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Clinical Significance of Hematological Parameters and Inflammatory Indices in Patients with Lichen Planus

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Research Article

History

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ABSTRACT

Aim: Lichen planus (LP) is a chronic, T-cell–mediated inflammatory dermatosis that affects the skin and mucous membranes. However, the relationship between LP and systemic inflammatory biomarkers remains controversial. This study aimed to evaluate the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), Systemic Immune-Inflammation Index (SII), and Systemic Inflammatory Response Index (SIRI) in patients with LP, compare these parameters with healthy controls, and investigate their diagnostic discriminative value.
Methods: In this retrospective study, 98 patients diagnosed with LP who presented to Sivas Numune Hospital between 1 August 2022 and 1 October 2025 were screened; after applying exclusion criteria, 52 LP patients and age- and sex-matched 52 healthy controls were included in the analysis. Demographic data and hemogram parameters were recorded for all participants, and NLR, PLR, SII, and SIRI were calculated using standard formulas. Diagnostic performance was assessed using a receiver operating characteristic (ROC) curve analysis.
Results: The LP and control groups were similar in terms of age and sex distributions. The hemoglobin, NLR, PLR, SII, and SIRI levels did not differ significantly between the groups ($p>0.05$). None of the indices demonstrated significant diagnostic discrimination in the ROC analysis (AUC <0.60 for all parameters). A subgroup analysis of 15 patients with oral mucosal involvement also revealed no significant differences in systemic inflammatory indices.
Conclusion: The findings indicate that LP does not substantially elevate the systemic inflammatory response, and hematological indices such as NLR, PLR, SII, and SIRI have limited value in diagnosing LP or distinguishing clinical variants such as oral involvement. These results support the view that LP is predominantly a localized immune-mediated inflammatory condition. Prospective studies with larger cohorts integrating molecular- and cytokine-based biomarkers are needed to better elucidate the systemic inflammatory profile of LP.

Keywords: Lichen planus, Oral mucosa, Systemic inflammation, Biomarker

Liken Planus Hastalarında Hematolojik Parametreler ve İnflamatuvar İndekslerin Klinik Önemi

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ÖZ

Amaç: Liken planus (LP), deri ve mukozaları etkileyen kronik, T hücre aracılı bir inflamatuvar dermatozdur. Hastalığın sistemik inflamatuvar belirteçlerle ilişkisi literatürde tartışmalıdır. Bu çalışma, LP hastalarında nötrofil/lenfosit oranı (NLR), trombosit/lenfosit oranı (PLR), Sistemik İmmün-İnflamasyon İndeksi (SII) ve Sistemik İnflamatuar Yanıt İndeksi (SIRI) düzeylerini değerlendirmeyi, sağlıklı kontrollerle karşılaştırmayı ve bu parametrelerin tanısal ayırt ediciliğini incelemeyi amaçlamıştır.
Yöntem: Retrospektif olarak planlanan çalışmada 1 Ağustos 2022–1 Ekim 2025 tarihleri arasında Sivas Numune Hastanesi'ne başvuran 98 LP hastası taranmış; dışlama kriterleri sonrası 52 LP hastası ve yaş-cinsiyet eşleşmeli 52 sağlıklı kontrol analize dâhil edilmiştir. Tüm katılımcıların demografik özellikleri ve hemogram parametreleri kaydedilmiş, NLR, PLR, SII ve SIRI standart formüllerle hesaplanmıştır. Tanısal performans ROC eğrileriyle değerlendirilmiştir.
Bulgular: LP ve kontrol grupları yaş ve cinsiyet açısından benzerdi. Hemoglobin, NLR, PLR, SII ve SIRI değerleri iki grup arasında anlamlı fark göstermedi ($p>0,05$). ROC analizinde hiçbir indeks anlamlı tanısal ayırt edicilik sunmadı (AUC tüm parametrelerde <0,6). Oral mukoza tutulumu olan 15 hasta alt grup analizinde de hematolojik inflamasyon indekslerinin hiçbirinde anlamlı değişiklik izlenmedi.
Sonuç: Bulgular, LP'nin sistemik inflamatuvar yanıtı belirgin düzeyde artırmadığını ve NLR, PLR, SII ve SIRI gibi hematolojik indekslerin LP'nin tanısında veya oral tutulum gibi klinik varyantların ayrımında sınırlı değer taşıdığını göstermektedir. LP'nin temel olarak deri ve mukozaya lokalize immun aracılı inflamatuvar bir hastalık olduğu düşünülmektedir. Daha geniş örneklemlerle yapılacak prospektif çalışmaların moleküler ve sitokin temelli biyobelirteçlerle birlikte değerlendirilmesi, LP'nin sistemik inflamasyon profiline yönelik daha kapsamlı sonuçlar sağlayacaktır.

Anahtar Kelimeler: Liken planus, Oral mukoza, Sistemik inflamasyon, Biyobelirteç

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Introduction

Lichen planus (LP) is a chronic, immune-mediated inflammatory dermatosis that was first systematically described by Erasmus Wilson, and can involve the skin, mucous membranes, scalp, and nails.[1] Although its epidemiology and pathogenesis have not been fully elucidated, current evidence indicates that both genetic susceptibility and environmental triggers play an important role in disease development. The prevalence of LP varies considerably across geographical regions with an estimated incidence of approximately 1% in the general population. Oral LP (OLP) has a global prevalence of approximately 1%, and occurs more frequently in women.[2] Follicular variants, such as lichen planopilaris and frontal fibrosing alopecia, predominantly affect postmenopausal women, whereas pediatric LP accounts for only 1–4% of all cases.[3]

The pathogenesis of LP is multifactorial, with T-cell-mediated cytotoxicity constituting the core mechanism.[4] Additional mechanisms, including mast cell activation, microRNA dysregulation, Notch and Toll-like receptor signaling, inflammasome activation, and enhanced angiogenesis have also been shown to sustain the inflammatory process. Drug reactions, infections, vaccinations, contact allergens, trauma (Koebner phenomenon), and radiotherapy are among the major triggering factors.[5]

Cutaneous LP typically presents with violaceous, flat-topped pruritic papules that are often accompanied by Wickham striae. Mucosal involvement may cause burning pain, whereas follicular and nail involvement can lead to scarring, alopecia, or pterygium formation. Current guidelines recommend topical corticosteroids and topical calcineurin inhibitors as first-line therapies, with the strongest evidence. For widespread disease, high-potency topical steroids and NB-UVB phototherapy are effective options, whereas systemic retinoids, methotrexate, cyclosporine, and mycophenolate mofetil are reserved for third-line treatment.[6]

In recent years, LP has been increasingly recognized not only as a localized cutaneous disorder but also as a condition potentially associated with systemic inflammation. Several studies have demonstrated elevated oxidative stress, cytokine imbalance, and immune activation in the peripheral circulation of patients with.[7-9] This has led to growing interest in identifying biomarkers that may reflect the extent of disease, activity, and treatment response.

Hematological inflammatory indices, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), Systemic Immune-Inflammation Index (SII), Systemic Inflammatory Response Index (SIRI), and Pan-Immune-Inflammation Value (PIV), are practical, low-cost, and easily accessible markers for assessing systemic inflammation. Elevated NLR and SII levels may indicate proinflammatory activity, whereas lymphopenia may reflect impaired immune regulation. These indices have been associated with disease activity and prognosis of dermatological disorders such as psoriasis, hidradenitis suppurativa, and vitiligo.[10-13] However, studies evaluating the diagnostic or prognostic value of LP are limited.[14-16]

This study aimed to assess the levels of NLR, SII, SIRI, and PIV in patients with cutaneous LP, compare these indices with those in healthy individuals, and determine their diagnostic performance using ROC curve analysis. These findings are expected to contribute to a better understanding of the systemic inflammatory components of LP and support the development of biomarker-based clinical assessments and treatment strategies.

Methods

This study was designed as a retrospective analysis and included patients who presented to the Dermatology and Venereology Outpatient Clinic of Sivas Numune Hospital between August 1, 2022, and October 1, 2025. During the study period, 98 patients were evaluated consecutively and diagnosed with lichen planus (LP) by a dermatologist, along with individuals assessed for the healthy control group. After applying the exclusion criteria, the final study population comprised 52 patients with LP.

The exclusion criteria were as follows:

- Presence of acute infection (n = 13),
- History of malignancy or ongoing diagnostic evaluation for malignancy (n = 4),
- Chronic inflammatory disease (n = 9),
- Incomplete hemogram, and missing demographic data (n = 20).

No additional laboratory tests or investigations were ordered for the purposes of the study, and all data were obtained from the hospital information management system. A 1:1 age- and sex-matched healthy control group, consisting of 52 individuals, was created to correspond to the 52 included patients with LP.

Demographic characteristics (age and sex) and routine hemogram parameters, including hemoglobin (Hb, g/dL), neutrophils (N, $10^3/mm^3$), lymphocytes (L, $10^3/mm^3$), monocytes (M, $10^3/mm^3$), and platelets (P, $10^3/mm^3$), were recorded for all participants. Four systemic inflammatory indices were calculated to reflect the systemic inflammatory status. The neutrophil-to-lymphocyte ratio (NLR) was calculated as N/L, platelet-to-lymphocyte ratio (PLR) as P/L; the Systemic Inflammatory Response Index (SIRI) as (N/L) × M; and the Systemic Immune-Inflammation Index (SII) as (N/L) × P.[17]

Ethical Approval

Ethical approval for this study was obtained from the Ethics Committee of Health Sciences Research at Sivas Cumhuriyet University (Decision No: 2025-10/31, October 9, 2025).

Statistical Analysis

The sample size was calculated using the G*Power 3.1.9.4 program. Assuming an effect size of 0.40, a type I error rate of 0.05, and a test power of 90%, the analysis indicated that a minimum of 100 patients+control groups would be required for the study. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) for Windows version 22 (IBM SPSS Inc., Chicago, IL, USA). The normality of continuous variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Variables with normal distribution are expressed as

mean ± standard deviation, whereas non-normally distributed variables are presented as medians (min–max).

The predictive accuracy of NLR, PLR, SII, and SIRI for lichen planus was evaluated using a receiver operating characteristic (ROC) curve analysis. Optimal cut-off values for the SII, SIRI, NLR, and PLR were determined using the Youden Index within the ROC analysis. Associations between inflammatory indices and clinicopathological or biochemical variables were examined using the chi-square test, Fisher's exact test, Mann–Whitney U test, and Kruskal–Wallis test, as appropriate. Spearman's correlation coefficient was used to compare the frequency distributions between the tested groups. Statistical significance was set at $P < 0.05$.

Results

In total, 104 patients were included in this study. Of these, 43 (41.3%) were under 45 years of age, and 61 (58.7%) were 45 years or older. The sex distribution was comparable, with 53 males (51.0%) and 51 females (49.0%). Oral mucosal involvement was present in 15 patients (28.8%), whereas 37 (71.2%) had no oral lesions. Topical corticosteroids were the most frequently used treatment modality (n = 27, 52.0%). A combination of topical corticosteroids and calcineurin inhibitors was administered to 18 patients (34.6%), systemic corticosteroid therapy to 4 patients (7.6%), and methotrexate to 3 patients (5.8%). Demographic and clinical data are summarized in Table 1.

ROC curve analysis was performed to assess the diagnostic discriminatory ability of hemoglobin, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII). The area under the curve (AUC) for hemoglobin was 0.577 (p = 0.176; 95% CI: 0.466–0.687). AUC values for NLR, PLR, and SII were 0.558 (p = 0.312; 95% CI: 0.447–0.668), 0.560 (p = 0.294; 95% CI: 0.448–0.671), and 0.576 (p = 0.180; 95% CI: 0.466–0.687), respectively. None of the evaluated hematological or inflammatory parameters demonstrated a statistically significant diagnostic performance (Figure 1, Table 2).

The age distribution was similar between the groups: individuals <45 years comprised 48.1% of the control group and 34.6% of the LP group (p = 0.116). Gender distribution was likewise comparable, with males representing 51.9% of the controls and 50.0% of the LP group (p = 0.500). A comparison of hematological and inflammatory parameters revealed no significant differences between the groups. Hemoglobin levels were 14.45 ± 1.6 g/dL in the control group and 14.02 ± 1.78 g/dL in the LP group (p = 0.176). NLR values were 2.07 ± 0.69 and 1.93 ± 0.71 (p = 0.311), PLR values were 128.18 ± 42.7 and 123.9 ± 54.14 (p = 0.294), and SII values were 585.49 ± 224.19 and 548.04 ± 302.79 (p = 0.180), respectively. SIRI values were 1.08 ± 0.50 in controls and 1.14 ± 0.71 in the LP group (p = 0.732). Collectively, these findings indicate no significant difference between patients with LP and healthy controls in terms of systemic hematologic inflammatory markers (Figure 2, Table 3).

Table 1. *The demographic and clinical data*

Variable	Category	N	%
Age (n=104)	<45 years	43	41.3%
	≥45 years	61	58.7%
Sex (n=104)	Male	53	51.0%
	Female	51	49.0%
Oral Mucosal Involvement (n=52)	Absent	37	71.8%
	Present	15	28.8%
Treatment (n=52)	Topical corticosteroid	27	52.0%
	Topical corticosteroid + calcineurin inhibitor	18	34.6%
	Systemic corticosteroid	4	7.6%
	Methotrexate	3	5.8%

Table 2. *ROC Curve Analysis*

Test Result Variable(s)	Area	Std. Error	p-value	95% CI Lower Bound	95% CI Upper Bound
Hemoglobin	0.577	0.056	0.176	0.466	0.687
NLR	0.558	0.056	0.312	0.447	0.668
PLR	0.560	0.057	0.294	0.448	0.671
SII	0.576	0.056	0.180	0.466	0.687
SIRI	0.519	0.058	0.733	0.405	0.634

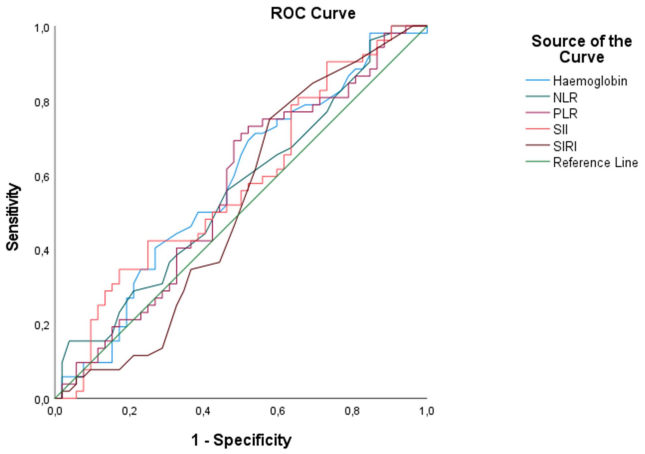


Figure 1: *ROC curve analysis.*

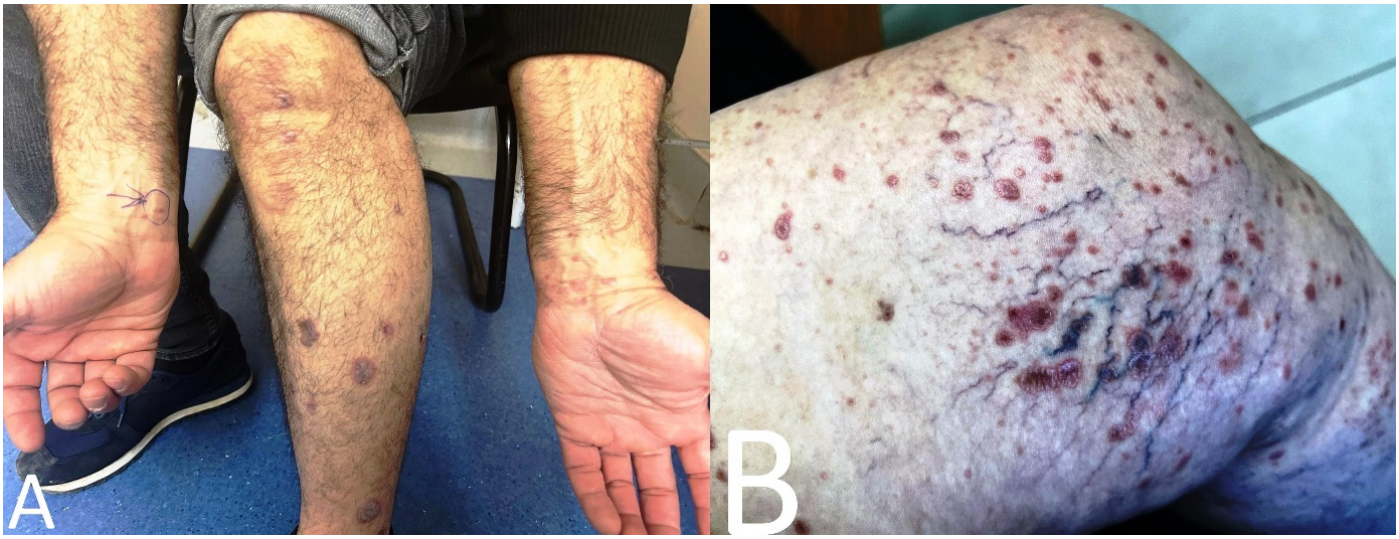


Figure 2. *(A) Lichenified violaceous flat papules and plaques are present on the wrists, knees, and lateral aspects of the legs. Several lesions exhibit post-inflammatory hyperpigmented macules consistent with the healing phase. (B) Numerous lichenified flat papules and plaques are observed on the knee, some demonstrating an annular configuration. Prominent superficial venous dilatations are also noted on the lower extremity.*

Table 3. *Comparison of clinical parameters and systemic inflammatory indices between the control group and patients with lichen planus*

		Control	LP	p
Age, n (%)	<45	25 (%48.1)	18 (34,6%)	0.116
	≥45	27(%51.9)	34 (65,4%)	
Sex, n (%)	Male	27(%51.9)	26(%%50.0)	0.500
	Female	25(%48.1)	26(%%50.0)	
Hemoglobin (g/dL), mean (SD)		14.45 ± 1.6	14.02 ± 1.78	0.176
NLR, mean (SD)		2.07 ± 0.69	1.93 ± 0.71	0.311
PLR, mean (SD)		128.18 ± 42.7	123.9 ± 54.14	0.294
SII(10^3/mm^3), mean (SD)		585.49 ± 224.19	548.04 ± 302.79	0.180
SIRI(10^3/mm^3), mean (SD)		1.08 ± 0.5	1.14 ± 0.71	0.732

Table 4. *The association between oral mucosal involvement and systemic inflammatory indices*

	Oral Mucosal Involvement		
	Absent	**Present**	**p-value**
Hemoglobin (g/dL), mean (SD)	13.95 ± 1.69	14.21 ± 2.00	0.312
NLR mean (SD)	1.98 ± 0.76	1.83 ± 0.57	0.620
PLR mean (SD)	128.92 ± 60.28	111.51 ± 33.2	0.551
SII(10^3/mm^3), mean (SD)	588.79 ± 342.85	447.51 ± 128.50	0.173
SIRI(10^3/mm^3), mean (SD)	1.12 ± 0.70	1.18 ± 0.78	0.839

Discussion

Lichen planus (LP) is a chronic inflammatory dermatosis characterized by T-cell–mediated immune response. Histopathologically, LP demonstrates a dense, band-like lichenoid infiltrate, basal keratinocyte damage, and cytokine-driven inflammatory environment. However, whether this localized immune activation is reflected in systemic hematological parameters remains a matter of debate. While some studies have suggested that cytokine-mediated inflammation in the LP extends to the systemic level,[18] others have reported that localized disease does not significantly alter peripheral cellular inflammatory markers.[19]

In the present study, the absence of statistically significant differences in hemoglobin, NLR, PLR, SII, and SIRI levels between patients with LP and healthy controls supports the latter view. Similarly, the lack of association between oral mucosal involvement, an LP subtype, and hematologic inflammatory indices indicates that oral LP does not reflect systemic immune activation. Although oral LP is known for its more persistent course and potential for poorer prognosis, most studies emphasize the predominant contribution of local factors, such as microbial flora, trauma, and site-specific cytokine production.[20,21]

Studies investigating systemic inflammatory biomarkers in LP remain limited. NLR and PLR have been shown to increase in several dermatological conditions, including psoriasis, atopic dermatitis, and chronic urticaria, as indicators of systemic inflammation.[16,22] Some small-scale studies focusing on LP have reported mildly elevated NLR levels; however, the findings have been inconsistent. Jornet et al. reported higher NLR values in patients with LP than in controls,[14] whereas another study reported no significant difference.[23] The comparable NLR levels observed in our study suggest that neutrophil–lymphocyte balance may not play a major role in LP pathophysiology.

The PLR and SII are widely used markers for assessing systemic inflammatory activity, particularly in autoimmune and inflammatory diseases. However, the role of platelet-mediated inflammation in LP has been minimally explored. Although platelets are recognized modulators of immune responses, the predominance of T-cell-driven dermal infiltration in LP may explain the lack of significant alterations in PLR and SII.[23] Therefore, our findings support the notion that these indices are unsuitable inflammatory biomarkers for LP.

More comprehensive indices such as the SIRI and SII have demonstrated prognostic value in cancer, sepsis, and severe inflammatory disorders.[24-27] Nevertheless, their sensitivity may be limited in localized skin diseases, such as LP. The absence of a significant elevation in SIRI and SII levels in our LP cohort strengthens the view that LP is associated with low-to-moderate systemic inflammatory activity rather than robust systemic inflammation.

A subgroup analysis focusing on oral mucosal involvement offers additional insights. Oral LP is categorized as an oral potentially malignant disorder (OPMD) and is sometimes hypothesized to be linked to systemic inflammation due to its reported malignant transformation risk.[28] However, the present findings indicate that oral involvement does not significantly influence hematological inflammatory indices. This suggests that the risk of malignant transformation in oral LP may be more closely related to local environmental factors and molecular alterations than to systemic inflammatory markers.

This study had several limitations. Due to its retrospective design, causal relationships cannot be established. Additionally, inflammatory markers were assessed using a single laboratory measurement, and prospective studies incorporating serial measurements would provide more robust conclusions.

Despite these limitations, this study contributes meaningfully to the literature by systematically evaluating commonly used hematological inflammatory indices in LP. Demonstrating that NLR, PLR, and SII do not serve as discriminatory biomarkers for LP, clinicians should avoid overinterpreting these indices in LP assessments.

In conclusion, the findings of this study indicate that LP does not markedly enhance systemic inflammatory activity and that hematologic inflammatory indices have limited value in diagnosing LP or distinguishing clinical variants such as oral involvement. These results support the conceptualization of LP as a predominantly localized immune-mediated inflammatory condition. Future large-scale prospective studies incorporating cytokine profiling, oxidative stress markers, and immunohistochemical biomarkers are needed to elucidate the systemic inflammatory components of LP.

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Author contributions: F.K.B. conceived the study, collected and analyzed the data, interpreted the results, and wrote the manuscript.

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