

Serum Uric Acid Levels in Patients with Psoriasis: A Cross-Sectional Study

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ABSTRACT

Background: Psoriasis is a chronic inflammatory skin disease associated with various systemic comorbidities, including metabolic syndrome and hyperuricemia. Elevated serum uric acid (SUA) levels have been reported among individuals with psoriasis, although findings have been variable. Therefore, the present study aims to evaluate serum uric acid levels in patients with psoriasis and to analyze their correlation with demographic variables.

Methods: This cross-sectional study included 60 psoriasis patients (36 males, 24 females) aged 18–75 years attending a tertiary care center. Serum uric acid concentrations were measured and compared with established normal reference values. Patients were categorized based on uric acid levels (normal/high). Statistical analyses, including one-sample t-tests and descriptive statistics, were performed.

Results: The mean serum uric acid level was 9.39 ± 3.03 mg/dl in males and 7.70 ± 2.20 mg/dl in females, both significantly higher than the respective reference values ($p < 0.001$). High uric acid was found in 71.7% patients (75% of males and 66.7% of females). Uric acid levels followed a normal distribution. A positive correlation with psoriasis was seen, consistent with prior studies.

Conclusion: This study observed a significantly increased prevalence of hyperuricemia among patients with psoriasis, irrespective of sex. Routine SUA evaluation and management may be advisable in such patients to prevent further metabolic and cardiovascular complications.

Key-words: Psoriasis, Serum Uric Acid (SUA), Hyperuricemia, Metabolic syndrome, Uric acid

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disease that primarily affects the skin but has systemic implications far beyond cutaneous involvement. Clinically, it is characterized by erythematous, sharply demarcated plaques covered with silvery-white scales, with symptoms such as itching, pain, and bleeding that impair quality of life [1-4].

While often perceived as a skin disorder, psoriasis is increasingly recognized as a multisystem disease with implications for the nails, joints, and a range of associated comorbidities. Its chronic course, recurrent flares, and association with metabolic and cardiovascular disorders make psoriasis a significant global health concern [1].

The prevalence of psoriasis varies across geographic regions and ethnic groups. In India, estimates suggest a prevalence of 0.44% to 2.8% among adults, with a male predominance at nearly twice the rate compared to females [3]. Onset can occur at any age, though bimodal peaks are noted between 15–20 years and 55–60 years [5]. Psoriasis vulgaris, also known as plaque psoriasis, is the most frequently encountered type, representing

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approximately 90% of all cases ^[5]. Other clinical subtypes include flexural psoriasis, guttate psoriasis, generalized pustular psoriasis, and palmoplantar psoriasis, each with distinct morphological and prognostic characteristics ^[6,7]. Psoriasis has profound effects on physical and psychological well-being. In addition to visible lesions, patients often experience stigma, depression, anxiety, and reduced productivity, further underscoring the burden of this disease. Importantly, psoriasis is not merely confined to the skin. It serves as an indicator of systemic inflammation with implications for multiple organ systems.

The pathogenesis of psoriasis is multifactorial, involving genetic susceptibility, environmental triggers, and immune dysregulation ^[2]. The disease is characterized by hyperproliferation of keratinocytes, shortened epidermal cell turnover time, and altered differentiation. Histologically, psoriatic lesions exhibit acanthosis, parakeratosis, and inflammatory infiltrates in the dermis and epidermis. A hallmark feature is the accelerated turnover of epidermal keratinocytes from the usual 28 days to as little as 3–5 days in affected individuals ^[8].

This hyperproliferative state is accompanied by increased purine nucleotide metabolism, leading to greater production of metabolites such as uric acid ^[9]. Importantly, squamous cells in psoriasis proliferate in a highly organized manner without breaching the basement membrane, distinguishing the disease from neoplastic processes such as squamous cell carcinoma, where proliferation is disordered and invasive ^[10].

Serum uric acid (SUA), the final product of purine metabolism, is generated from both dietary sources and endogenous nucleic acid breakdown. The purines adenine and guanine are enzymatically degraded into xanthine and hypoxanthine, which are subsequently oxidized by xanthine oxidase to form uric acid ^[11-15]. Approximately two-thirds of uric acid is excreted via the kidneys, while the remainder is eliminated through the gastrointestinal tract.

In psoriasis, elevated SUA levels may occur due to increased nucleic acid turnover from accelerated keratinocyte proliferation. This biochemical link suggests that psoriasis may predispose patients to hyperuricemia, which in turn is associated with systemic complications. Interestingly, in psoriatic arthritis, SUA levels may not correlate with the severity of cutaneous involvement but may reflect metabolic disturbances ^[16].

Over the past two decades, psoriasis has been increasingly recognized as a systemic inflammatory disease. Numerous studies have documented its association with metabolic syndrome (MetS) and related conditions such as cardiovascular disease (CVD), type 2 diabetes mellitus, hypertension, dyslipidemia, and obesity ^[7,12-14]. Chronic low-grade inflammation, mediated by cytokines such as tumor necrosis factor- α (TNF- α), interleukin (IL)-17, and IL-23, underlies these associations. Elevated SUA levels may act as both a marker and mediator of such metabolic derangements. Hyperuricemia has been implicated in endothelial dysfunction, oxidative stress, and vascular inflammation, all of which contribute to the heightened cardiovascular risk observed in psoriasis patients.

The interplay between psoriasis, SUA, and metabolic disorders suggests a common pathophysiological pathway linking cutaneous inflammation with systemic disease. Therefore, investigating SUA levels in psoriasis patients may provide valuable insights into disease pathogenesis, severity assessment, and identification of individuals at risk for comorbid conditions.

Although psoriasis has long been studied as a dermatological condition, its systemic nature requires broader evaluation, particularly in the context of metabolic markers such as serum uric acid. Elevated SUA may represent a critical biomarker not only for disease activity but also for predicting long-term complications, especially cardiovascular and metabolic disorders. Understanding this relationship may facilitate better clinical management, early identification of at-risk individuals, and the development of targeted therapeutic strategies.

Given the limited number of studies in the Indian population examining the correlation between psoriasis and serum uric acid, there is a need for systematic evaluation in this area. This research seeks to contribute to the growing body of evidence on psoriasis as a systemic disease and highlight the significance of biochemical markers in its clinical assessment.

MATERIALS AND METHODS

Study Design and Setting- This cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Dermatology at Era's Lucknow Medical College & Hospital, Era University,

Lucknow, Uttar Pradesh. The study was conducted over a period of six months.

Study Participants- A total of 60 psoriasis patients aged 18–75 years, including both males and females, attending the Dermatology Outpatient Department (OPD) and Inpatient Department (IPD) were included in the study. Details of patient history, clinical presentation, diagnosis, and treatment were recorded using a case study form.

Inclusion Criteria

- ❖ Patients aged 18–75 years.
- ❖ Both male and female patients.
- ❖ Known patients with psoriasis.

Exclusion Criteria

- ❖ Patients with previously diagnosed gouty arthritis.
- ❖ Patients with renal disease.
- ❖ Cancer patients.
- ❖ Patients with known liver disease.
- ❖ Patients with excessive alcohol consumption.
- ❖ Pregnant women.

Sample Size Calculation- The sample size was calculated using G*Power software. A one-sample t-test for the difference from a constant was used for sample size estimation. The analysis was performed using an a priori calculation with a one-tailed test, an effect size (d) of 0.4753086, an alpha error probability of 0.05, and a power of 0.95. The minimum calculated sample size was 50, and the suggested sample size was 60. The actual power was 0.9524543. The standard value of serum uric acid used for comparison was based on the predefined reference value used in the sample size calculation.

Sample Collection- Venous blood (2–3 mL) was collected from the median cubital vein using a sterile disposable plastic syringe and transferred into a plain vial. The samples were centrifuged, and serum was separated for estimation of serum uric acid.

Serum Uric Acid Estimation- Serum uric acid was estimated by the Uricase/Peroxidase (POD) method using a semi-autoanalyzer and a kit method. The kit was manufactured by Beacon (Lot No. LS31-098; Mfg. 12/2023; Expiry 11/2025).

Principle- Uric acid is oxidized to allantoin by uricase with the production of hydrogen peroxide (H_2O_2). The hydrogen peroxide reacts with 4-aminoantipyrine (4-AAP) and dihydroxybenzene sulfonic acid (DHBS) in the presence of peroxidase to form a quinoneimine dye. The absorbance of the dye at 505 nm is proportional to the uric acid concentration in the sample.

Reagent Composition- The reagents contained HEPES buffer (>60 mmol/L), 4-AAP (>0.2 mmol/L), uricase (>300 U/L), peroxidase (>1000 U/L), and DHBS (>0.75 mmol/L).

Procedure- The sample was mixed and incubated for 5 minutes at 37°C. The absorbance of the test and standard was measured against the reagent blank.

Calculation

$$\text{Uric acid (mg/dL)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times \text{Concentration of Standard (mg/dL)}.$$

Reference Values- The expected serum uric acid values were 4.0–7.2 mg/dL for males and 2.7–6.5 mg/dL for females.

Statistical Analysis- Data were analyzed using descriptive statistics and a one-sample t-test. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. Serum uric acid levels were compared with the respective reference values using a one-sample t-test. The Shapiro–Wilk test was used to assess the distribution of serum uric acid levels. A p-value <0.05 was considered statistically significant.

Ethical Approval- Permission for the study was obtained from the Institutional Ethics Committee of Era's Lucknow Medical College & Hospital, Era University, Lucknow, Uttar Pradesh.

RESULTS

Table 1 shows the gender distribution of the study participants. Of the 60 participants, 36 (60%) were males and 24 (40%) were females, indicating a higher proportion of male participants in the study.

**Table 1:** Distribution of Subjects according to Gender

Sex	Frequency	Percentage (%)
Female	24	40
Male	36	60
Both	60	100

Table 2 presents the distribution of participants according to age group. The highest proportion of participants belonged to the 30–44 years age group (21,

35%), followed by 18–29 years (15, 25%). The 45–59 years and 60–75 years age groups each comprised 12 (20%) participants.

Table 2: Distribution of Subjects according to Age Group of Patients

Age group	Frequency	Percentage (%)
18-29	15	25
30-44	21	35
45-59	12	20
60-75	12	20
Total (18 -75)	60	100

Table 3 shows the gender distribution according to age group. Among females, 8 (33.3%) participants were aged 18–29 years, 8 (33.3%) were aged 30–44 years, 3 (12.5%) were aged 45–59 years, and 5 (20.8%) were aged 60–75

years. Among males, 7 (19.4%) were aged 18–29 years, 13 (36.1%) were aged 30–44 years, 9 (25.0%) were aged 45–59 years, and 7 (19.4%) were aged 60–75 years.

Table 3: Gender Distribution according to Age Group

Sex	Age Group	Frequency	Percent
Female	18-29	8	33.3
	30-44	8	33.3
	45-59	3	12.5
	60-75	5	20.8
	Total (18 - 75)	24	100
Male	18-29	7	19.4
	30-44	13	36.1
	45-59	9	25
	60-75	7	19.4
	Total (18 - 75)	36	100

Table 4 presents the descriptive statistics of serum uric acid levels according to gender. The mean serum uric acid level was 7.70 ± 2.20 mg/dL in females, with values

ranging from 3.69 to 13.09 mg/dL, whereas in males, the mean level was 9.39 ± 3.03 mg/dL, with values ranging from 4.50 to 18.97 mg/dL.

Table 4: Descriptive Statistics of Uric acid level

Uric Acid					
Gender	N	Minimum	Maximum	Mean	Std. Deviation
Female	24	3.69	13.09	7.6992	±2.20149
Male	36	4.50	18.97	9.3942	±3.03244

Table 5 shows the distribution of participants according to serum uric acid category. Overall, 43 (71.7%) participants were classified as having high serum uric acid levels, while 17 (28.3%) had normal levels.

Table 5: Distribution of Subjects according to Uric Acid Category

Uric Acid Category	Frequency	Percentage (%)
High	43	71.7
Normal	17	28.3

Table 6 presents the distribution of serum uric acid categories according to gender. Among females, 16 (66.7%) had high serum uric acid levels and 8 (33.3%) had normal levels. Among males, 27 (75.0%) had high serum uric acid levels and 9 (25.0%) had normal levels.

Table 6: Distribution of Gender according to Uric Acid Category

Sex	Uric Acid Category	Frequency	Percentage (%)
Female	High	16	66.7
	Normal	8	33.3
	Total	24	100
Male	High	27	75
	Normal	9	25
	Total	36	100

One-sample t-test showed that serum uric acid levels were significantly higher than the respective test values in both males and females. In males, the mean serum uric acid level was 9.39 ± 3.03 mg/dL, which was significantly higher than the test value of 5.6 mg/dL ($t = 7.507$, $df = 35$, $p < 0.001$). Similarly, in females, the mean serum uric acid level was 7.70 ± 2.20 mg/dL, which was significantly higher than the test value of 4.6 mg/dL ($t = 6.897$, $df = 23$, $p < 0.001$).

DISCUSSION

In the present study, serum uric acid (SUA) levels were significantly elevated in patients with psoriasis, with male patients exhibiting higher levels than females (9.39 ± 3.03 mg/dL vs. 7.69 ± 2.20 mg/dL, respectively).

This finding is consistent with the gender-related differences in SUA reported in previous studies and highlights the occurrence of hyperuricemia among patients with psoriasis. The higher SUA levels observed in males may be related to differences in urate metabolism and hormonal influences ^[1,2].

The pathophysiological mechanism underlying hyperuricemia in psoriasis is not completely understood. Enhanced epidermal turnover, a hallmark of psoriasis, contributes to accelerated nucleic acid metabolism and subsequent purine breakdown, resulting in increased uric acid production ^[3]. Previous studies have also suggested a relationship between inflammatory pathways and metabolic alterations in psoriasis. Inflammatory mediators such as IL-17 may contribute to

metabolic disturbances associated with psoriasis, including alterations in uric acid metabolism [4]. Hyperuricemia is clinically relevant because it may be associated with comorbid conditions such as gout, cardiovascular disease, hypertension, and renal impairment [5].

Our findings are consistent with several previous reports. Syami *et al.* [6] demonstrated significantly elevated SUA levels in Nepalese patients with psoriasis compared with controls, with gender-specific differences similar to those observed in the present study. Gisoni *et al.* [7] reported a higher prevalence of asymptomatic hyperuricemia among patients with psoriasis. Zhao *et al.* [8] in India and Hu *et al.* [9] in Turkey also reported elevated SUA levels and an association between SUA and psoriasis severity, including PASI scores. These findings from different populations support the association between psoriasis and increased SUA levels.

However, conflicting findings have also been reported. Minghui Hu *et al.* [10] found no significant association between SUA and psoriasis, including after subgroup analyses according to gender and ethnicity. Such differences may be related to variations in study design, sample size, population characteristics, and potential confounding factors such as diet, obesity, and metabolic syndrome. In contrast, a meta-analysis by Zhang *et al.* [11] reported significantly higher SUA levels and increased odds of hyperuricemia among patients with psoriasis, providing further evidence supporting an association between psoriasis and hyperuricemia.

The present study also showed that 71.7% of the participants had high uric acid levels, with a higher proportion observed among males (75%) than females (66.7%). These findings indicate that elevated SUA is common among the studied psoriatic population and may warrant attention during clinical assessment. The mean SUA level was 9.39 ± 3.03 mg/dL in males and 7.69 ± 2.20 mg/dL in females, and both values were significantly higher than the respective reference values ($p < 0.001$).

Taken together, the findings of the present study, along with previous reports, indicate an association between psoriasis and elevated SUA levels. Routine assessment of serum uric acid may therefore be useful in psoriatic patients, particularly in those with other metabolic or cardiovascular risk factors. Future studies should further clarify the causal relationship between psoriasis and

hyperuricemia, the role of inflammatory cytokines in uric acid metabolism, and whether interventions targeting uric acid can improve outcomes in psoriatic patients.

CONCLUSIONS

The present study demonstrates that serum uric acid (SUA) levels were significantly elevated in both male and female patients with psoriasis compared with the respective established reference values. The mean SUA level was higher in males (9.39 ± 3.03 mg/dL) than in females (7.70 ± 2.20 mg/dL), with the difference from the respective reference values being statistically significant ($p < 0.001$). Overall, 71.7% of the patients had high SUA levels, including 75% of males and 66.7% of females. These findings indicate that hyperuricemia is frequent among patients with psoriasis and may represent an important metabolic abnormality associated with the disease. Regular monitoring of SUA levels in psoriatic patients may therefore help in the early identification of hyperuricemia and related complications. Further studies with larger sample sizes are required to clarify the relationship between psoriasis and hyperuricemia and its potential clinical implications.

CONTRIBUTION OF AUTHORS

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