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Relationship between nutritional-inflammatory markers and postoperative outcomes in ovarian cancer: a retrospective study

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Background: Elevated inflammatory markers are commonly linked to poor prognoses in cancer patients, while optimal nutritional status correlates with improved survival outcomes. This study aimed to explore the interplay between nutritional and inflammatory markers and their impact on postoperative outcomes in ovarian cancer patients through a retrospective analysis.

Methods: Data were retrospectively retrieved from patients diagnosed with ovarian cancer who required surgical intervention at the Department of Obstetrics and Gynecology. Overall survival (OS) and cancer-specific survival (CSS) were monitored during follow-up. Kaplan-Meier survival curves were employed to assess OS and CSS across different patient cohorts, evaluating the prognostic significance of nutritional and inflammatory markers. Nomograms for predicting OS and CSS at one, three, and five years postoperatively were constructed, followed by external validation.

Results: The prognostic nutritional index (PNI) and Naples prognostic score (NPS) exhibited a significant correlation with OS and CSS in postoperative ovarian cancer patients (p < 0.05). Analysis indicated that patients with a PNI > 51.2 demonstrated the most favorable survival outcomes. Furthermore, those with a low-NPS (L-NPS) had notably better survival rates compared to their high-NPS (H-NPS) counterparts. Independent OS predictors included age, PNI, NPS, histological type, tumor size, targeted therapy, and diabetes. Similarly, the CSS prediction model incorporated age, NPS, tumor size, targeted therapy, and diabetes. The nomograms demonstrated robust predictive accuracy for three-and five-year survival, though one-year calibration curves showed limited agreement. Despite slightly reduced external validation performance compared to the initial sample, the model maintained strong predictive capability.

Conclusions: The nutritional inflammatory index serves as a key independent prognostic marker for OS and CSS in ovarian cancer patients. Nomograms based on PNI and NPS provide valuable prognostic insights for postoperative management. Incorporating these indices into clinical practice could improve patient stratification and guide personalized treatment plans.

KEYWORDS

nutritional inflammatory markers, PNI, NPS, ovarian cancer, prognosis

1 Introduction

Ovarian cancer ranks among the three most prevalent gynecologic malignancies and remains a leading cause of mortality in this category. Its typically late-stage diagnosis, often resulting from the absence of distinct symptoms and the lack of early-stage screening, significantly compromises clinical outcomes (1, 2). Consequently, the efficacy of current treatments and the prognosis for ovarian cancer remain suboptimal. Modern therapeutic strategies must be informed by molecular profiling, as molecular biology plays a central role in shaping treatment paradigms (3). Increasing evidence underscores the complex interplay between nutrition, inflammation, and cancer prognosis, spurring ongoing investigations into these interactions across various malignancies, including ovarian cancer.

Several scoring systems that assess inflammation and nutritional status, including the lymphocyte-to-monocyte ratio (LMR), systemic inflammatory score (SIS), systemic immune-inflammatory index (SII), neutrophil-to-lymphocyte ratio (NLR), and COUNT, have demonstrated clinical relevance in gynecological cancers (4, 5). Among these, the prognostic nutritional index (PNI), Naples prognostic score (NPS), NLR, and LMR are particularly notable as nutritional-inflammation markers. PNI, initially introduced by Onodera (6), serves as a key indicator of both nutritional and inflammatory conditions, calculated from serum albumin levels and lymphocyte counts. Recent studies have revealed a strong correlation between PNI and prognosis in various cancers, as well as conditions like myocardial infarction and congenital heart disease (7, 8). Similarly, the NPS, developed in 2017 (9), integrates both inflammatory and nutritional biomarkers-albumin, total cholesterol (TC), NLR, and LMR-offering a comprehensive assessment of immune and nutritional status. The NPS has been validated as a reliable predictor of prognosis in multiple cancers, including colon, gallbladder, endometrial, and lung cancers (10-12). Emerging data emphasize the prognostic role of nutritional status in cancer patients, suggesting that markers like PNI and NPS may notably impact OS and cancerspecific survival (CSS) in ovarian cancer.

Elevated inflammatory markers often correlate with poor prognosis in cancer patients, while improved nutritional status is linked to enhanced survival outcomes. However, the specific influence of nutritional markers on ovarian cancer prognosis remains inadequately studied, revealing a notable research gap. This retrospective study seeks to explore the relationship between nutritional inflammatory markers and postoperative prognosis in ovarian cancer patients.

2 Methods

2.1 Study population

Between January 2022 and December 2023, this study enrolled 199 patients from the Department of Obstetrics and Gynecology at the Second People's Hospital Affiliated to Suzhou University, along with 120 patients from the Department of Obstetrics and Gynecology at Binhai County People's Hospital in Yancheng City, who were diagnosed with ovarian cancer and required surgical intervention. The study received approval and oversight from the Ethics Committee of the Second People's Hospital Affiliated of Soochow University (Approval No. JD-LK-2022-079-01) and Binhai County People's Hospital in Yancheng City (Approval No. 2024-BYKYLL-041). The study was conducted in accordance with the revised 2013 Helsinki Declaration. Written informed consent was obtained from each participant or their legal representative.

The inclusion criteria were as follows: (1) age \geq 18 years; (2) a confirmed diagnosis of ovarian cancer requiring surgical intervention, as verified by a physician; and (3) provision of signed informed consent by the patient.

The exclusion criteria were as follows: (1) incomplete laboratory data; (2) a diagnosis of ovarian cancer without surgical treatment; and (3) refusal to provide informed consent.

2.2 Clinical data collection and follow-up

The study included a broad spectrum of variables: (1) Demographic data: age and body mass index (BMI); (2) Laboratory markers: TC, albumin, neutrophil, lymphocyte, and monocyte counts, carbohydrate antigen 125 (CA125), CA199, human epididymis secretory protein 4 (HE4), and carcinoembryonic antigen (CEA); (3) Comorbid conditions: hypertension and diabetes; (4) Tumor characteristics: tumor size, type of surgery (PDS or IDS), residual tumor post-surgery (yes or no), coronavirus disease 2019 (COVID-19) infection (yes or no), pathological type, tissue type, lymph node metastasis, International Federation of Gynecology and Obstetrics (FIGO) grade, differentiation status, history of chemotherapy, and targeted therapy; and (5) Length of hospital stay. Trained professionals collected laboratory markers and tumor characteristics. Biochemical analyses were conducted using a standardized automated system under the supervision of laboratory physicians, while pathologists determined tumor diagnoses and grading. Data on preexisting conditions were extracted from patients' medical histories, and the hospital discharge system recorded the length of stay. Follow-up data were obtained through outpatient visits or telephone interviews. Survival outcomes were assessed at one, three, and five years post-surgery. The primary endpoints of the study were OS and CSS.

The study stratified variables according to criteria established by X-tile software (Figure 1). Age was categorized into three groups: \leq 59 years, 60–69 years, and \geq 70 years. Tumor size was classified as <8 cm, 8–12 cm, and >12 cm.

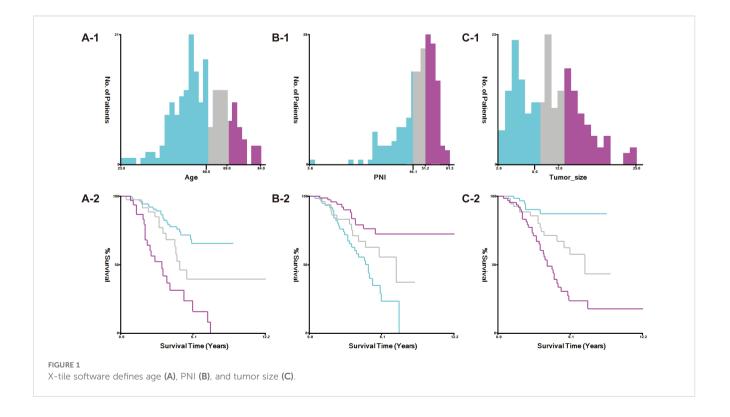
2.3 Calculation of nutritional inflammatory markers

The nutritional inflammatory markers, NLR, LMR, PNI, and NPS, were calculated using the following formulas: (1) NLR as the ratio of neutrophil to lymphocyte counts; (2) LMR as the ratio of lymphocyte to monocyte counts; (3) PNI as albumin (g/L) plus five times the lymphocyte count; and (4) NPS, determined based on serum albumin, TC, NLR, and LMR levels following established methodologies. Using X-tile software, PNI was stratified into three categories: <46.1, 46.1–51.2, and >51.2 (Figure 1). NPS was assigned

based on these criteria: (1) a score of 0 for serum albumin \geq 40 g/L, TC >4.68 mmol/L, NLR <2.96, and LMR >4.44; (2) a score of 1 for serum albumin <40 g/L, TC ≤4.68 mmol/L, NLR ≥2.96, and LMR ≤4.44. The total NPS was calculated by summing the scores of these four parameters. Cumulative NPS values of 0–2 were categorized as low-NPS (L-NPS), while scores of 3–4 were defined as high-NPS (H-NPS).

2.4 Statistical analysis

The baseline characteristics of all enrolled patients were stratified according to survival outcomes. Non-normally distributed variables were expressed as quartile ranges and analyzed using the Wilcoxon rank-sum test. Categorical variables were presented as percentages and compared with chi-square tests. Univariate and multivariate Cox regression models were applied to calculate hazard ratios (HRs) and assess the independence of nutritional inflammatory indices as prognostic factors for OS and CSS in ovarian cancer patients. Kaplan-Meier curves were generated to illustrate OS and CSS across patient groups. To further examine the relationship between nutritional inflammatory markers and survival outcomes, restricted cubic spline (RCS) plots were constructed. A nomogram incorporating statistically significant markers from the multivariate Cox regression was developed. The nomogram's diagnostic performance was evaluated using receiver operating characteristic (ROC) curves and calibration plots. ROC curves were also used to assess the prognostic value of different markers for ovarian cancer patients. External validation was performed concurrently. Finally, stratified analyses were conducted to explore the effects of various nutritional inflammatory indices on subpopulations of ovarian



cancer patients. Statistical analyses were performed with R software (version 4.3.0) and STATA 17.0 (64-bit). A bilateral *p*-value <0.05 was considered statistically significant.

3 Results

3.1 Demographic and clinical characteristics of patients with ovarian cancer

A cohort of 199 ovarian cancer patients who underwent surgical treatment at the Second Affiliated Hospital of Suzhou University was analyzed. Of these, 141 patients (70.85%) survived, while 58 patients (29.15%) succumbed to the disease. The demographic and clinical characteristics of both groups were summarized in Table 1. Increased mortality risk was associated with advanced age, lower albumin levels, reduced lymphocyte counts, decreased PNI, elevated neutrophil counts, higher NLR, increased HE4, H-NPS, and postoperative tumor residue. Additional high-risk factors included high-grade histology, larger tumor size, FIGO stage IV, poor tumor differentiation, absence of targeted therapy, and a history of diabetes. Statistically significant differences between survivors and deceased patients were observed for all these variables (all p < 0.05). Furthermore, deceased patients had longer hospital stays compared to survivors. No significant differences were found regarding monocyte counts, CA125, CA199, CEA, tumor histological type, surgical procedure, COVID-19 infection, or lymphatic metastasis. The lack of statistical significance in postoperative survival and prognosis may be attributed to the small sample size of this study.

An analysis of 120 patients from Binhai County People's Hospital (Table 1) yielded results consistent with those from the Second Affiliated Hospital to Suzhou University. Factors including advanced age, elevated BMI, low albumin levels, reduced lymphocyte count, increased NLR, elevated HE4, low PNI, H-NPS, lymphocyte metastasis, poor differentiation, absence of radiotherapy or targeted therapy, residual tumor post-surgery, and comorbid diabetes were all significantly associated with a higher risk of mortality (all p<0.05). In contrast, neutrophil levels did not exhibit statistical significance in the Binhai County cohort, which may be attributed to the smaller sample size in our study.

3.2 Identification of prognostic factors affecting OS and CSS in ovarian cancer postoperatively

Kaplan-Meier survival analysis revealed significant associations between PNI, NPS, and survival outcomes in ovarian cancer patients (p < 0.05), as depicted in Figure 2. Patients with PNI > 51.2 exhibited the best OS, whereas those with PNI < 46.1 had the poorest prognosis (Figures 2A-1). Likewise, survival outcomes were significantly better for patients with L-NPS compared to those with H-NPS (Figure 2A-2). These patterns were similarly observed for CSS, as shown in Figures 2A-3, 2A-4. Consistent results were obtained from the data of Binhai County People's Hospital (Figures 2B-1–4).

Univariate and multivariate Cox regression analyses were conducted to identify factors influencing OS and CSS. For OS, independent risk factors included age, PNI, NPS, tissue type, tumor size, targeted therapy status, and diabetes (Table 2). Specifically, patients with PNI between 46.1 and 51.2 demonstrated improved survival outcomes compared to those with PNI <46.1 (HR = 0.461; 95% CI, 0.234-0.864; p = 0.036), while those with PNI >51.2 exhibited even more pronounced protective effects (HR = 0.357; 95% CI, 0.095–0.538; *p* = 0.025). In contrast, patients with H-NPS had significantly worse survival compared to those with L-NPS (HR = 1.576; 95% CI, 1.252–2.719; *p* = 0.013). For CSS, multivariate Cox regression analysis (Table 3) identified age, NPS, tumor size, targeted therapy status, and diabetes as independent risk factors. Consistent with OS, patients with H-NPS experienced poorer outcomes compared to those with L-NPS (HR = 1.654; 95% CI, 1.278 - 3.138; p = 0.031).

3.3 Relationship between nutritional inflammatory markers and survival prognosis of postoperative patients with ovarian cancer

An RCS diagram (Figure 3) was constructed to explore the relationship between nutritional inflammatory markers and survival prognosis in postoperative ovarian cancer patients. OS analysis indicated a negative correlation between PNI and LMR with patient prognosis, suggesting that higher values of these indices were associated with improved survival outcomes (Figures 3A, C). In contrast, NLR exhibited a positive correlation with prognosis, with elevated NLR values linked to poorer survival (Figure 3B). The CSS analysis mirrored the findings from OS, revealing a negative correlation between both PNI and LMR with prognosis (Figures 3D, F), while NLR remained positively correlated, indicating worse outcomes as its value increased (Figure 3E). Additional analyses included markers such as albumin, TC, CA125, CA199, CEA, and HE4 (Supplementary Figure S1).

3.4 Construction and evaluation of the nomogram

Univariate and multivariate Cox regression analyses identified seven independent predictors of OS: age, PNI, NPS, tissue type, tumor size, targeted therapy use, and diabetes presence (p < 0.05; Table 2). Similarly, five variables were determined to be independent predictors of CSS (p < 0.05; Table 3). Nomograms incorporating these predictors were developed to estimate one-, three-, and five-year OS and CSS (Figures 4A, D). Risk scores for each factor in the nomogram, shown in Figure 4, indicated that higher scores corresponded to increased mortality risk. The predictive accuracy of the nomograms was assessed, yielding a concordance (C-index) of 0.715 for the OS nomogram and 0.704 for the CSS nomogram. ROC curves for one-, three-, and five-year OS and CSS were plotted, with the area under the curve (AUC) values for OS being 0.722, 0.801, and 0.890, respectively (Figure 4B). For

TABLE 1 Baseline demographic and clinical characteristics of patients with ovarian cancer.

Characteristic	Total No. (%)	The Second Peo Affiliated of Sood	ple's Hospital chow University	P value	Total No. (%)	Binhai County Pe	eople's Hospital	P value
		Survival, No. (%)	Dead, No. (%)			Survival, No. (%)	Dead, No. (%)	
Total	199	141 (70.85)	58 (29.15)		120	86	34	
Age, years				<0.001				< 0.001
≤59	114 (57.3%)	94 (66.7%)	20 (34.5%)		65 (54.2%)	55 (45.8%)	10 (8.3%)	
60-69	52 (26.1%)	35 (24.8%)	17 (29.3%)		39 (32.5%)	26 (21.7%)	13 (10.8%)	
≥70	33 (16.6%)	12 (8.5%)	21 (36.2%)		16 (13.3%)	5 (4.2%)	11 (9.2%)	
BMI	24.64(22.41, 28.58)	24.27 (22.15, 27.70)	27.06 (23.63, 30.14)	0.005	25.28 (22.64, 28.80)	25.42 ± 3.84	28.08 ± 6.31	0.027
Albumin	42.8 (38, 45.9)	43.9 (39.5, 46.9)	39.65 (34.8, 44.2)	< 0.001	41.14 ± 4.60	42.17 ± 4.13	38.54 ± 4.77	< 0.001
Total cholesterol	4.2 ± 1.25	4.41 ± 1.20	4.05 ± 1.33	0.064	4.44 ± 1.05	4.48 ± 1.06	4.32 ± 1.02	0.435
Lymphocyte	1.3 (1, 1.6)	1.4 (1.1, 1.7)	1.2 (0.93, 1.5)	0.010	1.33 (1.1, 1.7)	1.4 (1.11, 1.79)	1.22 (1.06, 1.41)	0.013
Neutrophil	4.3 (3.3, 6)	4.2 (3.2, 5.8)	5 (3.73, 6.68)	0.037	4.24 (3.21, 6.32)	4.15 (3.26, 5.46)	4.86 (3.15, 8.18)	0.137
Monocyte	0.4 (0.3, 0.5)	0.4 (0.3, 0.5)	0.4 (0.3, 0.5)	0.688	0.37 (0.26, 0.56)	0.37 (0.25, 0.57)	0.40 (0.3, 0.54)	0.358
NLR	3.25 (2.21, 4.96)	2.88 (2.15, 4.63)	4.11 (2.72, 5.93)	0.011	3.12 (1.94, 4.87)	2.84 (1.94, 4.61)	4.39 (2.94, 5.52)	0.031
LMR	3.67 (2.33, 5.0)	3.75 (2.5, 5.0)	3 (2, 4.63)	0.093	3.41 (2.52, 5.0)	3.5 (2.64, 5.39)	3.27 (2.0, 4.0)	0.055
CA125	297 (62.73, 969)	295.5 (50.08, 920.25)	331.7 (100, 969)	0.438	348 (102, 625)	289 (67, 568)	316 (375, 604)	0.211
CA199	12.08 (6.56, 23)	10.65 (6.40, 23.35)	13.92 (7.03, 22.8)	0.416	23.27 (11.86, 93.84)	22.38 (12.38, 83.84)	24.10 (11.07, 210.88)	0.606
CEA	1.4 (0.86, 2.33)	1.385 (0.86, 2.29)	1.56 (0.92, 2.62)	0.710	3.4 (2.18, 7.85)	2.97 (2.14, 6.08)	3.07 (2.34, 9.55)	0.088
HE4	167 (73.67, 458.2)	134.5 (67, 367.77)	202.6 (111.78, 536.38)	0.042	269.7 (149.41, 412.2)	245.75 (121.18, 406.05)	313.5 (252.45, 431.2)	0.020
PNI, n (%)				< 0.001				< 0.001
<46.1	61 (30.7%)	31 (22.0%)	30 (51.7%)		39 (32.5%)	20 (16.7%)	19 (15.8%)	
46.1-51.2	57 (28.6%)	42 (29.8%)	15 (25.9%)		44 (36.7%)	33 (27.5%)	11 (9.2%)	
>51.2	81 (40.7%)	68 (48.2%)	13 (22.4%)		37 (30.8%)	33 (27.5%)	4 (3.3%)	
NPS, n (%)				0.003				< 0.001
L-NPS	101 (50.8%)	81 (57.4%)	20 (34.5%)		77 (64.2%)	64 (53.3%)	13 (10.8%)	
H-NPS	98 (49.2%)	60 (42.6%)	38 (65.5%)		43 (35.8%)	22 (18.3%)	21 (17.5%)	
Pathological type, n (%)				0.362				0.087

TABLE 1 Continued

Characteristic	Total No. (%)	The Second Peo Affiliated of Sood	ple's Hospital chow University	P value	Total No. (%)	Binhai County Pe	eople's Hospital	P value
		Survival, No. (%)	Dead, No. (%)			Survival, No. (%)	Dead, No. (%)	
Serous adenocarcinoma	140 (70.4%)	95 (67.4%)	45 (77.6%)		81 (67.5%)	54 (45%)	27 (22.5%)	
Endometrioid adenocarcinoma	28 (14.1%)	20 (14.2%)	8 (13.8%)		17 (14.2%)	16 (13.3%)	1 (0.8%)	
Mucinous adenocarcinoma	14 (7%)	12 (8.5%)	2 (3.4%)		14 (11.7%)	9 (7.5%)	5 (4.2%)	
Clear cell carcinoma	17 (8.5%)	14 (9.9%)	3 (5.2%)		8 (6.7%)	7 (5.8%)	1 (0.8%)	
Histology type, n (%)				0.021				0.064
High-grade	168 (84.4%)	113 (80.1%)	55 (94.8%)		91 (75.8%)	62 (51.7%)	29 (24.2%)	
Medium-grade	9 (4.5%)	7 (5%)	2 (3.4%)		11 (9.2%)	7 (5.8%)	4 (3.3%)	
Low-grade	22 (11.1%)	21 (14.9%)	1 (1.7%)		18 (15%)	17 (14.2%)	1 (0.8%)	
Lymph node metastasis, n (%)				0.076				< 0.001
No	95 (47.7%)	73 (51.8%)	22 (37.9%)		57 (47.5%)	50 (41.7%)	7 (5.8%)	
Yes	104 (52.3%)	68 (48.2%)	36 (62.1%)		63 (52.5%)	36 (30%)	27 (22.5%)	
Tumor size				<0.001				0.295
<8	65 (32.7%)	59 (41.8%)	6 (10.3%)		34 (28.3%)	27 (22.5%)	7 (5.8%)	
8-12	68 (34.2%)	52 (36.9%)	16 (27.6%)		45 (37.5%	33 (27.5%)	12 (10%)	
>12	66 (33.2%)	30 (21.3%)	36 (62.1%)		41 (34.2%)	26 (21.7%)	15 (12.5%)	
FIGO stage, n (%)				0.055				< 0.001
Grade I	47 (23.6%)	40 (28.4%)	7 (12.1%)		28 (23.3%)	28 (23.3%)	0 (0%)	
Grade II	27 (13.6%)	19 (13.5%)	8 (13.8%)		16 (13.3%)	13 (10.8%)	3 (2.5%)	
Grade III	105 (52.8%)	71 (50.4%)	34 (58.6%)		61 (50.8%)	40 (33.3%)	21 (17.5%)	
Grade IV	20 (10.1%)	11 (7.8%)	9 (15.5%)		15 (12.5%)	5 (4.2%)	10 (8.3%)	
Degree of differentiation, n (%)				0.007				0.005
Low-differentiated	167 (83.9%)	111 (78.7%)	56 (96.6%)		98 (81.7%)	64 (53.3%)	34 (28.3%)	
Medium-differentiated	9 (4.5%)	8 (5.7%)	1 (1.7%)		6 (5%)	6 (5%)	0 (0%)	
High-differentiated	23 (11.6%)	22 (15.6%)	1 (1.7%)		16 (13.3%)	16 (13.3%)	0 (0%)	
Chemotherapy, n (%)				0.569				0.007

(Continued)

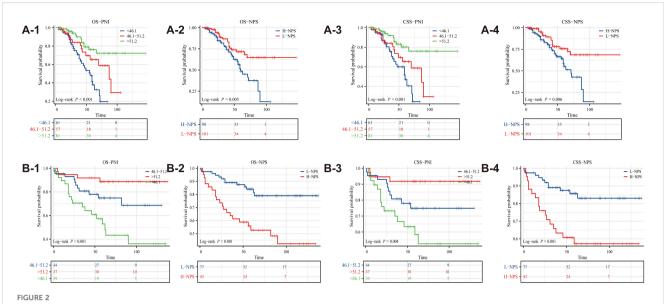
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TABLE 1 Continued

Characteristic	Total No. (%)	The Second Peo Affiliated of Sood	ple's Hospital chow University	P value	Total No. (%)	Binhai County Pe	eople's Hospital	P value
		Survival, No. (%)	Dead, No. (%)			Survival, No. (%)	Dead, No. (%)	
No	21 (10.6%)	16 (11.3%)	5 (8.6%)		17 (14.2%)	7 (5.8%)	10 (8.3%)	
Yes	178 (89.4%)	125 (88.7%)	53 (91.4%)		103 (85.8%)	79 (65.8%)	24 (20%)	
Targeted therapy, n (%)				< 0.001				< 0.001
No	128 (64.3%)	74 (52.5%)	54 (93.1%)		91 (75.8%)	57 (47.5%)	34 (28.3%)	
Yes	71 (35.7%)	67 (47.5%)	4 (6.9%)		29 (24.2%)	29 (24.2%)	0 (0%)	
Type of surgery, n (%)				0.085				0.030
PDS	191 (96.0%)	138 (97.9%)	53 (91.4%)		113 (94.2%)	84 (70%)	29 (24.2%)	
IDS	8 (4.0%)	3 (2.1%)	5 (8.6%)		7 (5.8%)	2 (1.7%)	5 (4.2%)	
Residual tumoral surgery, n (%)				<0.001				<0.001
No	175 (87.9%)	134 (95.0%)	41 (70.7%)		86 (71.7%)	79 (65.8%)	7 (5.8%)	
Yes	24 (12.1%)	7 (5.0%)	17 (29.3%)		34 (28.3%)	7 (5.8%)	27 (22.5%)	
COVD-19 infection				0.385				0.185
No	181 (90.95%)	133 (94.33%)	48 (82.76%)		99 (82.5%)	70 (58.33%)	29 (24.16%)	
Yes	18 (9.05%)	8 (5.67%)	10 (17.24%)		21 (17.5%)	16 (13.33%)	5 (4.17%)	
Hospital time	16 (14, 21)	16 (13, 20)	18.5 (14, 23)	0.036	18 (14, 23)	16 (14, 20)	21 (16.25, 24)	0.032
Hypertension, n (%)				0.583				0.506
No	136 (68.3%)	98 (69.5%)	38 (65.5%)		83 (69.2%)	61 (50.8%)	22 (18.3%)	
Yes	63 (31.7%)	43 (30.5%)	20 (34.5%)		37 (30.8%)	25 (20.8%)	12 (10%)	
Diabetes, n (%)				< 0.001				< 0.001
No	170 (85.4%)	129 (91.5%)	41 (70.7%)		103 (85.8%)	81 (67.5%)	22 (18.3%)	
Yes	29 (14.6%)	12 (8.5%)	17 (29.3%)		17 (14.2%)	5 (4.2%)	12 (10%)	

Percentages may not total 100 because of rounding. Bold values indicates p<0.05.

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Kaplan-Meier survival curves evaluated the effects of PNI and NPS on survival outcomes (A1-A4: The Second People's Hospital Affiliated of Soochow University; B1-B4: Binhai County People's Hospital).

TABLE 2 Univariate analysis of overall survival (OS) in patients with ovarian cancer.

	OS								
Characteristic	Univariate analysis	Multivariate analysis							
	Hazard Ratio (95% CI)	<i>P</i> value	Hazard Ratio (95% CI)	<i>P</i> value					
Age, years			Hazard Ratio (95% Cl) P value Reference 1.223 (0.583,2.564) 0.594 1.223 (0.583,2.564) 0.002 1 3.320 (1.565,7.046) 0.002 1 - - 1.052 (0.978,1.132) 0.170 - 1 - - 1 1.000 (0.798,1.254) 0.999 - - -						
≤59	Reference		Reference						
60-69	1.852 (0.969,3.542)	0.062	1.223 (0.583,2.564)	0.594					
≥70	5.158 (2.784,9.556)	<0.001	3.320 (1.565,7.046)	0.002					
BMI	1.006 (0.963,1.050)	0.801	-	-					
Albumin	0.951 (0.922,0.981)	0.001	1.052 (0.978,1.132)	0.170					
Total cholesterol	0.920 (0.751,1.128)	0.423	-	-					
Lymphocyte	0.671 (0.364,1.235)	0.200	-	-					
Neutrophil	1.157 (1.073,1.248)	< 0.001	1.000 (0.798,1.254)	0.999					
Monocyte	1.460 (0.870,2.448)	0.152	-	-					
NLR	1.071 (1.031,1.113)	< 0.001	1.038 (0.940,1.146)	0.459					
LMR	0.920 (0.799,1.059)	0.245	-	-					
CA125	1.000 (1.000,1.000)	0.461	-	-					
CA199	1.000 (0.999,1.001)	0.679	-	-					
CEA	1.002 (1.000,1.004)	0.087	-	-					
HE4	1.001 (1.000,1.001)	0.103	-	-					
PNI, n (%)	·	·	·	•					
<46.1	Reference		Reference						
46.1-51.2	0.517 (0.276,0.970)	0.040	0.461 (0.234,0.864)	0.036					

TABLE 2 Continued

Univariate analysisMultivariate analysisMultivariate analysisP valueHazard Ratio (95% Cl)P valueHazard Ratio (95% Cl)P valuePNI, n (%)S120.259 (0.133,0503)<0.0010.357 (0.095,0538)0.025NPS, n (%)LNPSReferenceReferenceLNPS2.139 (1.238,3696)0.0061.576 (1.252,2.719)0.013Pathological type, n (%)Serous adenocarcinomaReferenceSerous adenocarcinomaReferenceMucinous adenocarcinoma0.410 (0.099,1.691)0.217Nucinous adenocarcinoma0.410 (0.099,1.691)0.257Mucinous adenocarcinoma0.576 (0.179,1.854)0.355Histology type, n (%)Low-gradeReference0.0275.060 (1.106,10.722)0.039Medium-grade0.307 (0.551,6.091)0.0171.214 (0.466,2.709)0.795High-grade3.011 (1.23,3.258)0.0171.124 (0.466,2.709)0.795Yes1.912 (1.123,3.258)0.0171.124 (0.466,2.709)0.795Yes1.912 (1.23,3.258)0.0171.124 (0.466,2.709)0.795Furmor sizeSerenceSerenceSerenceSerenceSerence68Reference0.0661.502 (0.509,44.32)0.6165122.792 (2.45,13.769)<0.0015.011 (1.858,13.511)0.0015145.799	
Produe Produe Hazard Ratio (9% CI) Produe PNI, n (%) >51.2 0.259 (0.133.0503) <0.001	
51.20.259 (0.133,0.503)<0.0010.357 (0.095,0.538)0.025NPS, n (%)L-NPSReferenceReferenceReference0.006H-NPS2.139 (1.238,3.696)0.0061.576 (1.252,2.719)0.013Pathological type, n (%)Serous adenocarcinomaReferenceIndometrioid adenocarcinoma0.431 (0.397,1.790)0.657Mucinous adenocarcinoma0.410 (0.099,1.691)0.217Clear cell carcinoma0.576 (0.179,1.854)0.355Histology type, n (%)Low-gradeReferenceInfanceReferenceInfanceInfanceInfanceMedium-grade3.078 (0.551,6.091)0.1413.086 (0.551,6.183)0.1401.1400.039 <td< td=""><td></td></td<>	
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Serous adenocarcinoma Reference Image: Constraint of the series of the	
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Medium-grade 3.078 (0.551,6.091) 0.141 3.086 (0.551,6.183) 0.140 High-grade 5.321 (1.285,10.635) 0.027 5.060 (1.106,10.722) 0.039 Lymph node metastasis, ×/> No Reference Reference 0.141 1.124 (0.466,2.709) 0.039 Yes 1.912 (1.123,3.258) 0.017 1.124 (0.466,2.709) 0.795 Tumor size Stationary Reference Reference Stationary 0.401 8-12 2.412 (0.943,6167) 0.066 1.502 (0.509,4432) 0.401 >12 5.799 (2.443,13.769) <001	
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>12 5.799 (2.443,13.769) <0.001 5.011 (1.858,13.511) 0.001 FIGO stage, n (%)	
FIGO stage, n (%)	
Grade I Reference Reference	
Grade II 2.544 (0.907,7.135) 0.076 0.698 (0.177,2.753) 0.608	
Grade III 3.024 (1.325,6.902) 0.009 1.432 (0.412,4.975) 0.572	
Grade IV 4.357 (2.633,9.558) < 0.001 1.860 (0.387,8.946) 0.439	
Degree of differentiation, n (%)	
Low-differentiated Reference Reference	
Medium-differentiated 0.238 (0.033,1.734) 0.157 0.060 (0.003,1.420) 0.081	
High-differentiated 0.099 (0.014,0.722) 0.022 0.299 (0.074,1.722) 0.997	
Chemotherapy, n (%)	
No Reference	
Yes 0.635 (0.252,1.600) 0.336	
Targeted therapy, n (%)	
No Reference Reference	
Yes 0.111 (0.040,0.309) < 0.001 0.158 (0.048,0.516) 0.002	

TABLE 2 Continued

	OS								
	Univariate analysis	Multivariate analysis							
Characteristic	Hazard Ratio (95% CI)	<i>P</i> value	Hazard Ratio (95% CI)	P value					
Type of surgery, n (%)									
IDS	Reference		Reference	0.391					
PDS	0.338 (0.134,0.851)	0.021	0.530 (0.124,2.262)						
Residual tumoral surg	ery, n (%)								
No	Reference		Reference						
Yes	3.199 (1.813,5.643)	<0.001	2.106 (0.895,4.956)	0.088					
COVID-19 infection									
No	Reference								
Yes	1.112 (0.823,1.658)	0.321							
Hospital time	1.029 (0.995 - 1.064)	0.096	-	-					
Hypertension, n (%)									
No	Reference		-	-					
Yes	1.545 (0.897,2.660)	0.117	-	-					
Diabetes, n (%)									
No	Reference		Reference						
Yes	2.923 (1.654,5.165)	<0.001	1.603 (1.124,2.921)	0.041					

OS, Overall survival.

Bold values indicates p<0.05.

TABLE 3 Univariate analysis of cancer-specific survival (CSS) in patients with ovarian cancer.

	CSS			
Characteristic	Univariate analysis	Multivariate analysis		
	Hazard Ratio (95% CI)	<i>P</i> value	Hazard Ratio (95% CI)	P value
Age, years				
≤59	Reference		Reference	
60-69	1.602 (0.801,3.201)	0.182	1.116 (0.506,2.459)	0.786
≥70	4.354 (2.253,8.415)	<0.001	2.979 (1.346,6.596)	0.007
BMI	1.009 (0.964,1.056)	0.711	-	-
Albumin	0.957 (0.925,0.990)	0.011	1.067 (0.982,1.159)	0.126
Total cholesterol	0.962 (0.774,1.196)	0.725	-	-
Lymphocyte	0.571 (0.293,1.113)	0.100	-	-
Neutrophil	1.174 (1.084,1.271)	<0.001	0.995 (0.789,1.256)	0.969
Monocyte	1.507 (0.886,2.562)	0.130	-	-
NLR	1.074 (1.030,1.120)	<0.001	1.055 (0.957,1.163)	0.280
LMR	0.922 (0.792,1.072)	0.290	-	-
CA125	1.002 (1.000,1.004)	0.314	-	-

TABLE 3 Continued

	CSS				
Characteristic	Univariate analysis	Multivariate analysis			
	Hazard Ratio (95% CI)	<i>P</i> value	Hazard Ratio (95% CI)	P value	
Age, years					
CA199	1.000 (0.998,1.001)	0.596	-	-	
CEA	1.002 (1.000,1.004)	0.058	-	_	
HE4	1.001 (1.000,1.001)	0.072	-	-	
PNI, n (%)					
<46.1	Reference		Reference		
46.1-51.2	0.633 (0.328,1.220)	0.172	0.690 (0.236,2.022)	0.499	
>51.2	0.268 (0.129,0.555)	<0.001	0.321 (0.077,1.336)	0.118	
NPS, n (%)					
L-NPS	Reference		Reference		
H-NPS	2.230 (1.234,4.030)	0.008	1.654 (1.278,3.138)	0.031	
Pathological type, n (%)					
Serous adenocarcinoma	Reference		-	-	
Endometrioid adenocarcinoma	0.865 (0.386,1.936)	0.723	-	-	
Mucinous adenocarcinoma	0.478 (0.115,1.982)	0.309	-	-	
Clear cell carcinoma	0.439 (0.106,1.819)	0.256	-	-	
Histology type, n (%)			1		
Low-grade	Reference		Reference		
Medium-grade	3.086 (0.551,6.183)	0.140	2.086 (0.351,4.183)	0.540	
High-grade	5.060 (1.106,10.722)	0.039	3.060 (0.706,5.722)	0.340	
Lymph node metastasis, r	n (%)				
No	Reference		Reference		
Yes	1.905 (1.073,3.381)	0.028	1.167 (0.442,3.085)	0.755	
Tumor size					
<8	Reference		Reference		
8-12	2.713 (0.985,7.470)	0.053	1.680 (0.561,5.030)	0.354	
>12	5.789 (2.245,14.927)	<0.001	4.163 (1.529,8.333)	0.005	
FIGO stage, n (%)			1		
Grade I	Reference		Reference		
Grade II	3.104 (1.052,9.159)	0.040	0.933 (0.220,3.959)	0.925	
Grade III	2.886 (1.174,7.092)	0.021	1.372 (0.346,5.444)	0.653	
Grade IV	4.899 (3.012,8.286)	<0.001	2.671 (0.514,13.868)	0.242	
Degree of differentiation,	n (%)				
Low-differentiated	Reference		Reference		
Medium-differentiated	0.272 (0.037,1.993)	0.200	0.229 (0.026,2.021)	0.185	
High-differentiated	0.114 (0.016,0.835)	0.032	0.222 (0.026,1.878)	0.167	

TABLE 3 Continued

	CSS			
	Univariate analysis	Multivariate analysis		
Characteristic	Hazard Ratio (95% CI)	<i>P</i> value	Hazard Ratio (95% CI)	P value
Chemotherapy, n (%)				
No	Reference		-	-
Yes	0.545 (0.215,1.383)	0.201	-	-
Targeted therapy, n (%)				
No	Reference		Reference	
Yes	0.129 (0.046,0.361)	<0.001	0.204 (0.062,0.671)	0.009
Type of surgery, n (%)				
IDS	Reference		Reference	0.469
PDS	0.288 (0.114,0.732)	0.009	0.585 (0.137,2.495)	
Residual tumoral surgery,	n (%)			
No	Reference		Reference	
Yes	2.988 (1.608,5.552)	<0.001	1.537 (0.588,4.015)	0.381
COVID-19 infection				
No	Reference			
Yes	1.232 (0.873,1.418)	0.221		
Hospital time	1.031 (0.995,1.068)	0.089	-	-
Hypertension, n (%)		·	·	·
No	Reference		-	-
Yes	1.372 (0.756,2.491)	0.298	-	-
Diabetes, n (%)			· 	
No	Reference		Reference	
Yes	2.739 (1.471,5.100)	0.001	2.016 (1.459,2.209)	0.024
S Overall survival				<u> </u>

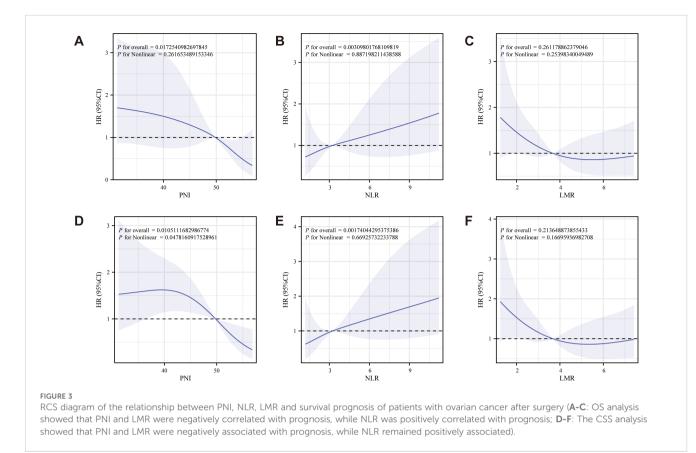
OS, Overall survival. Bold values indicates p<0.05.

CSS, the AUC values were 0.708, 0.827, and 0.840, respectively (Figure 4E). Calibration curves for three- and five-year predictions closely aligned with the diagonal, indicating high consistency and reliable calibration, whereas the one-year calibration curves deviated, suggesting lower robustness in predicting one-year outcomes (Figures 4C, F).

External validation was performed using data from 120 patients at Binhai County People's Hospital, applying the same characteristic variables to construct a nomogram for predicting 1-, 3-, and 5-year OS and CSS (Figures 5A, B). The nomogram's risk scores for each factor were presented in Figure 5, where higher scores correlated with an increased risk of mortality. Predictive accuracy was assessed by evaluating the C-index, which was 0.735 for the OS nomogram and 0.714 for the CSS nomogram. ROC curves for 1-year, 3-year, and 5year OS and CSS were also generated. For OS, AUC values were 0.753, 0.782, and 0.724, respectively (Figure 5A-2), while for CSS, AUC values were 0.743, 0.766, and 0.708 (Figure 5B-2). Calibration curves for 3and 5-year predictions closely approximated the diagonal, reflecting strong consistency and reliable calibration. In contrast, the 1-year calibration curve showed notable deviation from the diagonal, indicating reduced predictive accuracy for the 1-year outcome (Figures 5A-3, 5B-3). A comparison with data from the Second People's Hospital of Suzhou University revealed that while model performance at Binhai County People's Hospital was somewhat lower, the evaluation metrics still confirmed the model's overall effectiveness.

3.5 Predictive value of nutritional inflammatory markers for survival and prognosis of postoperative patients with ovarian cancer

Multivariate Cox regression analysis was used to develop a diagnostic nomogram, with correlated variables plotted alongside the ROC curve to evaluate its predictive accuracy for survival and prognosis in postoperative ovarian cancer patients (Figure 6). For



OS, the model exhibited the highest predictive value, yielding an AUC of 0.910 (Figure 6B-1). In comparison, the AUCs for individual variables were 0.678 for PNI, 0.627 for NPS, 0.424 for tissue type, 0.743 for tumor size, and 0.703 for targeted therapy (Figure 6A-1). In the CSS analysis, the model again demonstrated the highest predictive value, achieving an AUC of 0.844 (Figure 6B-2). The AUCs for other variables were 0.612 for NPS, 0.722 for tumor size, and 0.685 for targeted therapy (Figure 6A-2). These results confirm the nomogram's significant diagnostic utility, highlighting its capacity to accurately predict survival and prognosis in postoperative ovarian cancer patients.

Data analysis from Binhai County People's Hospital revealed that the OS model exhibited the highest predictive value, with an AUC of 0.898 (Figure 6B-3). In contrast, the AUC values for other variables were as follows: PNI, 0.703; NPS, 0.681; tissue type, 0.576; tumor size, 0.618; and targeted therapy, 0.669 (Figure 6A-3). Likewise, for CSS, the model again demonstrated the highest predictive value, with an AUC of 0.848 (Figure 6B-4). The AUC values for additional variables were: NPS, 0.662; tumor size, 0.604; and targeted therapy, 0.658 (Figure 6A-4). These results further substantiate the diagnostic utility of the nomogram, reinforcing its robust predictive capability for survival and prognosis in postoperative ovarian cancer patients.

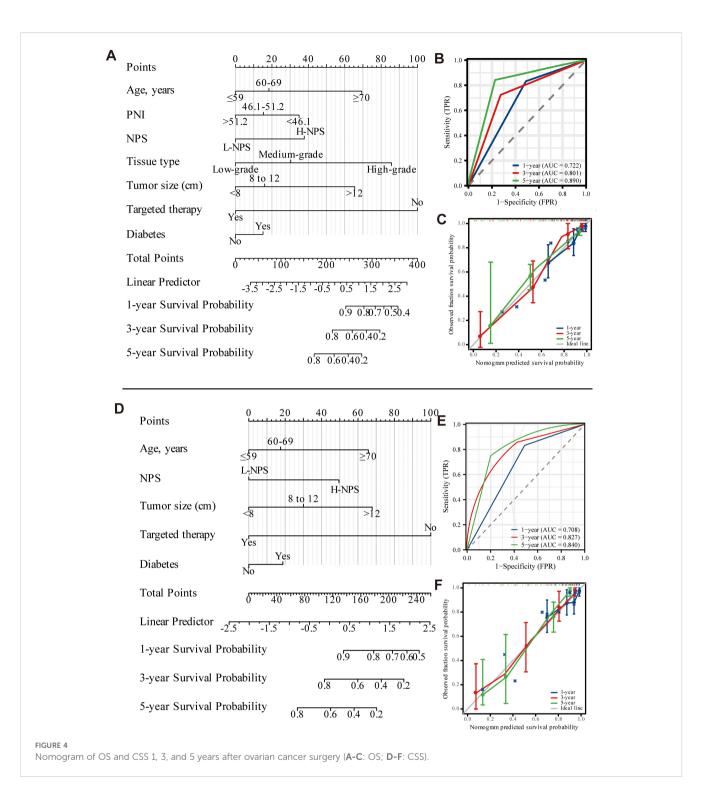
3.6 Subgroup analysis

Subgroup analysis of OS and CSS in postoperative ovarian cancer patients was performed based on factors such as age,

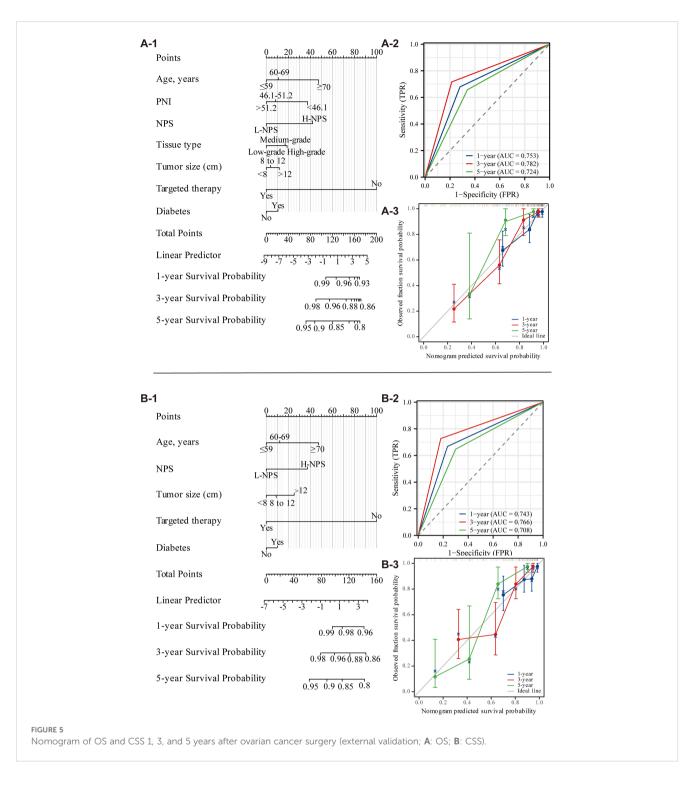
tumor size, histological type, and tissue characteristics to assess survival prognosis. OS analysis using the NPS (Figures 7A-C) revealed that patients aged 60-69 years with H-NPS faced a significantly higher mortality risk than those with L-NPS (HR = 3.534; 95% CI, 1.230–10.151; p = 0.019). Similar patterns were observed in patients with tumors >12 cm, endometrioid adenocarcinoma, FIGO grade I, as well as those undergoing surgery and chemotherapy (all p < 0.05). Additionally, among patients aged 60-69 years, those with tumors >12 cm, serous adenocarcinoma, low-grade histology, no lymphatic metastasis, absence of targeted therapy, and without hypertension or diabetes, demonstrated the lowest mortality when PNI >51.2 (p <0.05) (Figures 7D-F). In terms of CSS, the NPS analysis yielded comparable results (Figures 7G-I), with higher mortality rates in patients with tumors >12 cm and endometrioid adenocarcinoma, regardless of hypertension, surgical procedure, or residual tumor presence (all p < 0.05). Further PNI analysis for CSS (Figures 7J–L) indicated that patients aged 60-69 years with tumors >12 cm, serous adenocarcinoma, high-grade histology, absence of lymphatic metastasis, receiving chemotherapy, without targeted therapy, and regardless of hypertension or diabetes, surgery type, or residual tumor showed the lowest mortality rates when PNI >51.2 (all *p* < 0.05).

4 Discussion

Ovarian cancer ranks among the three most prevalent gynecologic malignancies and poses a significant public health

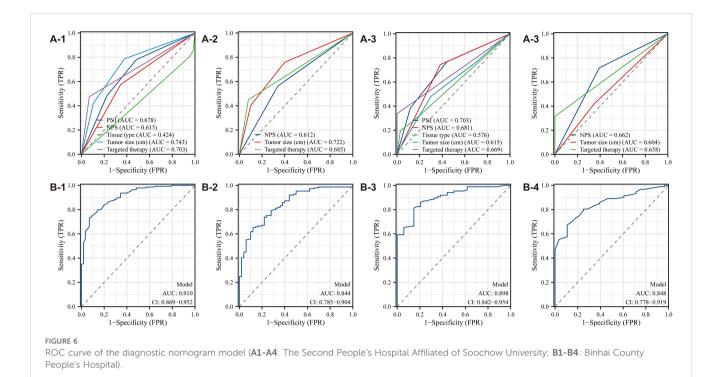


threat. As the deadliest of all gynecologic cancers, it is characterized by a notably high mortality rate (13). This poor prognosis is largely due to late-stage diagnoses, as most patients present with advanced disease at the time of detection (1). Key factors, including age, tumor size, histological subtype, and comorbidities, significantly impact survival outcomes (14). The advent of laparoscopy represents a notable advancement in the treatment of advanced ovarian cancer (15). Additionally, biological and molecular characteristics play a critical role in selecting the most precise and personalized therapeutic strategies for patients (16). By evaluating prognostic factors that influence postoperative outcomes, clinicians can adopt targeted treatments to address the underlying causes of poor prognosis. Early detection and timely intervention remain essential for improving patients' quality of life and extending



survival. This study emphasizes the growing relevance of nutritional and inflammatory markers as significant predictors of postoperative prognosis in ovarian cancer patients. A deeper understanding of how these markers correlate with survival outcomes offers valuable insights for patient management and could inform the development of innovative therapeutic strategies.

An imbalance between inflammatory and anti-inflammatory processes can lead to cell necrosis, apoptosis, coagulation abnormalities, and immune suppression (17). NLR serves as a wellestablished marker of systemic inflammatory response, reflecting both the activation and regulation of inflammation within the body (18). Neutrophils play a pivotal role in this process by secreting proinflammatory cytokines, regulatory cytokines, and chemokines, which initiate an inflammatory cascade during infection. In contrast, lymphocytes, integral to the immune system, counteract inflammation by releasing anti-inflammatory factors such as interleukin-10 (IL-10) (19). Monocytes contribute significantly to the innate immune response, continuously secreting pro-inflammatory cytokines, enzymes, and growth factors upon recruitment, thus amplifying the inflammatory response. Clinical data analysis indicates that elevated



NLR is associated with an increased risk of mortality. The RCS diagram further reveals that a higher LMR correlates with improved OS and CSS in postoperative ovarian cancer patients, whereas elevated NLR is linked to reduced OS and CSS. Although NLR and LMR reflect the systemic inflammatory response resulting from immune activation, they fail to capture the immune-nutritional status, a critical factor influencing ovarian cancer prognosis. Immune and nutritional disturbances are

Subgroup Agegeur	Valables HR95CI	<u>ili</u>	5 Lymph rode metan	VariaNes HR93 CT Autois	γT	L Subprop	Variables HR45CI	pα	Agayaar	Valiables HR15CI	100 P	Sabgroup Histology type Leve-grade	Variables HR95CI	F Sedges	ip Variables HR45Cl	
:59	L-NPS Reference		(48	L-NPS Reference	1071				- 29	<46.1 Reference		Tee-base	-961 Reference a61.4128-9628-2971089	4626	46.1 Reference 46.1-51.20.498/0.252.0983)	
60-69	10-NPS 1.003(1414,2430)	0.994	Ye	LAPS Reference		Type of surgery				461-31.2.046(0.362,3.020)	0.933 0.347	Medium-made	>11.2 0.327(0.168).0037)	0.001 Ya	+51.2 0.479(0.247,0.928)	
	L-NPS Reference HNPS3334(123616151)	0.819	R60 mage	BNPS 1248(8854).545)	4.127	105			68-69	<46.1 Reference			-36.1 Reference 46.1-51.28.731(8.190).1340)	9.286	-16.1 Reference 46.1-51.21.666/01473.910	
270	L-NPS Reference		Orade I	L-MPS Reference	1					46.1-31.20.134(8017,1047)	0.055 0.007	High-grade	1412 8525(8077)(816)	eite +	>512 1.126(02173.134)	_
Tumorsiae (am)	H-NPS 8:697(82242.166)	- 0.532	Grade II	HAPS 4483(1194343) L-MS Reference	4.034		L-NPS Reference		250	-46.1 Reference			46.1 Reference 46.1-51.2.8.541(8.283,0.03) 151.2.8.337(8.172,8.658)	0.063 No 0.001	-46.1 Reference	
4	L-MS Reference		Grade II	H-NPS 1272(0300,0400)	8744		ISNIS 1399(82293547)	0.716		461-5120748(02342391)	0.624 0.739	Lymph rode roctasta No	ota		46.1-51.20.426(0.192.0947) ++++++++++++++++++++++++++++++++++++	
Res 12	H-NPS 8/797(82742466)	0.998		L-NPS Reference H-NPS 1251(165242.598)	1.529	PTR			Tumor size (cm)	oki Reference			<86.1 Reference 46.1-512.0620(0.229).684)	0.324 Yes	said Beference	
	L-NPS Reference H-NPS 0793(0.2062.200)	0.656	Grade IV	L-MS Reference		PDS				461-3120378(01226188)	0.85	Ye	H12 8116(8000)8441) +	0.002	46.1-51.20979(0347,2.768) + + + + + + + + + + + + + + + + + + +	
>12	L-NPS Reference		Chenothenpy	H-NPS 4.251(0.581,8.897)	0.146		L-NPS Reference		8 10 12	(46.) Reference			-46.1 Reference 46.1-51.2.8.441(8.177).099) 	0.079 Diabete 0.024 Na		
Pathological type	II-NPS 4325(13263256)	0.006	200	L-NPS Reference H-NPS 3,460(1905,1273)	10%		BNPS 2213/12453983	4007		46.1-31.20.528(0.146,1348)	0.312 0.201	ff00stage Gende I	1012 8424(828)(828)	0.004	016.1 Reference 46.1-51.30.603(0.292.1.242)	
Serom adenocarcinom	L-NPS Reference		Yes	L-NPS Reference	1005		H-NPS 1213(1203/943)	4.047	>12	stal Beference	4.541	Cestern	46.1 Reference 46.1-51.2.8.350(8.067;2.155)	0.5% Yo	>512 0245(0111,0559) 🛶	
Indometricid adenocarcia	IS-NPS 1343(0.742,2.513) onto	0.318	Targetechberapy	H-NPS 1.894(107433329) 4	4.027	Residual tumorat un	any in the second se			461-5120883(8298,1567)	0.367	Oracle II	>51.2 #210(8.027,1.055)	0.258	46.1 Reference 46.1-51.20.283(0.068,1.313)	
	L-NPS Reference IE-NPS 1553(1792470)	0.014	Na	APS Peterson					Pathelogical type	H12 0303(0114,0385) +++	0.017		-46.1 Reference 46.1-51.28.731(8.190,3.340)	and Type of set	>512 0303(02142:301)	_
Macinossadorocarcino	L-MS Reference		Yo	II-NPS 1.567(E884(2.799)	6.125	NO			Servisiadenecarcinoma	-44.1 Reference		Orade III	112 8525(807/486)	→ 0.599 106	<46.1 Reference	
Clear cell carcinoma	H-NPS 1444(1216,1883)	0.912	Breatension	L-NPS Reference H-NPS 2149(1286,6136)	L158		L-NPS Reference			463-51.20(75(0.218,1002) +++ >51.2 0.385(0.151,0.779) +++	8.06 9.098		-46.1 Reference -66.1-51.2.8.569(8.224.).447) -51.2.8.6559(8.306.).484)	0.236 0.277 PDS	45.1-51.20.529(0.333,6459) >512_0.524(0.053,5151)	_
	L-NPS Reference H-NPS 2.542(81)84227)	0.906	Hypertension No	LAPS Reference			H-NES 1278(09463.313)	4,674	Informatificid advances in	+46.1 Reference		Grade IV	chi i Referenze		16.1 Reference	
Histology type Low-grade			Yes	H-NPS 2.121(1091,4.121)	4.027			board		461-5120200(01553.168)	0.643 0.367		461-5124177(80:63,153)	0.667 0.336 Residual tumor	46.1-51.20.496(0.298,0.960)	
	L-MPS Reference H-MPS 2,3488(2)903,1705	0.369		L-NPS Reference IS-NPS 1978(1790,3.218)	1.165	Yes			Macinom adenocarcinon	old.1 Reference		Chemotherapy No		Network Network	ald Reference	
Medium-grade	L-NPS Reference		Diabeles Na	LNIS Reference			L-NPS Reference			461-31.2(348(02342.391)	0.324 0.329		<46.1 Reference 46.1-512.0431(0.250),2340)	4.686	46.1-51.20.455(0.217,0.956)	
High-grade	H-NPS 1414(10882-638)	0.006		I-NPS Reference IS-NPS 1204(1930,1189)	L095		LOID MANNA		Charcell carcinona	sité 1 Beferrane		Yes	112 8325(8157).816)	0.599 Yes	SEL Reference	
	L-NPS Reference IN-NPS 1.6759159582.9280	407	10	L-NPS Reference H-NPS 2377(06368)450	6177		B-NPS 2.481(0.6933.879)	9.162		46.1-51.2046(0362,1000)	4.933 9.397		461-5128629(8328).280 -512 83(98).16186325	0.161 0.001	46.1-51.20.879(0.262.2.952) >51.2 0.902002932.7751	_
	ii.	23 53 13 160		65	13 34 13		6 5 5	0.0		1 1 1	1		0 0 0 0	2.8		1 1
Salagroup	Variables HR95CI	ली ज	Sebgrosp	Valabler 100350	28				Sabarran	Veiables BR45CI		Seberceo	Value IR90	pti Subges	ar Variables HR 65 CI	
Ageyear -59			Lymph node metant No	ADD Polymore		Salaprosp	Variables HR45 CI	Pft .	Age.year		—— K	Sabgroup Histology type Leve-grade	chi) Belance	Teptied In		
	L-NPS Reference H-NPS L1050L4472.728	0.829	Yes	I-NPS Reference H-NPS 2134(6853,5322)	• UB	There of surgery				<66.1 Reference 46.1-51.71.24.20.487.1.8171	1/02		-36.1 Reference 46.1-512.0.178(0.144).1374) 	0.432 0.117	46.1 Reference 46.1-51.20.812(0.002,1.248)	
63-89	L-NP5 Reference		та	L-NPS Reference HAPS 190703314149					63-69	>512 06110018620040	8,416	Notion-grade	Sil Reference	1012 Yes	>512 0.499(0242,1431)	
:20	BNPS260(08063.111)	0.081	RiO stage Grade I			ID5				<06.1 Reference 46.1-51.20.138.0028.1.2720	4.043		461-5128478(83142774)	0.582	<16.1 Reference 46.1.51.21.6660.1473.91F) >51.2 19990.2022.177)	
	L-NPS Reference H-NPS 1341(1297,6196)	0.703		L-NPS Reference H-NPS 4460(0.841,8.186)	4.078		L-NPS Reference		-30	>512 0219(0065,8715) +	0.012	High-grade	<46.1 Reference	Byperters 0.362 No	lon	-
Tamor siza (cm) <8			Grade II.	L-NPS Reference IENPS 1272(E3003.400)	4744					~#6.1 Reference 46.1.5120953928532340		lymph pode metasta	463-512867883443388 -512 835881718749	0.006	+86.1 Reference 46.1-51.20.521(0.228,1199)	
	L-NPS Reference H-NPS 1854(1012,2164)	0.899	Orade III	L-NPS Reference			16NPS 1399(0229,0547)	0.716	Turner size (cm)	>512 0.455(00583.594)	0.455	No	<46.1 Reference	Ye	>512 03550155,0310	
81012	L-NPS Reference		Grade IV	H-NPS 1209(06192983)	L445	105			-3	-16.1 Reference 46.1-51.21.299(0.162.3.207)	1635		461-5128/228/82642.002) >512 8:091(8:018/8451)	0.538 0.003	46.1 Reference	
>12	H-NPS 8 980(83182.550)	0.043		L-MPS Reference H-MPS 4.251(0.541,5.837)	1.146		LNPS Reference		81012	>512 0.853(0077,9438)	4.897	Yo	siti Befermer		>512 0.153(0431,0738)	
	L-NPS Reference H-NPS 4932(1483)8299)	0.009	Chemothempy Na	v L-NPS Reference			L-NPS Reference			486.1 Reference 46.1-51.20.507(0.142,130.0)	4.285	100mas	463-5128541(82113.386) >512 8484(82143.086)	0.201 Na 0.002 Na	46.1 Beference	
Pathological type Scrowadcrocarc inore			Ye	B-NPS 3.468(1926,6273)	4.058		H-NPS 2333(1245,4371)	0.008	>12	>512 0373(0106,1330)	9.127	Cende 1	(46.1 Reference		46.1-31.20.742(0.348,1.584) ++++++++++++++++++++++++++++++++++++	
	L-NPS Reference H-NPS 1.510(0.180(2.523)	0.221		L-NPS Reference IE-NPS 1963/1079.3640	6.072	Residual temerat un				-46.1 Reference 46.1-51.2034903360,20040	4.788		463-5128-573(8078,4285)	0.584 You 0.319	viti.1 Reference	
indometrial denocarch	L-NPS Reference		Targetedtherapy No			Advantation (191) (1)	~		Pathological type	45.131.03490390,2000 >512 0.000(0100,0920)	4.005	Cinde II	46.1 Reference		461-5120335(0808,1619)	
Macinous adenocarcino	II-NPS-4540(1376;84513) na	0.0.024	¥-	L-NPS Reference 31-NPS 1429(8492,1084)	011	No			Pathological type Serousadenocarcinoma	-46.) Reference		Grade III	461-5128731(81603340) >512 8525(8067,4816)	0.686 Type of ner 0.569 105	-46.1 Reference	
	L-NPS Reference H-NPS 1444(1842)2903	0.891	та	L-MPS Reference HMPS 2149(E2665136)	1455		L-NPS Reference			46.1 Reference 46.1-51.20.556(0:250,1297) +512_03720(1340815) +++	0.15	cence al	-46.1 Reference 46.1-51.287823832.089	4671	46.1 Reference 46.1-51.20.825(0.343,4.359) 3512 0.5240.08335151)	
Clear cell carcinoma	L-NPS Reference		Hypertension No						Indometri si da denocarci is		4.013	Grade IV	>51.2 8777(8310).755)	0.623 0.491 PDS	Sila Beference	
Hotology type	H-NPS 2734(1.342,4.512)	0.631		L-MPS Reference H-MPS 2029(103),405%	1.047		H-NPS 2022(10283/977)	0.041		46.1 Reference 46.1-31.21.919(0183.3.612)	4.587		46.1 Reference 46.1-51.2.8.137(0.006.0.155)	0.067	46.1-51.20.623(0.313.1.241)	
Lew-grade	L-NPS Reference		Ye	L-NPS Reference		Yes			Macinous adenocarci non		a 435	Chemotherapy	>512 8624(8314)2796	0.670 Residual tumor No	at surgery	
Nedium-grade	H-NPS 2388(0721,4521)	0.443	Diabetes	IS-NPS 2435(8834(8330)	1.099					46.1 Reference 46.1-51.21.199(0592,2707)	0.235	No	46.1 Reference		46.1 Reference 46.1-51.20.542(0.251,1171)	
	L-NPS Reference H-NPS 1.414(1.088,1.638)		14	L-NPS Reference H-NPS 1929(1990.1797)	4.057		L-NPS Reference		Clearcell catcinona	>512 0803000772400	8,717	Yes	>31.2 #007(8000).155)	0.563 0.211 Yo	>512 0.194(0079,0475) -	
	L-NPS Reference		Yo	LNPS Reference			IS-NPS 1997(8527,1287)	0,316		-96.1 Reference 46.1-51.20398(8662,1.667)	4.685		46.1 Reference 46.1-51.248897914053.6040	154	+86.1 Reference 46.1-51.21.092(0.065.3.914)	
High-grade	11-NPS 1.747(1.953,3,214)	23 50 75 108		HNES 1828(1499,6639)	25 50 55 160		8 1 1	11		-512 0823(0297,4118)	1.021		312 83498162A738 -	4 5	>512 0916(02573.259)	1 1
High-grade																
High-gudo																
JRE 7																
JRE 7	analysis	(A-F : OS	G-L	(SS)												

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integral to this prognosis. PNI, which assesses the nutritional and inflammatory state, is derived from serum albumin levels and lymphocyte counts. In contrast, NPS is a novel prognostic tool that combines NLR, LMR, albumin, and TC. Both PNI and NPS are simple to calculate and accurately represent the systemic nutritional, immune, and inflammatory status of patients (6, 9). PNI has been extensively used as a prognostic marker in various cancers, including colon, liver, and esophageal cancers. Furthermore, its clinical relevance has been demonstrated in noncancerous conditions such as COVID-19, cardiovascular diseases, Crohn's disease, and sepsis (20-22). However, the prognostic value of PNI in ovarian cancer remains inconsistent (23). For example, Yoshikawa et al. identified a PNI threshold of 46.5 for patients with ovarian clear cell carcinoma, showing that those in the high-PNI group had significantly better OS compared to the low-PNI group (24). This study identified significant associations between both PNI and NPS with OS and CSS in postoperative ovarian cancer patients. A PNI >51.2 was found to be a protective factor, correlating with improved survival outcomes. Patients with L-NPS demonstrated significantly better prognoses than those with H-NPS. RCS analysis further corroborated the relationship between higher PNI and extended OS and CSS. Subgroup analysis reinforced these findings, indicating that PNI is also linked to muscle loss during ovarian cancer treatment. Specifically, low PNI and reduced skeletal muscle mass independently predicted increased all-cause mortality (25). A separate investigation into preoperative PNI and OS in ovarian cancer patients reported significantly longer OS in the high-PNI group compared to the low-PNI group (26).

The role of nutritional inflammatory markers in postoperative prognosis for ovarian cancer is an important area of study, as demonstrated in this research. Prior studies established a scoring system-termed the peripheral blood score (PBS)-based on various peripheral blood parameters, including neutrophil count, lymphocyte count, monocyte count, albumin level, TC, and fibrinogen. This model revealed that lower PBS values were associated with better outcomes, while higher values were linked to poorer prognosis. Furthermore, PBS, along with FIGO grade and residual lesions, was identified as an independent predictor of OS and progression-free survival (PFS) in patients with epithelial ovarian cancer (EOC) (27). Our findings reinforce the notion that nutritional inflammatory markers are independent prognostic factors for OS and CSS in postoperative ovarian cancer patients. These results are consistent with previous studies (28), which highlight the significant influence of systemic inflammation and nutritional status on cancer prognosis (29). By analyzing a comprehensive cohort of clinical characteristics, laboratory parameters, and treatment modalities, this research addresses a notable gap in the current literature by establishing a direct link between nutritional inflammatory markers and survival outcomes in ovarian cancer patients. This study represents the first integration of nutritional inflammatory markers into predictive models for postoperative OS and CSS in ovarian cancer. Independent predictors for OS included age, PNI, NPS, tissue type, tumor size, targeted therapy, and diabetes, while those for CSS comprised age, NPS, tumor size, targeted therapy, and diabetes. Using these variables, nomograms were constructed that exhibited robust predictive accuracy for 3- and 5-year survival. However, calibration curves for 1-year survival revealed some discrepancies. The OS model demonstrated the highest predictive accuracy, with an AUC of 0.910, while the CSS model achieved an AUC of 0.844. External validation yielded AUC values of 0.898 for OS and 0.848 for CSS. This validation confirms the model's generalizability, reliability, and clinical applicability. Although the external validation results showed slightly reduced efficacy compared to the initial cohort, the model still displayed strong predictive performance. The observed differences may be due to the smaller sample size in the external validation cohort and the population heterogeneity between the two hospitals. In conclusion, the prognostic nomogram incorporating PNI and NPS offers considerable diagnostic value for postoperative ovarian cancer patients.

The findings carry important implications for clinical practice. Identifying nutritional inflammatory markers as reliable prognostic indicators provides healthcare providers with a robust tool for stratifying patients according to their postoperative risk. This approach could inform more personalized treatment strategies, such as enhanced postoperative monitoring or targeted nutritional interventions to optimize inflammatory status both pre- and postoperatively. Additionally, the results support incorporating nutritional inflammatory markers into routine clinical assessments, which may improve the predictive accuracy of survival outcomes, thereby enabling more informed decisionmaking and enhancing the overall management of ovarian cancer patients.

This study has several limitations that warrant consideration. The retrospective design may have introduced bias, and the relatively small sample size could limit the generalizability of the results. Additionally, the research methodology is somewhat restricted due to the small sample size and the limited statistical approaches employed, which diminish methodological diversity. Despite data collection from two hospitals, the lack of broader diversity remains a concern. Future research should prioritize validating these results in larger, multicenter cohorts and exploring the underlying mechanisms by which nutritional and inflammatory status influence cancer progression and treatment response.

5 Conclusion

In summary, the nutritional inflammatory index serves as a significant independent prognostic factor for OS and CSS in ovarian cancer patients. Its integration into clinical practice may improve patient stratification and enable more tailored therapeutic strategies. The development of a nomogram based on key predictors highlights its potential value in clinical decisionmaking. Nevertheless, additional research is required to validate these results and investigate the mechanisms connecting nutritional inflammation to tumor dynamics and patient outcomes. A more comprehensive understanding of these interactions could improve prognostic models and optimize management approaches for ovarian cancer patients.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by Ethics Committee of the Second People's Hospital Affiliated to Soochow University (Approval No. JD-LK-2022-079-01). The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

YZ: Conceptualization, Data curation, Investigation, Methodology, Writing – original draft, Writing – review & editing. WX: Formal analysis, Methodology, Project administration, Writing – original draft. XL: Funding acquisition, Methodology, Software, Supervision, Validation, Writing – original draft. ZY: Data curation, Formal analysis, Writing – original draft. YM: Data curation, Investigation, Methodology, Writing – original draft. YC: Conceptualization, Formal analysis, Methodology, Writing – original draft, Writing – review & editing. WZ: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The author(s) declare that no Generative AI was used in the creation of this manuscript.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fonc.2025.1531987/ full#supplementary-material

SUPPLEMENTARY FIGURE 1

RCS diagram of the relationship between albumin, total cholesterol, CA125, CA199, CEA and HE4 and survival prognosis in patients with ovarian cancer after surgery.

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