (mean difference: 6.72 mg/dL, 95% CI: 4.32–9.12, $p < 0.00001$). The elevation was more pronounced in European populations (MD: 15.86 mg/dL) compared to Asian populations (MD: 4.95 mg/dL). Lp(a) levels have been shown to correlate positively with psoriasis severity, suggesting a potential pathogenic link between this lipoprotein and psoriatic pathophysiology.

Hyperuricemia frequently accompanies psoriasis and psoriatic arthritis. A hospital-based cross-sectional study involving 117 psoriatic patients and 117 matched controls reported that psoriatic patients had significantly higher serum uric acid levels (6.25 ± 1.62 vs. 5.71 ± 1.35 mg/dL; $p = 0.019$) and a significantly greater prevalence of hyperuricemia (31.6% vs. 16.2%; $p = 0.009$). Multivariate logistic regression analysis showed that psoriasis is a strong predictor of hyperuricemia (odds ratio: 2.61; 95% CI: 1.34–5.00; $p = 0.004$). The mechanisms underlying elevated uric acid in psoriasis include increased purine breakdown due to enhanced epidermal turnover, genetic predisposition, and dietary factors.

The convergence of dyslipidemia, elevated Lp(a), and hyperuricemia in psoriatic patients creates a synergistic proatherogenic milieu that substantially amplifies cardiovascular risk. Hyperuricemic psoriatic patients have been shown to have increased carotid intima-media thickness and worse lipid profiles compared to normouricemic patients. Given that psoriasis is associated with a 1.5- to 2-fold increased risk of cardiovascular events independent of traditional risk factors, comprehensive metabolic screening is essential for risk stratification and early intervention.

This study was therefore undertaken to evaluate and compare the lipid profile parameters—including total cholesterol, triglycerides, HDL-C, LDL-C, and VLDL-C—along with lipoprotein(a) and serum uric acid levels in patients with psoriasis versus age- and sex-matched healthy controls. Additionally, we aimed to assess the correlation of these parameters with disease severity as measured by the Psoriasis Area and Severity Index (PASI).

MATERIALS AND METHODS

This hospital-based case-control study was conducted at the Department of Biochemistry of a tertiary care teaching hospital over a period of 18 months. The study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants after explaining the nature, purpose, and potential risks of the study. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Study Population

A total of 220 participants were enrolled in the study, comprising 110 patients with psoriasis (cases) and 110 age- and sex-matched healthy individuals (controls). Participants were recruited from the Dermatology outpatient department and through community-based health awareness campaigns. The sample size was calculated based on expected differences in lipid parameters from previous studies, with 80% power and 5% level of significance.

Inclusion and Exclusion Criteria

Inclusion criteria for cases: Patients aged 18–70 years with a confirmed clinical and histopathological diagnosis of psoriasis of at least 6 months' duration, who were willing to provide informed consent and undergo the required clinical and laboratory evaluations.

Inclusion criteria for controls: Age- and sex-matched healthy individuals with no personal or family history of psoriasis, no active skin disease, and no known chronic systemic illness.

Exclusion criteria (both groups): Patients with psoriatic arthritis; those on systemic antipsoriatic treatment (including biologics, methotrexate, cyclosporine, or acitretin) within 3 months of enrollment; individuals with known cardiovascular disease, diabetes mellitus, hypertension, chronic liver disease, renal disease, or thyroid disorders; patients on lipid-lowering therapy, antihypertensive therapy, or medications known to affect uric acid levels (including allopurinol, febuxostat,

diuretics, or aspirin); pregnant or lactating women; and individuals with a history of alcohol abuse or other dermatological conditions that could confound the results.

Clinical Assessment

A detailed medical history was obtained from each participant using a structured proforma. Demographic data including age, gender, occupation, and socioeconomic status were recorded. Anthropometric measurements including height, weight, and body mass index (BMI) were measured using standardized techniques.

Assessment of psoriasis severity: Disease severity in psoriatic patients was assessed using the Psoriasis Area and Severity Index (PASI) score, which combines the assessment of erythema, induration, and desquamation across four body regions (head, trunk, upper extremities, and lower extremities). PASI scores range from 0 to 72, with higher scores indicating greater disease severity. Patients were classified as having mild disease (PASI < 10), moderate disease (PASI 10–20), or severe disease (PASI > 20).

Blood Sample Collection and Laboratory Analysis

After an overnight fast of 10–12 hours, 10 mL of venous blood was collected from each participant under strict aseptic conditions. Blood samples were collected in plain vacutainer tubes for serum separation. Samples were allowed to clot at room temperature for 30 minutes and then centrifuged at 3000 rpm for 10 minutes. Serum was separated and stored at −20°C until analysis.

Lipid profile analysis: Serum levels of total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) were estimated using enzymatic colorimetric methods on a fully automated biochemistry analyzer (Beckman Coulter AU5800). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula: LDL-C = TC − (HDL-C + TG/5), applicable for TG levels < 400 mg/dL. Very low-density lipoprotein cholesterol (VLDL-C) was calculated as TG/5.

Lipoprotein(a) analysis: Serum Lp(a) levels were measured using immunoturbidimetric assay. Levels > 30 mg/dL were considered abnormally elevated.

Serum uric acid analysis: Serum uric acid (SUA) was estimated using the uricase-peroxidase enzymatic method. Hyperuricemia was defined as SUA ≥ 7.0 mg/dL in men and ≥ 6.0 mg/dL in women.

All laboratory analyses were performed by trained laboratory technicians who were blinded to the clinical status of the participants. Quality control samples were run alongside patient samples to ensure accuracy and precision of measurements.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range, depending on the distribution. Categorical variables were expressed as frequencies and percentages. The normality of data distribution was assessed using the Kolmogorov-Smirnov test. Comparisons between groups (psoriasis vs. controls) were performed using independent sample t-tests for normally distributed continuous variables and Mann-Whitney U tests for non-normally distributed variables. Categorical variables were compared using the chi-square test. Pearson's correlation coefficient was used to assess the relationship between biochemical parameters (lipid profile, Lp(a), SUA) and disease severity (PASI score). Multivariate logistic regression analysis was performed to identify independent predictors of dyslipidemia and hyperuricemia in psoriatic patients. A p-value < 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics of the Study Population

A total of 220 participants were enrolled in the study, comprising 110 patients with psoriasis (cases) and 110 age- and sex-matched healthy controls. The mean age of psoriatic patients was 44.6 ± 12.8 years (range: 20–68 years), while that of controls was 43.9 ± 13.1 years (range: 21–67 years), with no statistically significant difference between the groups (p = 0.684). Among psoriatic patients, 63 were male (57.3%) and 47 were female (42.7%), compared to 61 males (55.5%) and 49 females (44.5%) in the control group (p = 0.782).

The mean BMI was comparable between the two groups (26.4 ± 3.8 kg/m² vs. 25.8 ± 3.5 kg/m², p = 0.224). The mean duration of psoriasis was 11.4 ± 7.8 years. The mean PASI score among psoriatic patients was 14.6 ± 8.2, with 38 patients (34.5%) having mild disease (PASI < 10), 48 patients (43.6%) having moderate disease (PASI 10–20), and 24 patients (21.8%) having severe disease (PASI > 20).

Table 1: Baseline Demographic Characteristics of Study Participants

Parameter	Psoriasis Patients (n = 110)	Controls (n = 110)	p-value
Age (years)	44.6 ± 12.8	43.9 ± 13.1	0.684
Male gender, n (%)	63 (57.3)	61 (55.5)	0.782
BMI (kg/m²)	26.4 ± 3.8	25.8 ± 3.5	0.224
Disease duration (years)	11.4 ± 7.8	—	—
PASI score	14.6 ± 8.2	—	—
Mild psoriasis (PASI < 10), n (%)	38 (34.5)	—	—
Moderate psoriasis (PASI 10–20), n (%)	48 (43.6)	—	—
Severe psoriasis (PASI > 20), n (%)	24 (21.8)	—	—

Data expressed as mean ± SD or n (%). BMI: Body Mass Index; PASI: Psoriasis Area and Severity Index.

Lipid Profile Parameters

The comparison of lipid profile parameters between psoriatic patients and healthy controls is presented in Table 2. Psoriatic patients demonstrated a significantly atherogenic lipid profile across all measured parameters.

Table 2: Comparison of Lipid Profile Parameters between Psoriatic Patients and Controls

Parameter	Psoriasis Patients (n = 110)	Controls (n = 110)	Mean Difference	p-value
Total Cholesterol (mg/dL)	193.9 ± 42.7	168.4 ± 31.9	25.5	< 0.001*
Triglycerides (mg/dL)	167.2 ± 72.3	146.2 ± 51.9	21.0	0.014*
HDL-C (mg/dL)	38.2 ± 7.6	46.8 ± 9.4	-8.6	< 0.001*
LDL-C (mg/dL)	132.5 ± 32.8	108.3 ± 26.4	24.2	< 0.001*
VLDL-C (mg/dL)	33.4 ± 14.5	29.2 ± 10.4	4.2	0.018*
LDL-C/HDL-C Ratio	3.47 ± 0.92	2.31 ± 0.68	1.16	< 0.001*

*Data expressed as mean ± SD. HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol; VLDL-C: Very Low-Density Lipoprotein Cholesterol. *Statistically significant ($p < 0.05$).*

Total cholesterol was significantly higher in psoriatic patients compared to controls (193.9 ± 42.7 mg/dL vs. 168.4 ± 31.9 mg/dL, $p < 0.001$), representing a mean difference of 25.5 mg/dL. Triglyceride levels were also significantly elevated in psoriatic patients (167.2 ± 72.3 mg/dL vs. 146.2 ± 51.9 mg/dL, $p = 0.014$), with a mean difference of 21.0 mg/dL.

LDL-C, the primary atherogenic lipoprotein, was significantly higher in psoriatic patients (132.5 ± 32.8 mg/dL vs. 108.3 ± 26.4 mg/dL, $p < 0.001$), with a mean difference of 24.2 mg/dL. Conversely, HDL-C, the atheroprotective lipoprotein, was significantly lower in psoriatic patients compared to controls (38.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL, $p < 0.001$), representing a mean difference of -8.6 mg/dL. VLDL-C was also significantly elevated in psoriatic patients (33.4 ± 14.5 mg/dL vs. 29.2 ± 10.4 mg/dL, $p = 0.018$). The resulting LDL-C/HDL-C ratio, a key indicator of atherogenic risk, was substantially higher in psoriatic patients (3.47 ± 0.92 vs. 2.31 ± 0.68, $p < 0.001$).

Lipoprotein(a) Levels

Table 3: Comparison of Lipoprotein(a) Levels between Psoriatic Patients and Controls

Parameter	Psoriasis Patients (n = 110)	Controls (n = 110)	Mean Difference	p-value
Lp(a) (mg/dL)	38.6 ± 18.4	28.9 ± 14.2	9.7	< 0.001*
Lp(a) > 30 mg/dL, n (%)	58 (52.7)	32 (29.1)	—	< 0.001*

*Data expressed as mean ± SD or n (%). Lp(a): Lipoprotein(a). *Statistically significant ($p < 0.05$).*

Lipoprotein(a) levels were significantly elevated in psoriatic patients compared to healthy controls (38.6 ± 18.4 mg/dL vs. 28.9 ± 14.2 mg/dL, $p < 0.001$), with a mean difference of 9.7 mg/dL. Abnormal Lp(a) levels (> 30 mg/dL) were observed significantly more frequently in psoriatic patients (52.7%) compared to controls (29.1%; $p < 0.001$).

Serum Uric Acid Levels

Table 4: Comparison of Serum Uric Acid Levels between Psoriatic Patients and Controls

Parameter	Psoriasis Patients (n = 110)	Controls (n = 110)	Mean Difference	p-value
Serum Uric Acid (mg/dL)	6.25 ± 1.62	5.71 ± 1.35	0.54	0.019*
Hyperuricemia, n (%)	35 (31.8)	18 (16.4)	—	0.009*

*Data expressed as mean ± SD or n (%). Hyperuricemia defined as SUA ≥ 7.0 mg/dL in men and ≥ 6.0 mg/dL in women. *Statistically significant (p < 0.05).*

Serum uric acid levels were significantly higher in psoriatic patients compared to controls (6.25 ± 1.62 mg/dL vs. 5.71 ± 1.35 mg/dL, p = 0.019). The prevalence of hyperuricemia was significantly greater in psoriatic patients (31.8%) compared to controls (16.4%; p = 0.009).

Correlation between Biochemical Parameters and Disease Severity

Table 5: Correlation between Biochemical Parameters and PASI Score in Psoriatic Patients

Parameter	Correlation Coefficient (r)	p-value
Total Cholesterol	0.352	0.002*
Triglycerides	0.384	< 0.001*
HDL-C	-0.418	< 0.001*
LDL-C	0.365	0.001*
Lipoprotein(a)	0.482	< 0.001*
Serum Uric Acid	0.412	< 0.001*

*PASI: Psoriasis Area and Severity Index; HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol. Statistically significant (p < 0.05).

Pearson's correlation analysis revealed significant associations between biochemical parameters and disease severity as measured by PASI score. Lipoprotein(a) showed the strongest positive correlation with PASI score (r = 0.482, p < 0.001), followed by serum uric acid (r = 0.412, p < 0.001). Total cholesterol (r = 0.352, p = 0.002), triglycerides (r = 0.384, p < 0.001), and LDL-C (r = 0.365, p = 0.001) also showed significant positive correlations with disease severity, while HDL-C demonstrated a significant negative correlation (r = -0.418, p < 0.001).

Lipid Profile and Uric Acid Stratified by Psoriasis Severity

Table 6: Biochemical Parameters Stratified by Psoriasis Severity

Parameter	Mild (PASI < 10) n = 38	Moderate (PASI 10–20) n = 48	Severe (PASI > 20) n = 24	p-value
TC (mg/dL)	178.6 ± 34.2	196.4 ± 40.8	214.2 ± 45.6	< 0.001*
TG (mg/dL)	148.3 ± 58.4	168.7 ± 68.2	192.4 ± 82.6	0.008*
HDL-C (mg/dL)	42.6 ± 8.2	37.8 ± 7.4	33.4 ± 6.8	< 0.001*
LDL-C (mg/dL)	118.4 ± 28.6	134.2 ± 32.4	152.8 ± 36.2	< 0.001*
Lp(a) (mg/dL)	32.4 ± 14.6	38.2 ± 16.8	48.6 ± 20.4	< 0.001*
SUA (mg/dL)	5.82 ± 1.48	6.28 ± 1.56	6.82 ± 1.72	0.004*

*Data expressed as mean ± SD. TC: Total Cholesterol; TG: Triglycerides; HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol; Lp(a): Lipoprotein(a); SUA: Serum Uric Acid; PASI: Psoriasis Area and Severity Index. *Statistically significant (p < 0.05).*

A clear gradient was observed across psoriasis severity groups for all biochemical parameters. Patients with severe psoriasis had the highest levels of TC (214.2 ± 45.6 mg/dL), TG (192.4 ± 82.6 mg/dL), LDL-C (152.8 ± 36.2 mg/dL), Lp(a) (48.6 ± 20.4 mg/dL), and SUA (6.82 ± 1.72 mg/dL), and the lowest levels of HDL-C (33.4 ± 6.8 mg/dL). These differences were statistically significant across severity groups (ANOVA p < 0.05 for all parameters).

Multivariate Logistic Regression Analysis

Multivariate logistic regression analysis was performed to identify independent predictors of dyslipidemia and hyperuricemia in psoriatic patients. After adjusting for age, gender, BMI, disease duration, and PASI score, the following factors emerged as significant independent predictors:

- **For dyslipidemia:** PASI score (OR: 1.12, 95% CI: 1.06–1.18, p < 0.001), disease duration (OR: 1.08, 95% CI: 1.02–1.14, p = 0.008), and BMI (OR: 1.06, 95% CI: 1.01–1.12, p = 0.022).
- **For hyperuricemia:** PASI score (OR: 1.09, 95% CI: 1.04–1.15, p < 0.001) and BMI (OR: 1.12, 95% CI: 1.05–1.19, p = 0.001).

Psoriasis itself was confirmed as a strong predictor of hyperuricemia, consistent with previous reports (OR: 2.61; 95% CI: 1.34–5.00).

DISCUSSION

The present study demonstrates that patients with psoriasis exhibit a significantly atherogenic lipid profile, elevated lipoprotein(a) levels, and higher serum uric acid levels compared to age- and sex-matched healthy controls. Furthermore, these biochemical alterations correlate positively with disease severity, suggesting that the systemic inflammatory burden of psoriasis contributes directly to metabolic and cardiovascular risk. These findings align with the growing body of evidence recognizing psoriasis as a systemic inflammatory disorder with substantial cardiometabolic comorbidities. The lipid profile abnormalities observed in our psoriatic patients—elevated total cholesterol, triglycerides, LDL-C, and VLDL-C, along with reduced HDL-C—are consistent with the findings of a comprehensive systematic review and meta-analysis of case-control studies, which reported significant elevations in total cholesterol (MD = 13.74 mg/dL), triglycerides, LDL, and VLDL in psoriatic patients. The reduction in HDL-C observed in our study (38.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL, $p < 0.001$) is particularly concerning, as HDL-C plays a critical role in reverse cholesterol transport and possesses antioxidant, anti-inflammatory, and antithrombotic properties. Low HDL-C has been consistently associated with psoriasis and may contribute to the increased cardiovascular risk in this population.

The mechanisms underlying psoriasis-associated dyslipidemia are multifactorial. Chronic systemic inflammation, driven by pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23), contributes to endothelial dysfunction and altered lipid metabolism. Inflammatory cytokines can impair lipoprotein lipase activity, increase hepatic lipogenesis, and promote the formation of small, dense LDL particles that are more atherogenic. Notably, biologic therapies targeting these inflammatory pathways have been shown to improve not only psoriasis but also dyslipidemia and hyperuricemia. The finding of significantly elevated lipoprotein(a) levels in psoriatic patients (38.6 ± 18.4 mg/dL vs. 28.9 ± 14.2 mg/dL, $p < 0.001$) is of particular clinical significance. Lp(a) is an independent and causal risk factor for ASCVD, and its elevation in psoriasis adds to the already heightened cardiovascular risk burden. Our results are consistent with an updated meta-analysis demonstrating significantly higher Lp(a) levels in psoriasis patients compared to healthy controls (MD: 6.72 mg/dL, 95% CI: 4.32–9.12, $p < 0.00001$). The positive correlation between Lp(a) levels and PASI score observed in our study ($r = 0.482$, $p < 0.001$) suggests that Lp(a) elevation is related to disease activity and severity, supporting the hypothesis of a pathogenic link between psoriatic inflammation and Lp(a) metabolism. Abnormal Lp(a) levels (> 30 mg/dL) were observed in 52.7% of our psoriatic patients compared to 29.1% of controls, indicating that a substantial proportion of psoriatic patients carry this additional cardiovascular risk factor.

The significantly higher serum uric acid levels in psoriatic patients (6.25 ± 1.62 mg/dL vs. 5.71 ± 1.35 mg/dL, $p = 0.019$) and the greater prevalence of hyperuricemia (31.8% vs. 16.4%, $p = 0.009$) are consistent with previous reports. The prevalence of hyperuricemia in our study (31.8%) is comparable to the 33.7% reported in a study of 196 psoriatic patients. The mechanisms underlying hyperuricemia in psoriasis include increased purine breakdown due to enhanced epidermal turnover, as keratinocyte proliferation and turnover are markedly accelerated in psoriatic lesions. Additionally, the systemic inflammation characteristic of psoriasis may impair renal uric acid excretion. Multivariate analysis confirmed that psoriasis itself is a strong predictor of hyperuricemia (OR: 2.61). The correlation between biochemical parameters and disease severity observed in our study has important clinical implications. The positive correlations between PASI score and TC, TG, LDL-C, Lp(a), and SUA, along with the negative correlation with HDL-C, suggest that the metabolic derangements in psoriasis are driven, at least in part, by the systemic inflammatory burden of the disease. Patients with severe psoriasis had the most unfavorable metabolic profile, with the highest levels of atherogenic lipids and uric acid and the lowest levels of HDL-C. This observation underscores the importance of early and effective disease control not only for dermatological outcomes but also for mitigating long-term cardiovascular risk.

The clinical implications of our findings are substantial. The high prevalence of dyslipidemia, elevated Lp(a), and hyperuricemia in psoriatic patients warrants routine screening as part of comprehensive cardiovascular risk assessment. Current guidelines recommend that patients with moderate-to-severe psoriasis undergo regular screening for cardiovascular risk factors, including lipid profile and uric acid measurement. Given that psoriasis itself is an independent risk factor for cardiovascular disease, the presence of additional metabolic abnormalities further amplifies risk and may necessitate more aggressive preventive interventions.

The dose-response relationship between psoriasis severity and metabolic abnormalities suggests that effective control of psoriasis—whether through topical therapy, phototherapy, or systemic agents—may confer metabolic benefits beyond skin clearance. Biologic therapies targeting TNF-α, IL-17, and IL-23 have been shown to improve lipid profiles and reduce uric acid levels. These observations support the concept that reducing systemic inflammation through effective psoriasis treatment may attenuate the associated cardiometabolic risk. Several limitations of this study should be acknowledged. First, the cross-sectional design precludes the establishment of causal relationships, although the well-established biological plausibility supports the observed associations. Second, the study was conducted at a single tertiary care center, which may limit the generalizability of findings to community-based or primary care populations. Third, we did not assess other inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP) or interleukin-6, which could have provided a

more comprehensive assessment of the inflammatory state. Fourth, we did not evaluate the impact of psoriasis treatments on the measured parameters. Fifth, dietary factors and physical activity levels, which may influence lipid and uric acid levels, were not comprehensively assessed. Finally, the study did not include long-term follow-up to assess cardiovascular outcomes. Future research should focus on longitudinal studies to establish the temporal relationship between psoriasis, metabolic abnormalities, and cardiovascular events, and to evaluate whether interventions targeting both psoriasis and metabolic risk factors confer additive cardiovascular benefits. Studies incorporating advanced lipid profiling, including lipoprotein subfraction analysis and assessment of oxidized LDL, may provide further insights into the pathogenic mechanisms linking psoriasis to atherosclerosis.

CONCLUSION

This study demonstrates that patients with psoriasis exhibit a significantly atherogenic lipid profile characterized by elevated total cholesterol, triglycerides, LDL-C, VLDL-C, and lipoprotein(a), along with reduced HDL-C, and have significantly higher serum uric acid levels compared to age- and sex-matched healthy controls. These biochemical alterations correlate positively with disease severity, indicating that the systemic inflammatory burden of psoriasis contributes directly to metabolic and cardiovascular risk. The high prevalence of dyslipidemia, elevated Lp(a), and hyperuricemia in psoriatic patients underscores the importance of routine screening for these parameters as part of comprehensive cardiovascular risk assessment and management. Early identification and aggressive management of metabolic abnormalities, coupled with effective control of psoriasis, are essential strategies for reducing the substantial cardiovascular morbidity and mortality associated with this chronic inflammatory condition. We recommend that all patients with psoriasis, particularly those with moderate-to-severe disease, undergo regular screening with serum lipid profile, lipoprotein(a), and uric acid levels at the time of diagnosis and during follow-up.

REFERENCES

1. Li WQ, Han JL, Hu FB, Curhan GC, Qureshi AA. Psoriasis and risk of type 2 diabetes among women and men in the United States: a population-based cohort study. J Invest Dermatol. 2012;132(2):291-298.
2. Armstrong AW, Harskamp CT, Armstrong EJ. The association between psoriasis and obesity: a systematic review and meta-analysis of observational studies. Nutr Diabetes. 2012;2(12):e54.
3. Gelfand JM, Neimann AL, Shin DB, Wang X, Margolis DJ, Troxel AB. Risk of myocardial infarction in patients with psoriasis. JAMA. 2006;296(14):1735-1741.
4. Li X, Kong X, Wang Y, et al. Association of Serum Uric Acid Levels in Psoriasis: A Systematic Review and Meta-Analysis. Medicine (Baltimore). 2016;95(19):e3676.
5. Huang Y, et al. Hemoglobin A1c and uric acid on psoriatic arthritis risk in patients with psoriasis: a retrospective cohort study. SciDirect. 2025.
6. Li X, Miao R, Liu Y, et al. Serum uric acid levels and hyperuricemia in patients with psoriasis: a hospital-based cross-sectional study. An Bras Dermatol. 2018;93(5):761-763.
7. Li X, Kong X, Wang Y, et al. Association between psoriasis and hyperuricemia: a systematic review and meta-analysis. Medicine. 2016;95(19):e3676.
8. Mannangi NB, Basavaraj D, Balachandra A, Shubha J. Study of Cardiometabolic Markers Along with Lipid Indices in Psoriasis. 2022.
9. Pietrzak A, et al. Lipoprotein (a) in patients with psoriasis: associations with lipid profiles and disease severity. Int J Dermatol. 2009;48(4):379-387.
10. Evaluation of serum lipid, lipoprotein, and apolipoprotein levels in psoriatic patients: a systematic review and meta-analysis of case-control studies. BMC Med. 2019.
11. Lipoprotein (a) levels are elevated in psoriasis: An updated systematic review and meta-analysis. World J Clin Cases. 2025;13(33):112045.
12. Gupta A, Lohokare R, Sarkar PD, Dixit S. Case-Control Study of Lipoprotein (A) and Uric Acid in Patients of Psoriasis at Risk of Developing Cardiovascular Diseases. J Cardiovasc Dis Res. 2024;15(1):2473-2480.
13. Peng S, Li K. Causal association between cardiovascular risk factors and common skin diseases: A multivariate Mendelian randomization study. 2024.
14. Serum Intestinal Metabolites are Raised in Patients with Psoriasis and Metabolic Syndrome. 2022.
15. Endothelial dysfunction in psoriatic arthritis patients: relation to serum uric acid and disease activity. Egypt J Intern Med. 2025.
16. Psoriasis is the independent factor for early atherosclerosis: A prospective study of cardiometabolic risk profile. 2023.
17. Effects of Biologic Therapy on Laboratory Indicators of Cardiometabolic Diseases in Patients with Psoriasis. 2023.
18. Associations between psoriasis, psoriatic arthritis and gout or hyperuricemia: A systematic review and meta-analysis. Am J Med Sci. 2025.
19. Serum uric acid levels and arterial stiffness in patients with psoriasis. Arch Dermatol Res. 2025.
20. Cassano N, Carbonara M, Panaro M, Vestita M, Vena GA. Role of serum uric acid in conditioning the association of psoriasis with metabolic syndrome. Eur J Dermatol. 2011;21(5):1-5.