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Brief Report

Serum uric acid and lipid profile changes in lichen planus: A case–control study exploring metabolic imbalance

Sadhu Haarika[1], Prakash Bhanu[1], Samagani Akshay[2]

[1]Department of Dermatology, Vydehi Institute of Medical Sciences and Research Centre (VIMS & RC), Bangalore, India, [2]Department of Dermatology, Rajarajeswari Medical College and Hospital, Dr. M. G. R. Educational and Research Institute, India

Corresponding author: Sadhu Haarika, MD, E-mail: haarika.dermdoc@gmail.com

ABSTRACT

Background: Lichen planus is a chronic inflammatory skin condition that is increasingly linked to changes in metabolism. Uric acid, usually known for its role in gout, has become a marker associated with oxidative stress, inflammation, and issues with lipids. However, research on serum uric acid levels and their connection to lipid problems in lichen planus is limited. This study aimed to examine serum uric acid and lipid profile changes in patients with cutaneous lichen planus and to explore any correlations between them. **Materials and Methods:** This case-control study included 51 participants, consisting of clinically diagnosed cases of cutaneous lichen planus and matched healthy controls based on age and sex. We measured fasting serum uric acid levels and lipid profile parameters, including total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol, using standard lab techniques. We used appropriate statistical tests to compare cases and controls. Correlation analysis helped evaluate the relationship between serum uric acid and individual lipid parameters among those with lichen planus. **Results:** Patients with lichen planus showed significant differences in serum uric acid levels and lipid profile parameters when compared to controls, indicating a metabolic imbalance. Correlation analysis found notable connections between serum uric acid and certain lipid parameters. This suggests a possible link between purine metabolism and lipid problems in lichen planus. These findings support the idea that uric acid is a marker associated with systemic inflammation and metabolic risks in these patients. **Conclusions:** This study highlights the link between lichen planus, changes in serum uric acid levels, and lipid problems, as well as significant correlations between uric acid and lipid parameters. These findings emphasise the need to assess metabolism in patients with lichen planus and suggest that serum uric acid could be a useful marker for identifying patients at risk for metabolic issues. Further large-scale studies are needed to clarify the clinical implications of these links.

Key words: Lichen planus, Lipid profile, Serum uric acid

INTRODUCTION

In Lichen Planus (LP), studies have found dyslipidemia, suggesting a connection with systemic biochemical changes [1-3]. One biochemical marker, serum uric acid (SUA), has been studied in LP due to its known role in systemic inflammatory and oxidative disorders [4]. However, the role of SUA as a disease-related biomarker is still not clearly defined, as the levels show inconsistent patterns across LP studies [5,6]. Previous research has not looked into the relationship between SUA and lipid profiles, which are biologically linked in inflammatory disorders.

This study used modern visual analytic tools, like Orange data mining, to explore a less studied aspect of metabolic changes related to LP. This approach offered new insights into SUA-lipid interactions, moving beyond the assessment of individual biomarkers [7].

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MATERIALS AND METHODS

This was a case-control study done in a tertiary care centre. A total of 102 participants were included, comprising cases and controls equally. Cases with LP aged between 18 to 60 years were included. Those having lichenoid drug eruption, under systemic treatment for LP (in the last 3months) or with other comorbidities were excluded from the study.

After obtaining an informed written consent, detailed history was taken and a thorough dermatological examination was carried out. Laboratory estimation of SUA and lipid profile were done by drawing 5ml of fasting venous blood sample.

Statistical analysis was done using SPSS 22 version software. Exploratory data visualization was performed using Orange Data Mining software to assess patterns and relationships between SUA and lipid parameters. This analysis was used solely for visual corroboration of trends and did not replace inferential statistical testing.

Ethics Statement

The study was conducted after obtaining approval from the Institutional Ethics Committee. All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

RESULTS

The mean age of LP cases was 37.7 ± 12.8 years, compared to 39.6 ± 10.4 years for controls, with no statistically significant difference observed between the groups. A male preponderance was noted, with comparable sex distribution across the two groups (Table 1).

Among cases, the duration of disease was less than 1 year in 35.3% (n=18), while 47.1% (n=24) had disease duration between 1 and 3 years. A smaller proportion had longer disease durations of 4–6 years (9.8%, n=5), 7–9 years (5.9%, n=3), and ≥10 years (2%, n=1). Pruritus was the most prevalent presenting symptom, reported by 76.5% (n=39) of patients. followed by scaling in 74.5% (n=38). Burning sensation was noted in 25.5% (n=13), pain in 19.6% (n=10) and discharge in 7.8% (n=4). Koebnerization was observed in 51% (n=26) of cases. In terms of clinical morphology, classic lichen planus was the predominant pattern, including 27.5% (n=14), succeeded by hypertrophic lichen planus at 17.6% (n=9) and oral lichen planus at 13.7% (n=7). Other variants, including actinic, guttate, linear, lichen planopilaris and pigmentosus forms were less frequent. Mucosal involvement was present in 25.5% (n=13) of patients, with reticular (38.5%, n=5) and plaque-type lesions (30.8%, n=4) being the predominant oral subtypes. On severity assessment, the majority of patients had mild disease. Grade 1 severity was observed in 94.1% (n=48), while only 5.9% (n=3) had grade 2 disease (Table 2).

The mean SUA level in patients was 4.2 ± 1.2 mg/dL, whereas in controls it was 5.6 ± 0.75 mg/dL, indicating a statistically significant difference (Table 3). Lower SUA levels were more frequently observed in patients aged 31–40 years and with an LP duration exceeding one year; however, no consistent trend across age groups or disease duration was evident. Both male and female patients mainly showed normal serum uric acid (SUA) levels. There was no significant link between SUA levels and the clinical signs of lichen planus, comorbidities, smoking, alcohol use, or family history. Most patients in these groups had SUA values within the normal range.

The average total cholesterol level was significantly higher in lichen planus cases (177.5 ± 29.5 mg/dL)

Table 1: Baseline demographic characteristics of lichen planus cases and controls

Variable	Lichen planus cases (n = 51)	Controls (n = 51)	*p* value
Age (years)			
Mean±SD	37.7±12.8	39.6±10.4	0.399†
Median (Q1–Q3)	35 (27–50)	39 (31–49)	
Sex, n (%)			
Male	32 (62.7)	28 (54.9)	0.421‡
Female	19 (37.3)	23 (45.1)	

†Mann–Whitney U test; ‡Chi-square test; Statistically significant if $p < 0.05$

Table 2: Clinical characteristics of lichen planus cases (n = 51)

Clinical variable	Category	n (%)
Duration of disease	<1 year	18 (35.3)
	1–3 years	24 (47.1)
	≥4 years	9 (17.7)
Presenting symptom	Pruritus	39 (76.5)
	Scaling	38 (74.5)
Koebnerisation	Present	26 (51.0)
Clinical pattern	Classic LP	14 (27.5)
	Hypertrophic LP	9 (17.6)
	Oral LP	7 (13.7)
Mucosal involvement	Oral involvement	13 (25.5)
Severity grading*	Grade 1	48 (94.1)
	Grade 2	3 (5.9)

*Lichen Planus Severity Index (LPSI) based on the extent of disease

Table 3: Comparison of Serum Uric Acid and Lipid Profile Parameters between Lichen Planus Patients and Controls

Parameter (mg/dL)	Cases (n = 51) Mean ± SD	Median (IQR)	Controls (n = 51) Mean ± SD	Median (IQR)	p-value
Serum uric acid	4.2±1.2 Min–Max (mg/dL) 2.3–7.7	4.1 (3.5–4.7)	5.6±0.75 Min–Max (mg/dL) 4.1–8.0	5.7 (5.2–6.2)	<0.001*
Total cholesterol	177.5±29.5	177 (159–195)	168.3±40.1	161 (145–187)	0.036*
Triglycerides	144.5±44.1	139 (107–175)	115.3±40.7	115 (82–139)	0.0007*
LDL cholesterol	134.2±27.1	130 (118–148)	110.3±24.5	112 (97–126)	<0.001*
HDL cholesterol	34.4±6.4	34 (31–41)	46.9±7.6	46 (41–52)	<0.001*

Statistical test: Mann–Whitney U test
* Statistically significant ($p < 0.05$)

Table 4: Spearman correlation between serum uric acid and lipid profile in lichen planus cases and controls

Parameter correlated with SUA	LP cases (n = 51) ρ	p value	Controls (n = 51) ρ	p value
Total cholesterol	-0.027	0.852	0.041	0.774
Triglycerides	-0.109	0.446	0.067	0.641
LDL cholesterol	-0.143	0.317	0.147	0.305
HDL cholesterol	-0.155	0.277	-0.072	0.614

ρ = *Spearman's rank correlation coefficient; p values are two-tailed.*

compared to controls (168.3 ± 40.1 mg/dL), showing a statistically significant difference upon analysis ($p = 0.036$). Serum triglyceride levels were also significantly higher in the cases, with an average of 144.5 ± 44.1 mg/dL in patients, compared to 115.3 ± 40.7 mg/dL in controls ($p = 0.0007$). Similarly, low-density lipoprotein (LDL) cholesterol levels were noticeably higher in lichen planus patients (134.2 ± 27.1 mg/dL) than in controls (110.3 ± 24.5 mg/dL; $p = 0.00001$).

In the lichen planus group, high-density lipoprotein (HDL) cholesterol levels were significantly lower, with a mean HDL of 34.4 ± 6.4 mg/dL, compared to 46.9 ± 7.6 mg/dL in the control group ($p < 0.001$). These findings support the presence of a harmful lipid profile in the cases group. Spearman's rank correlation analysis was used to investigate the relationship between SUA and the lipid profile in both groups. In the cases, SUA showed a weak negative correlation with total cholesterol ($\rho = -0.027$), triglycerides ($\rho = -0.109$), LDL cholesterol ($\rho = -0.143$), and HDL cholesterol ($\rho = -0.155$). While these associations were minor and statistically insignificant, the correlations consistently pointed in the opposite direction across all lipid categories.

The control group showed weak and inconsistent correlations between SUA and lipid measurements, with no statistically significant links for total cholesterol ($\rho = 0.041$), triglycerides ($\rho = 0.067$), LDL cholesterol ($\rho = 0.147$), or HDL cholesterol ($\rho = -0.072$) (Table 4). The absence of similar associations in the control group suggests that the correlations found in lichen planus may be specific to the disease rather than coincidental.

Within the cases, lipid levels displayed statistically significant positive associations: total cholesterol correlated with triglycerides ($\rho = 0.510$, $p < 0.001$), total cholesterol with LDL cholesterol ($\rho = 0.616$, $p < 0.001$), and triglycerides with LDL cholesterol ($\rho = 0.307$, $p = 0.029$), indicating some consistency in dyslipidemia. This pattern was also observed in the controls, particularly for total cholesterol and LDL ($\rho = 0.738$, $p < 0.001$). Orange data mining software confirmed the SPSS results. Scatter plots of SUA against triglycerides and SUA against HDL showed significant variation with consistent trends but no strong linear correlation. The data points were spread across severity grades, supporting the weak inverse correlations found in the statistical analysis. The control group did not show these visual patterns.

DISCUSSION

In this study, we found that LP cases had much lower SUA levels compared to controls. This supports findings from other studies by Sree et al. (3.1 vs. 4.9 mg/dL) and Chakraborti et al. (3.6 vs. 3.9 mg/dL) [6,8]. The latter study indicated that SUA levels generally drop with longer disease duration, especially after six months [6]. A 2021 meta-analysis on oral lichen planus (OLP) confirmed that total antioxidant capacity and SUA levels are significantly lower in OLP patients. These patients also show higher signs of oxidative stress, such as malondialdehyde (MDA) and nitric oxide (NO) [9]. In contrast, Eşberk et al. found no significant difference in SUA levels between LP cases and controls, which suggests there may be differences in population or methods used [10]. Overall, most evidence, including ours, supports the idea that the

ongoing inflammation and oxidative stress in LP lower SUA levels [9,10].

Uric acid serves as an important plasma antioxidant. However, research shows that intracellular or crystalline urate can activate NLRP3 inflammasome pathways and increase pro-inflammatory signaling (IL-1β and TNF-α) under certain conditions. This requires us to understand SUA in the context of tissue redox and immune activation dynamics [4,11-13].

This dual role helps explain why SUA decreases in peripheral blood; it is consumed as an antioxidant while also promoting inflammation at the tissue level. This distinction is important for LP, where the skin's immune environment may differ greatly from what is happening in the bloodstream [12,14]. This paradox highlights the need to view SUA as more than just a static antioxidant marker; it acts as a dynamic mediator in inflammatory conditions. LP cases exhibited a pro-atherogenic lipid profile (58% vs. 30% in controls, p=0.016). They had significantly higher levels of total cholesterol (TC), triglycerides (TG), and LDL, along with lower HDL compared to controls. This aligns with many recent studies, including those by Shaila KN et al., Ozbagcivan et al., and Xiao et al. [2,15,16]. Similarly, Jeyraveena et al. did a case-control study which reported that LP patients exhibited elevated TC and LDL levels, with TG levels being greater in men compared to controls [1]. Collectively, these studies indicate that LP frequently correlates with dyslipidemia. The mechanistic role of T-cell mediated inflammation in LP is believed to interfere with lipid metabolism; pro-inflammatory cytokines (IL-6, TNF-α, IFN-γ, etc.) and reactive oxygen species produced in LP lesions might provoke hepatic dyslipidemia [2,15].

Nonetheless, consistency among reports is lacking. García-Roza et al. discovered that OLP patients were not inherently dyslipidemic, however were more inclined to be prescribed statins or other lipid-lowering medications [3]. In that study, OLP was correlated with the utilization of lipid-modifying medicine, but not with increased lipid levels, potentially indicating preventive treatment in those patients. These disparities may indicate regional, dietary or study-design issues [3]. However, meta-analyses demonstrate that LP markedly elevates the risk of dyslipidemia [16]. Thus, LP patients require regular metabolic screening to reduce the risk of cardiovascular diseases [2,15].

Severe disease implies a higher degree of systemic inflammation, leading to more metabolic disruption. Evidence indicates that extensive or erosive LP demonstrates elevated dyslipidemia and oxidative stress markers compared to the milder variants [3,17].

Though this study did not focus on biochemical comparisons based on severity, the clustering of metabolic issues across severity levels shows the potential of SUA and lipid indices as additional indicators of disease burden in future groups. Therefore, LP is connected to a condition that promotes oxidation and atherosclerosis, which requires monitoring of metabolic risk factors [9]. Screening for lipid disorders and evaluating interventions that support antioxidants, as shown in some trials, could be helpful [9,10]. In conclusion, our research improves understanding of LP by linking its skin-related immune issues to changes in systemic metabolism.

Ali et al. (2025) found that in a healthy population, SUA positively correlated with TG, TC, and LDL, while it negatively correlated with HDL [7]. In contrast, our LP cases showed a consistent opposite trend: lower SUA was linked to higher TC, TG, and LDL levels, along with lower HDL, although these correlations were weak and statistically insignificant. This inverse pattern did not appear in the control group. In the Orange visualization of scatterplot matrices, LP cases showed clustering with low UA and high TG/LDL levels, while controls did not display a clear pattern. Thus, LP patients with more lipid disorders had lower antioxidant levels (UA). No previous research has looked into SUA-lipid correlations in LP patients; therefore, our findings offer new insights. We demonstrated this using Orange, a contemporary open-source data-mining suite that facilitated interactive exploration of scatterplots and correlations [18]. It supported the findings from SPSS, revealing directional patterns, such as an inverse relationship between UA and TG in LP, despite modest statistical significance (Figs. 1a and 1b).

In lichen planus, initial immunological activation may induce oxidative stress, leading to uric acid accumulation and subsequent cytokine-mediated hepatic lipid dysregulation, suggesting a temporal sequence rather than direct causation. Conversely, pre-existing metabolic abnormalities may diminish the threshold for immune dysregulation, hence promoting the onset of disease [18,19]. Our cross-sectional findings cannot establish temporal sequence.

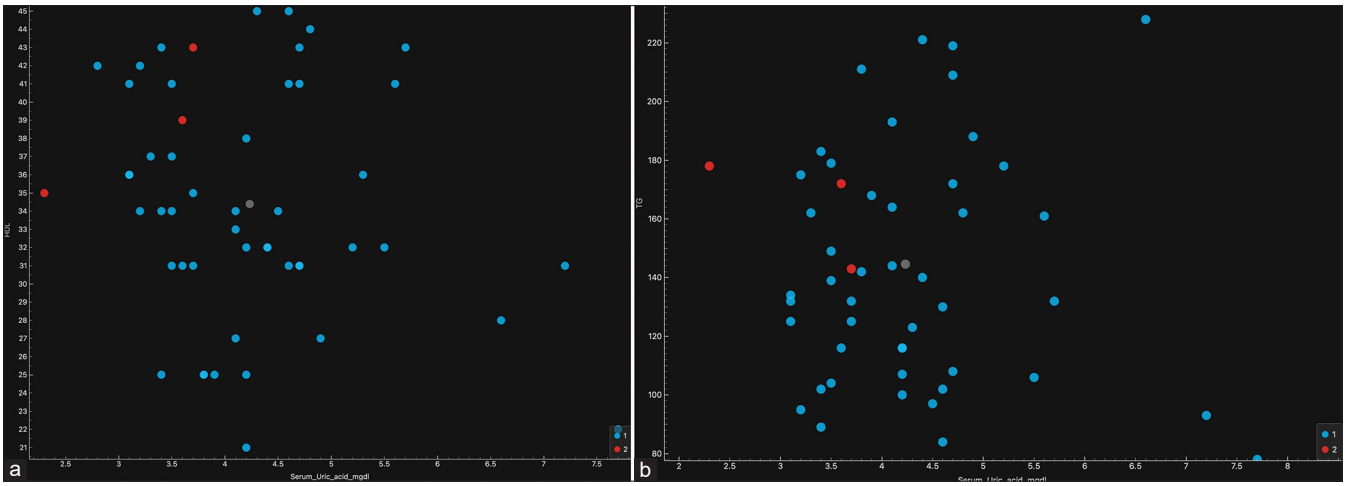

Figure 1: Scatter plot visualization of correlations between serum uric acid and lipid parameters in lichen planus cases (Orange software). Points are colored according to disease severity (severity grade 1 and grade 2). (a) The plot demonstrates a wide dispersion with no strong linear association. (b) A weak inverse association with considerable dispersion is observed.

But subsequent research should use serial biomarker sampling, disease activity scoring and targeted metabolomics to elucidate causality and determine if SUA–lipid patterns forecast progression or systemic comorbidity [20].

Inferences from other inflammatory dermatoses suggest a credible role for metabolic-microbial interactions in disease progression [21], though direct data connecting abnormalities in the gut microbiome to LP is few. In view of the gut-skin axis, dyslipidemia may induce dysbiosis and systemic endotoxemia, whereas uric acid metabolism is acknowledged as a regulator of gut inflammation and microbial diversity [20,21]. Dysregulation of these two in LP may foster a conducive gut-inflammatory milieu, exacerbating systemic immune activation and cutaneous immunological responses.

We hereby propose a metabolic–oxidative–immune model of lichen planus, wherein chronic T-cell-mediated inflammation induces persistent oxidative stress, depleting serum uric acid, while concurrently facilitating hepatic and systemic lipid abnormalities.

This metabolic environment may trigger immune activation through endothelial dysfunction, inflammasome signaling, and changes in lipid mediator production, creating a continuous cycle of inflammation. [22] The strong inverse relationship between the two in lichen planus supports this idea, confirming the SUA-lipid interaction as a specific biochemical marker of the disease instead of just an accidental observation. This framework lays the groundwork for future studies that aim to redefine lichen planus as a systemic inflammatory condition with measurable metabolic links. Looking back, additional therapy strategies could be effective. Lipid-lowering treatments, especially statins, have shown anti-inflammatory and immune-modulating effects that might help with lichen planus. Antioxidant-based treatments have proven useful in managing lichen planus. However, changing uric acid levels through diet or supplements is still uncertain because of its dual role in immunity and needs further exploration through clinical trials. [19]

CONCLUSION

Lichen planus is associated with reduced serum uric acid levels and a pro-atherogenic lipid profile. While SUA–lipid correlations were weak, their consistent inverse trend suggests a potential link between oxidative stress and metabolic dysregulation in LP, meriting further investigation.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the committee responsible on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

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