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ORIGINAL ARTICLE/ORİJİNAL MAKALE

The Association Between Systemic Inflammatory Markers and CIN3+ in Women Positive for HPV 16/18: A Retrospective Study

HPV 16/18 Pozitif Kadınlarda CIN3 Tanısında Sistemik Enflamatuvar Belirteçlerin Yeri: Retrospektif Bir Çalışma

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ABSTRACT

Objectives: To evaluate whether the systemic immune-inflammation index (SII) is independently associated with CIN3+ lesions in women positive for HPV 16 and/or 18.

Methods: This retrospective study included 203 women aged 25 to 65 years with confirmed HPV 16 and/or 18 positivity who underwent colposcopic biopsy. Pre-biopsy complete blood count parameters were used to calculate neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, and SII. Receiver operating characteristic curve analysis was performed to determine the optimal SII cut-off for predicting CIN3+. Multivariate logistic regression analysis was used to assess whether SII independently predicted CIN3+ after adjustment for age, HPV genotype, and hemoglobin level.

Results: Among the evaluated markers, only SII demonstrated a statistically significant association with CIN3+ (p = 0.004). The area under the curve was 0.647. An optimal cut-off value of 809.450 yielded 77.5% sensitivity and 48.4% specificity. In multivariate analysis, SII values at or above this threshold were independently associated with CIN3+ (odds ratio 3.24, 95% confidence interval 1.45 to 7.23; p = 0.004). However, the overall explanatory power of the model was limited (Nagelkerke R^2 = 0.072).

Conclusion: SII was independently associated with CIN3+ in women positive for HPV 16 and/or 18; however, its discriminatory performance was limited. These findings suggest that SII reflects systemic immune-inflammatory status rather than serving as a robust standalone predictive marker.

Keywords: Cervical intraepithelial neoplasia, human papillomavirus, systemic inflammation markers

ÖZET

Amaç: Bu çalışmada, HPV 16 ve/veya 18 pozitif kadınlarda sistemik immün-enflamasyon indeksinin (SII) CIN3+ lezyonlarıyla bağımsız olarak ilişkili olup olmadığını değerlendirmek amaçlandı.

Gereç ve Yöntemler: Retrospektif olarak tasarlanan bu çalışmaya, kolposkopik biyopsi yapılan ve HPV 16 ve/veya 18 pozitifliği saptanan 203 kadın dahil edildi. Biyopsi öncesi tam kan sayımı parametrelerinden nötrofil-lenfosit oranı, trombosit-lenfosit oranı ve sistemik immün-inflamasyon indeksi hesaplandı. CIN3+ öngörüsünde SII'nin performansı için ROC eğrisi analizi yapıldı ve optimal eşik değer Youden indeksi ile belirlendi. Bağımsız ilişkiyi değerlendirmek amacıyla çok değişkenli lojistik regresyon analizi uygulandı.

Sonuçlar: Hastaların %19,7'sinde CIN3+ saptandı. Değerlendirilen inflamatuvar belirteçler arasında yalnızca SII ile CIN3+ arasında anlamlı ilişki bulundu (p=0,004). Eğri altında kalan alan 0,647 olarak hesaplandı. 809,450 eşik değeri için duyarlılık %77,5 ve özgüllük %48,4 idi. Çok değişkenli analizde SII ≥809,450 değeri CIN3+ ile bağımsız olarak ilişkili bulundu (OR: 3,24; %95 GA: 1,45–7,23; p=0,004). Modelin açıklayıcılığı sınırlıydı (Nagelkerke R^2=0,072).

Tartışma: SII, HPV 16 ve/veya 18 pozitif kadınlarda CIN3+ ile bağımsız olarak ilişkilendirilmiştir; ancak ayırt edici performansı sınırlıdır.

Anahtar Kelimeler: Servikal intraepitelyal neoplazi, insan papilloma virüsü, sistemik immün-inflamasyon indeksi

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INTRODUCTION

Human papillomavirus is the principal etiological factor in cervical cancer, a malignancy that remains a significant global public health concern despite the availability of effective screening and vaccination strategies (1,2). The transition from cytology-based screening to primary HPV testing has improved sensitivity for detecting high-grade cervical lesions and has led to the development of risk-based management models (3). The 2019 American Society for Colposcopy and Cervical Pathology guidelines emphasize individualized risk estimation for CIN3+ and recommend management strategies based on calculated risk rather than isolated test results (4,5). Women who test positive for HPV 16 and/or 18 are considered at substantially elevated risk and are therefore referred directly for colposcopic evaluation (6). Nevertheless, high-grade lesions are not identified in a substantial proportion of these patients. Reported detection rates of CIN3+ among HPV 16 or 18 positive women vary widely, and a considerable number of colposcopic procedures do not reveal clinically significant pathology (7-11). This situation raises concerns regarding potential overuse of invasive diagnostic procedures and highlights the need for additional markers that may refine risk stratification within this already high-risk subgroup.

Chronic inflammation plays a well-recognized role in carcinogenesis, including in HPV-related cervical neoplasia (8,12). Inflammatory responses influence tumor initiation, progression, and host immune surveillance. Peripheral blood-derived inflammatory indices such as the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio have been investigated as accessible biomarkers reflecting systemic inflammatory status and have demonstrated prognostic relevance in several malignancies, including cervical cancer (13,14). The systemic immune-inflammation index, calculated as platelet count multiplied by neutrophil count divided by lymphocyte count, integrates both inflammatory and immune components and has been proposed as a composite marker with potentially greater discriminatory ability than individual ratios (9,15). However, data regarding the association between the systemic immune-inflammation index and high-grade cervical intraepithelial lesions are limited, particularly in women positive for HPV 16 and/or 18 (12,13). Given the clinical importance of optimizing triage strategies in this subgroup, evaluation of this index as a potential adjunct marker may provide incremental value within risk-based management frameworks.

The aim of the present study was to determine whether the systemic immune-inflammation index is independently associated with CIN3+ lesions in women positive for HPV 16 and/or 18 and to compare its performance with other systemic inflammatory indices.

MATERIALS AND METHODS

Study Design and Population

Our study included women who underwent colposcopic biopsy between January 2020 and January 2025 at Afyonkarahisar State Hospital following confirmed positivity for HPV 16 and/or 18. HPV genotyping was performed using a polymerase chain reaction-based assay as part of routine cervical cancer screening. A total of 215 women were initially evaluated. Inclusion criteria were age between 25 and 65 years, confirmed HPV 16 and/or 18 positivity, availability of complete blood count parameters obtained within two weeks prior to colposcopy, and accessible histopathological results

from colposcopic biopsy performed at our institution. Exclusion criteria included positivity for other high-risk HPV types without 16 or 18, prior treatment for cervical intraepithelial neoplasia or cervical cancer, incomplete laboratory data, external biopsy without retrievable histopathology, and the presence of acute or chronic inflammatory, autoimmune, or hematologic disorders potentially affecting hematologic parameters. After application of eligibility criteria, 203 women were included in the final analysis. Patients were categorized according to histopathological findings. The non-CIN3+ group included normal histology, chronic cervicitis, CIN1, and CIN2. The CIN3+ group included CIN3 and invasive carcinoma. After applying these criteria, 203 patients were included in the final analysis. Patients were divided into two groups according to their colposcopic biopsy results. Group 1 included those with pathology findings of normal cervix, chronic cervicitis, CIN1, or CIN2. Group 2 included patients diagnosed with CIN3 or invasive carcinoma.

Laboratory Parameters and Calculation of Inflammatory Indices

Peripheral venous blood samples were obtained prior to biopsy. Complete blood counts were analyzed using an automated hematology analyzer. Absolute neutrophil, lymphocyte, and platelet counts were recorded. The neutrophil-to-lymphocyte ratio was calculated as neutrophil count divided by lymphocyte count. The platelet-to-lymphocyte ratio was calculated as platelet count divided by lymphocyte count. The systemic immune-inflammation index was calculated as platelet count multiplied by neutrophil count divided by lymphocyte count.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA). Prior to comparative analyses, continuous variables were assessed for distributional normality using the Kolmogorov–Smirnov test. Normally distributed variables were expressed as mean ± standard deviation and compared using the independent samples Student's t-test. Non-normally distributed variables were summarized as median and range and compared using the Mann–Whitney U test. Categorical variables were presented as frequencies and percentages and compared using the chi-square test or Fisher's exact test, as appropriate. The discriminatory performance of the systemic immune-inflammation index for identifying CIN3+ lesions was evaluated using receiver operating characteristic curve analysis. The area under the curve was calculated as a measure of overall diagnostic performance. The optimal cut-off value was determined using Youden's index, defined as the maximum value of sensitivity plus specificity minus one. Sensitivity and specificity corresponding to the selected threshold were reported. To assess whether SII was independently associated with CIN3+, multivariate logistic regression analysis was conducted. Variables considered clinically relevant and potentially associated with high-grade lesions were included in the model, specifically age, HPV genotype, and hemoglobin level. The SII variable was entered into the model according to the cut-off value identified in ROC analysis. Adjusted odds ratios with 95% confidence intervals were calculated. Model fit was evaluated using the Nagelkerke R^2 statistic to estimate explained variance. A two-sided p-value less than 0.05 was considered statistically significant.

RESULTS

Patient Characteristics

The median age of the cohort was 37 years (range 25–63). Histopathological evaluation identified CIN3+ in 40 patients (19.7%), including 39 cases of CIN3 and 1 case of adenocarcinoma. The remaining 163 patients were classified as non-CIN3+, including normal histology, chronic cervicitis, CIN1, and CIN2. HPV genotype distribution was similar between groups. Overall, 86.2% of patients were positive for HPV 16, 10.3% for HPV 18, and 3.5% for both types. Detailed clinicopathological characteristics are presented in **Table 1**.

Table 1. Clinicopathological characteristics of women positive for HPV 16 and/or 18 according to CIN3+ status

	Group 1 (n=163)	Group 2 (n=40)
Age (years), mean ± SD	38.74 ± 10.1	40.9 ± 10.1
HPV		
Type 16	139 (85.3%)	36 (90.0%)
Type 18	18 (11.0%)	3 (7.5%)
Type 16 and 18	6 (3.7%)	1 (2.5%)
Biopsy result		
Normal	103	-
Chronic cervicitis	22	-
CIN 1	14	-
CIN 2	24	-
CIN 3	-	39
Adenocarcinoma	-	1

HPV: Human papillomavirus, CIN: Cervical intraepithelial neoplasia
Data are presented as mean ± standard deviation or number (percentage), unless otherwise indicated

Association of Systemic Inflammatory Markers with CIN3+

There were no statistically significant differences in neutrophil-to-lymphocyte ratio or platelet-to-lymphocyte ratio between the CIN3+ and non-CIN3+ groups. Mean NLR values were comparable between groups ($p = 0.788$), and PLR did not demonstrate a significant association with high-grade pathology ($p = 0.45$). In contrast, the systemic immune-inflammation index showed a statistically significant association with CIN3+. Receiver operating characteristic curve analysis yielded an area under the curve of 0.647 ($p = 0.004$). The optimal cut-off value determined by Youden's index was 809.450, corresponding to a sensitivity of 77.5% and specificity of 48.4% **(Table 2, Figure 1)**. In multivariate logistic regression analysis adjusting for age, HPV genotype, and hemoglobin level, SII values at or above the identified threshold remained independently associated with CIN3+ (odds ratio 3.24, 95% confidence interval 1.45–7.23; $p = 0.004$). The overall explanatory power of the model was modest (Nagelkerke $R^2 = 0.072$).

Table 2. Receiver operating characteristic curve analysis of the systemic immune-inflammation index for prediction of CIN3+ in women positive for HPV 16 and/or 18

	AUC (95% CI)	Cut-off	Youden's Index	Sensitivity (%)	Specificity (%)	p value
SII	0.647 (0.560 to 0.735)	809.450	0.259	77.5	48.4	0.004

p: ROC Curve Analysis
AUC: Area Under the Curve
SII: Systemic Immune-Inflammation Index
CI: Confidence interval

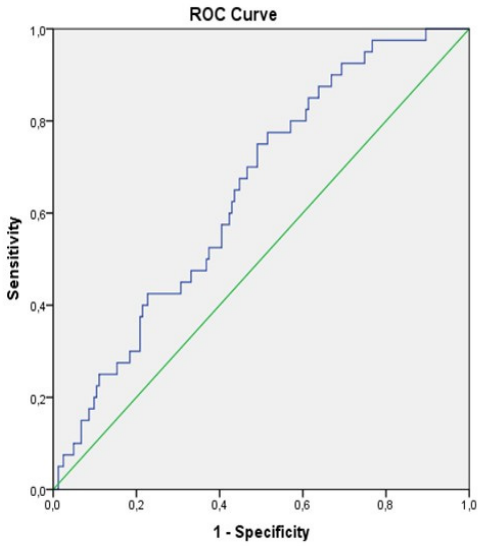

Figure 1. Receiver Operating Characteristic (ROC) curve analysis of the systemic immune-inflammation index (SII) for predicting CIN3+ lesions in women positive for HPV types 16 and/or 18. The area under the curve (AUC) was 0.647 (p = 0.004), with an optimal SII cut-off value of 809.450, yielding a sensitivity of 77.5% and specificity of 48.4%.

DISCUSSION

In this study, we evaluated the association between systemic inflammatory indices and CIN3+ lesions in women positive for HPV 16 and/or 18, a subgroup considered at substantially increased oncogenic risk. Our findings demonstrated that among the evaluated markers, only the systemic immune-inflammation index was independently associated with CIN3+. However, its overall discriminatory performance was modest. Current risk-based management strategies emphasize individualized estimation of CIN3+ probability, particularly following the 2019 ASCCP guideline revision (4,5). Immediate colposcopy is recommended for women positive for HPV 16 or 18 due to the established carcinogenic potential of these genotypes (6). Nevertheless, the proportion of patients ultimately diagnosed with CIN3+ remains limited. In our cohort, CIN3+ was identified in 19.7 percent of patients, indicating that a considerable number of colposcopic evaluations did not reveal high-grade pathology. Similar variability in detection rates has been reported in previous studies (6,11). These observations underscore the ongoing need for adjunctive tools that may improve risk stratification within

this already high-risk population.

The role of chronic inflammation in cervical carcinogenesis has been recognized for decades (7). Persistent HPV infection induces local and systemic immune modulation, contributing to microenvironmental changes that may facilitate neoplastic progression (12,13). In advanced cervical cancer, alterations in peripheral blood parameters, including neutrophilia, lymphopenia, and thrombocytosis, have been documented (14,15). In this context, systemic inflammatory indices such as neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio have been investigated as potential prognostic markers in cervical cancer (10,16). In contrast to some prior studies conducted in patients with invasive cervical cancer, we did not observe significant differences in neutrophil-to-lymphocyte ratio or platelet-to-lymphocyte ratio between women with and without CIN3+. This discrepancy may reflect differences in disease stage, as our population consisted primarily of preinvasive lesions rather than advanced malignancies. It is plausible that systemic inflammatory alterations become more pronounced as tumor burden increases. The systemic immune-inflammation index, which integrates platelet, neutrophil, and lymphocyte counts into a single composite parameter, demonstrated a statistically significant association with CIN3+ in our analysis. This finding is consistent with previous reports suggesting that elevated SII levels may correlate with the severity of cervical lesions (12,13). Qin et al. reported increased SII values in patients with higher-grade cervical pathology and proposed a lower cut-off threshold in their cohort (13). Although cut-off values vary across studies, likely due to population differences and methodological heterogeneity, the overall direction of association appears similar. Despite statistical significance, the discriminatory capacity of SII in our study was limited. The area under the curve was 0.647, and the Nagelkerke R^2 value indicated that only a small proportion of variance in CIN3+ status was explained by the model. These findings suggest that while SII reflects an aspect of systemic immune-inflammation balance associated with lesion severity, it does not provide sufficient accuracy to function as a standalone triage tool. In oncology, elevated SII has been associated with poorer survival outcomes and more aggressive disease behavior in several solid tumors, including cervical cancer (17-19). Recent evidence also suggests that high SII levels may correlate with inferior treatment response and radiotherapy outcomes in cervical cancer patients (20). These observations support the biological plausibility of SII as a marker of tumor-related systemic immune modulation. However, extrapolation from invasive cancer settings to preinvasive lesions should be made cautiously. From a clinical perspective, the potential utility of SII lies not in replacing established screening algorithms but in complementing them. Integration of systemic biomarkers into multifactorial risk models may theoretically enhance predictive precision when combined with cytological findings, virological data, and clinical variables (4,5). Nevertheless, this hypothesis remains speculative and requires prospective validation. Notably, the relatively low specificity observed in our analysis (48.4%) indicates a considerable false-positive rate, which limits the clinical applicability of SII as a triage tool. In practice, this may lead to unnecessary diagnostic procedures if SII is used in isolation. Therefore, its potential role should be considered within combined risk models rather than as a standalone marker.

Several limitations should be acknowledged. The retrospective design, single-center setting, and relatively limited sample size restrict generalizability. The absence of external validation may limit the robustness of the identified cut-off value. Furthermore, potentially relevant behavioral and clinical factors, including smoking status, vaccination history, and cytology results, were not available for analysis and may have influenced lesion risk. These limitations highlight the need for larger, prospective, multicenter studies to clarify the clinical utility of SII in this context. Our findings indicate that SII is independently associated with CIN3+ in women positive for HPV 16 and/or 18, but its predictive performance is modest. Rather than serving as a definitive triage marker, SII may represent a supplementary parameter reflecting systemic immune-inflammatory status within broader risk-based assessment frameworks.

CONCLUSİON

The systemic immune-inflammation index was independently associated with CIN3+ in women positive for HPV 16 and/or 18; however, its predictive performance was limited. SII may serve as a supplementary parameter reflecting systemic immune-inflammatory status rather than a clinically reliable standalone triage tool. Prospective validation is warranted.

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Ethics Committee Approval

The present study was approved by the Ethical Committee of Afyonkarahisar Health Sciences University Hospital (grant no: 2025/8 – date: 13.06.2025). The study was conducted in accordance with the Declaration of Helsinki. The necessary institutional permissions for this study were obtained from Afyonkarahisar Provincial Health Directorate and Afyonkarahisar State Hospital.

Author Contributions:

CYO: Conception and design, writing the article, acquisition of data, statistical analysis, NC: Writing the article, statistical analysis, conception and design, AOG: Acquisition of data, editing the article, conception and design, NO: Editing the article, statistical analysis, ECO: Acquisition of data, writing the article, CD: Acquisition of data, writing the article, DTA: Critical revision of the manuscript, control/supervision, All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Conflict of Interests

The authors declare no conflict of interest.

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Data Availability

Data are available from the lead authors with the permission of Afyonkarahisar State Hospital, Afyonkarahisar, Turkiye.

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