

ORIGINAL RESEARCH

SERUM URIC ACID AND LIPID PROFILES IN PATIENTS WITH PSORIASIS: A CROSS-SECTIONAL COMPARATIVE STUDY

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ABSTRACT

Background: Psoriasis is associated with metabolic comorbidity, but the relationship between circulating uric acid, lipid abnormalities and cutaneous disease severity remains uncertain. **Objective:** To compare uric acid and lipid concentrations between patients with psoriasis and healthy controls, and to describe their variation across Psoriasis Area and Severity Index (PASI) categories. **Methods:** This single-centre observational study included 25 patients with psoriasis without psoriatic arthritis and 25 healthy controls between August 2025 and January 2026. Fasting plasma was analysed for uric acid and lipid parameters. Patients were grouped by PASI-defined severity. Results were summarised as mean $\pm$ standard deviation, and Student's t-test was used for between-group comparisons. **Results:** Patients with psoriasis had higher reported mean total cholesterol, triglyceride, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol and uric acid concentrations, with lower high-density lipoprotein cholesterol. Mean uric acid was 8.29 ± 1.61 mg/dL in patients and 4.69 ± 1.11 mg/dL in controls. The mild, moderate and severe subgroups comprised 4, 8 and 13 patients, respectively; their mean uric acid concentrations were 4.42 ± 1.16 , 6.72 ± 1.31 and 8.18 ± 1.81 mg/dL. Total cholesterol and triglyceride concentrations also increased across the severity categories. **Conclusion:** The reported patterns suggest an association of psoriasis with adverse lipid concentrations and higher uric acid. The small sample, descriptive severity comparisons and unadjusted analysis preclude conclusions about an independent relationship with severity or the clinical value of uric acid as a biomarker.

Keywords: Psoriasis; Uric acid; Dyslipidaemia; Lipid profile; Psoriasis Area and Severity Index.

INTRODUCTION

Psoriasis is a chronic immune-mediated inflammatory disease whose burden extends beyond the skin. Observational studies have reported associations with metabolic syndrome and its components, including obesity, diabetes, hypertension and dyslipidaemia [1,2]. These comorbidities are relevant when evaluating the systemic health of patients with psoriasis, although their coexistence does not establish that cutaneous inflammation is their sole cause.

Lipid abnormalities have been documented at the onset of psoriasis and in established disease [3-5]. Individual lipid fractions do not behave consistently across studies: higher triglyceride and low-density lipoprotein cholesterol concentrations have been reported, whereas findings for high-density lipoprotein cholesterol vary [3,4]. Differences in study populations, disease characteristics, treatment exposure and metabolic risk factors may contribute to this variation.

Uric acid is the final product of purine metabolism in humans and participates in redox processes whose biological effects depend on context [6]. Its relationship with psoriasis has been investigated for several decades [7]. Subsequent clinical studies and meta-analyses have reported higher circulating uric acid or a greater frequency of hyperuricaemia in patients with psoriasis [8-10]. Nevertheless, evidence linking uric acid specifically to the severity of skin disease remains inconsistent [9].

Increased epidermal turnover provides a possible link between psoriasis and purine metabolism, while systemic inflammation and shared metabolic factors may influence both uric acid and lipid concentrations [6,7,9]. These mechanisms cannot be inferred from circulating measurements alone. We therefore compared fasting uric acid and lipid concentrations in patients with psoriasis and healthy controls, and examined the reported distributions across PASI-defined severity categories.

MATERIALS AND METHOD

Study design and participants

This single-centre observational study used a cross-sectional comparison of 25 patients with psoriasis and 25 healthy controls. Patients were recruited from the dermatology outpatient department between August 2025 and January 2026. Patients with psoriatic arthritis were excluded. Controls were selected from medical college and hospital employees. Information on psoriasis duration, medication use and dietary habits was recorded. Informed consent was obtained from participants.

Assessment of disease severity

Cutaneous disease severity was assessed using the Psoriasis Area and Severity Index (PASI) [11]. The study classified disease as mild at PASI<3, moderate at PASI 3.0– 6.0 and severe at PASI>6.1. These are the study's reported categories; comparisons with other studies require consideration of the severity thresholds used.

Biochemical measurements

After an overnight fast, 5– 7 mL of blood was collected into heparinised tubes. Plasma was separated by centrifugation at 3500 rpm for 10 minutes. Total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and uric acid were measured. Very-low-density lipoprotein cholesterol (VLDL-C) and low-density lipoprotein cholesterol (LDL-C) were calculated from the measured lipid parameters. All concentrations were expressed in mg/dL.

Statistical analysis

Continuous variables were summarised as mean $\pm$ standard deviation (SD). Student's t-test was used for between-group comparisons, with $P<0.05$ considered statistically significant. Biochemical values across the three PASI categories are presented descriptively.

RESULTS

Participants and disease severity

The study included 50 participants: 25 patients with psoriasis and 25 healthy controls. Among patients, 4 (16%) had mild disease, 8 (32%) had moderate disease and 13 (52%) had severe disease according to the study's PASI categories.

Comparison with controls

The reported group means indicated higher total cholesterol, triglycerides, VLDL-C, LDL-C and uric acid in patients with psoriasis, with lower HDL-C (Table 1). Mean total cholesterol was 214.85 ± 28.12 mg/dL in patients and 161.86 ± 31.82 mg/dL in controls; corresponding triglyceride concentrations were 247.78 ± 22.16 and 114.64 ± 32.21 mg/dL. Mean uric acid was 8.29 ± 1.61 mg/dL in patients and 4.69 ± 1.11 mg/dL in controls.

Table 1. Lipid and uric acid concentrations in controls and patients with psoriasis

Parameter	Controls (n = 25)	Psoriasis (n = 25)
Total cholesterol, mg/dL	161.86 ± 31.82	214.85 ± 28.12
Triglycerides, mg/dL	114.64 ± 32.21	247.78 ± 22.16

Parameter	Controls (n = 25)	Psoriasis (n = 25)
VLDL-C, mg/dL	28.35 ± 9.08	50.70 ± 11.12
HDL-C, mg/dL	58.27 ± 8.68	45.52 ± 9.38
LDL-C, mg/dL	104.86 ± 20.28	123.85 ± 17.28
Uric acid, mg/dL	4.69 ± 1.11	8.29 ± 1.61

Values are mean ± SD. HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; VLDL-C, very-low-density lipoprotein cholesterol. All concentrations are in mg/dL.

Biochemical profiles across severity categories

Mean total cholesterol increased across the mild, moderate and severe categories from 142.68 ± 18.18 to 172.12 ± 14.12 and 208.36 ± 20.12 mg/dL, respectively. Corresponding triglyceride concentrations were 110.16 ± 22.13, 152.26 ± 18.18 and 242.16 ± 24.14 mg/dL. Mean uric acid increased from 4.42 ± 1.16 mg/dL in mild disease to 6.72 ± 1.31 mg/dL in moderate disease and 8.18 ± 1.81 mg/dL in severe disease. VLDL-C and LDL-C followed an increasing pattern, whereas HDL-C decreased across the categories (Table 2). These comparisons describe subgroup means and do not quantify a patient-level correlation with PASI.

Table 2. Lipid and uric acid concentrations by psoriasis severity

Parameter	Mild (n = 4)	Moderate (n = 8)	Severe (n = 13)
Total cholesterol, mg/dL	142.68 ± 18.18	172.12 ± 14.12	208.36 ± 20.12
Triglycerides, mg/dL	110.16 ± 22.13	152.26 ± 18.18	242.16 ± 24.14
VLDL-C, mg/dL	20.18 ± 6.16	36.63 ± 7.12	49.12 ± 16.18
HDL-C, mg/dL	56.16 ± 16.16	49.13 ± 15.12	43.12 ± 16.16
LDL-C, mg/dL	100.66 ± 21.22	112.12 ± 18.12	122.80 ± 16.16
Uric acid, mg/dL	4.42 ± 1.16	6.72 ± 1.31	8.18 ± 1.81

Values are mean ± SD, in mg/dL. Study categories: mild, PASI<3; moderate, PASI 3.0– 6.0;severe, PASI>6.1. PASI, Psoriasis Area and Severity Index. Lipoprotein abbreviations are defined in Table 1.

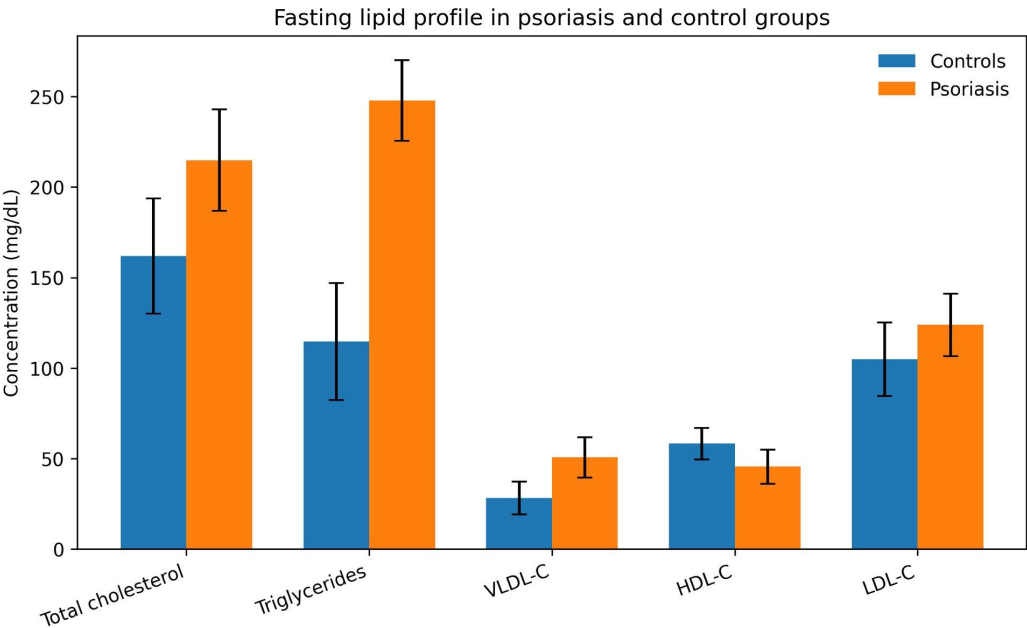


Figure 1. Error bars represent SD

DISCUSSION

The reported biochemical profiles suggest that patients with psoriasis had higher concentrations of several atherogenic lipid fractions and uric acid than healthy controls, together with lower HDL-C. Within the psoriasis group, uric acid, total cholesterol and triglyceride means increased across the study-defined severity categories. These patterns are descriptive. The small, unevenly distributed subgroups and absence of an adjusted analysis limit their interpretation as evidence of an independent relationship with disease severity.

The lipid findings are broadly consistent with earlier studies, although the direction of change in individual fractions has varied. Akhyani and colleagues reported higher triglycerides, total cholesterol and LDL-C in psoriasis, without a statistically significant difference in HDL-C [4]. Mallbris and colleagues identified lipid abnormalities at disease onset, including higher cholesterol concentrations in the VLDL and HDL fractions [3]. Thus, the lower HDL-C in the present study should not be assumed to characterise all patients with psoriasis. Differences in metabolic health, treatment exposure and participant selection may contribute to variation between cohorts.

Oxidative modification of lipoproteins offers a plausible biological connection between inflammation and altered lipid metabolism. Solak Tekin and colleagues investigated oxidised LDL in psoriatic skin together with circulating lipid changes [12]. Such evidence provides context for the present observations, but this study did not assess lipid oxidation, inflammatory pathways or vascular outcomes. Its lipid measurements therefore cannot establish a mechanism or quantify cardiovascular risk.

The uric acid findings also fit a broader observational literature. The study by Isha and colleagues reported higher uric acid in psoriasis, although its cohort included patients with arthritis [8]. Zhang and colleagues' meta-analysis found higher serum uric acid in psoriasis overall, with substantial between-study heterogeneity and inconsistent relationships with PASI [9]. A more recent meta-analysis by Liu and colleagues also found an association between psoriasis and hyperuricaemia [10]. These reports support further investigation of uric acid in psoriasis, while leaving uncertainty about its value as a marker of cutaneous severity.

Increased epidermal turnover could increase purine breakdown, and uric acid may reflect aspects of oxidative metabolism [6,7]. However, the present measurements cannot distinguish increased production from reduced excretion or other metabolic influences. Renal function, adiposity, diabetes, diet, alcohol intake and medication use may affect uric acid or lipid

concentrations. Excluding psoriatic arthritis narrows the clinical population but does not remove these potential sources of confounding. The increase in subgroup mean uric acid across PASI categories warrants cautious interpretation. Four patients formed the mild subgroup, making its summary estimates sensitive to individual observations. Moreover, an ordered series of group means does not establish a continuous correlation, temporal relationship or independent predictive value. The same caution applies to the apparent lipid gradients. Prospective studies with repeated measurements and adequate control of metabolic, renal, lifestyle and treatment-related factors would better address whether changes in uric acid accompany changes in skin disease.

The clinical context is the recognised need to assess comorbid disease in psoriasis. The joint American Academy of Dermatology–National Psoriasis Foundation guideline addresses cardiometabolic risk assessment as part of psoriasis care [13]. The present study adds exploratory observations in that setting, but it does not establish a role for routine uric acid measurement as a severity assessment or provide evidence that lowering uric acid improves psoriasis.

Limitations

The study was conducted at a single centre over six months and included only 25 patients with psoriasis. The small and uneven severity groups restrict precision and generalisability. Hospital employees may differ from dermatology outpatients in ways that introduce selection bias. The cross-sectional design cannot establish the direction of an association, and the analysis did not adjust for potential confounders. Although information on treatment and diet was collected, its influence

on the findings was not quantified. The study-specific PASI thresholds also limit direct comparison with cohorts using different severity classifications. Finally, comparisons of group means do not establish the prevalence of dyslipidaemia or hyperuricaemia in individual participants.

CONCLUSION

The reported results suggest that psoriasis is associated with adverse lipid concentrations and higher circulating uric acid, with descriptive increases in several parameters across PASI severity categories. These observations remain exploratory. Larger, well-characterised prospective cohorts are needed to determine whether uric acid is independently associated with psoriasis severity and whether its measurement adds clinically useful information.

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