

Research Article

The Impact of the Prognostic Nutritional Index and Systemic Inflammatory Indices on Survival in Patients Receiving Neoadjuvant FLOT Chemotherapy for Gastric and Gastroesophageal Junction Adenocarcinoma: A Single-Center Retrospective Cohort Study

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Abstract

Objectives: Neoadjuvant/perioperative FLOT chemotherapy is standard of care in locally advanced gastric/GEJ adenocarcinoma, yet survival varies among similarly treated patients. We evaluated PNI and systemic inflammatory indices as predictors of overall survival (OS) and progression-free survival (PFS).

Methods: We retrospectively reviewed 196 patients with gastric/GEJ adenocarcinoma who received neoadjuvant FLOT (2016–2025) at Koç University Hospital. PNI and ten inflammatory markers were calculated from pretreatment blood tests and tested against OS/PFS using Cox regression, with Benjamini–Hochberg FDR correction for multiple comparisons.

Results: Median age was 62 years; median follow-up was 30.9 months. Fifty-two patients (26.5%) died; median OS was 86.4 months, median PFS was 53.6 months, and the pCR rate was 28.3%. None of the inflammatory indices was associated with OS or PFS after FDR correction. PNI<45 was associated with significantly shorter OS (HR 3.39; 95% CI 1.55–7.40; $p=0.002$; FDR $q=0.047$), which persisted on multivariable analysis (HR 3.75; 95% CI 1.53–9.19; $p=0.004$). The PFS association was directionally consistent but of borderline significance (HR 1.90; $p=0.070$).

Conclusion: None of the inflammatory indices was prognostic. Only PNI<45 was independently associated with shorter OS; given the low-PNI group's small size ($n=17$), this finding is hypothesis-generating and requires prospective validation.

Keywords: FLOT, gastric cancer, inflammatory markers, neoadjuvant therapy, prognostic nutritional index, survival

Cite This Article: Demir N, Kıkılı Cİ, Köylü B, Kemik F, Genç TB, Tunalı D, et al. The Impact of the Prognostic Nutritional Index and Systemic Inflammatory Indices on Survival in Patients Receiving Neoadjuvant FLOT Chemotherapy for Gastric and Gastroesophageal Junction Adenocarcinoma: A Single-Center Retrospective Cohort Study. EJMI 2026;10(4):610–617.

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Submitted Date: September 2, 2026 **Accepted Date:** September 15, 2026

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Gastric cancer is the fifth most commonly diagnosed cancer and the fifth leading cause of cancer-related death worldwide.^[1] In locally advanced, resectable gastric and gastroesophageal junction (GEJ) adenocarcinoma, a perioperative chemotherapy approach has become the standard of care, supported by trials demonstrating that systemic therapy added to surgery improves survival.^[2,3] In the FLOT4 trial, the fluorouracil, leucovorin, oxaliplatin, and docetaxel (FLOT) regimen significantly increased both the pathological complete response (pCR) rate and overall survival compared with ECF/ECX, establishing FLOT as the current standard perioperative regimen.^[3,4] More recently, the addition of immunotherapy to perioperative treatment has been reported to yield further gains in pCR rates.^[5]

Nevertheless, substantial heterogeneity in pathological response and survival is observed among patients with similar stages who receive the same regimen. This underscores the continued need for prognostic biomarkers that are inexpensive and routinely obtainable before treatment. Tumor-associated systemic inflammation is recognized as one of the hallmarks of cancer.^[6,7] Indices based on peripheral blood cell ratios, the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII), have been associated with poor prognosis across various solid tumors.^[8] On the nutritional axis, the prognostic nutritional index (PNI) described by Onodera et al.^[9] is a simple measure combining serum albumin and total lymphocyte count; in series of patients undergoing gastric cancer surgery, low PNI has been shown at the meta-analytic level to be associated with shorter survival.^[10] In addition, composite indices such as the C-reactive protein (CRP)/albumin ratio (CAR), hemoglobin, albumin, lymphocyte, and platelet (HALP), and C-reactive protein-albumin-lymphocyte index (CALLY) have similarly been proposed to carry prognostic value.^[11,12]

However, most of these indices have been evaluated in different stages and treatment contexts, largely in series testing a single index. Data are limited on the simultaneous testing of inflammatory and nutritional indices, with correction for multiple comparisons, in a homogeneous cohort receiving a contemporary, intensive regimen such as neoadjuvant FLOT. In this study, we aimed to evaluate the association of PNI and commonly used systemic inflammatory indices with overall survival (OS) and progression-free survival (PFS) in patients with gastric/GEJ adenocarcinoma receiving neoadjuvant FLOT, using pre-specified cut-offs and false discovery rate (FDR) control.

Materials and Methods

Study Design and Patient Selection

This single-center retrospective cohort study included 196 consecutive patients with histologically confirmed gastric

or GEJ adenocarcinoma who received neoadjuvant FLOT chemotherapy at Koç University Hospital between October 2016 and July 2025. The FLOT regimen was administered either as a total neoadjuvant strategy (8 cycles before surgery) or a perioperative strategy (4 cycles before and 4 cycles after surgery). Demographic, clinicopathological, laboratory, and survival data were obtained from electronic medical records. The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Koç University Ethics Committee (Decision No: 2026.395.IRB2.225, dated 10 August 2026).

Calculation of Indices

All indices were calculated from routine complete blood count and biochemistry values obtained before the start of neoadjuvant treatment: $PNI = 10 \times \text{albumin (g/dL)} + 0.005 \times \text{total lymphocyte count (/mm}^3\text{)}$ ^[9]; $NLR = \text{neutrophils/lymphocytes}$; $PLR = \text{platelets/lymphocytes}$; $SII = \text{platelets} \times \text{neutrophils/lymphocytes}$; $CAR = \text{CRP (mg/L)/albumin (g/dL)}$; $HALP = \text{hemoglobin} \times \text{albumin} \times \text{lymphocytes/platelets}$ ^[11]; $CALLY = \text{albumin} \times \text{lymphocytes}/(\text{CRP} \times 10^4)$.^[12] The previously described cut-off of 45 was used for PNI.^[9,10] CRP, lactate dehydrogenase (LDH), carcinoembryonic antigen (CEA), and carbohydrate antigen (CA) 19-9 levels were also evaluated individually.

Endpoints

Overall survival was defined as the time from diagnosis to death or last known follow-up; progression-free survival was defined as the time from diagnosis to progression, recurrence, or death from any cause. Patients without an event were censored at their last follow-up visit. Pathological complete response was defined as the absence of residual invasive tumor and lymph node metastasis in the resection specimen (ypT0N0).^[13] Because pCR could only be assessed in patients who underwent surgical resection with a recorded pathological evaluation, 23 patients, death before surgery (n=9), inability to undergo surgery due to progression or unresectable disease (n=2), and absence of recorded surgical/pathological data (n=12), were excluded from this analysis, which was therefore performed in 173 patients. The patient flow diagram and analysis subsets are summarized in Figure 1.

Statistical Analysis

Descriptive statistics were presented as median (IQR) or minimum–maximum and as number (percentage). Survival curves were plotted using the Kaplan–Meier method, and groups were compared with the log-rank test. Associations between indices and OS/PFS were tested with univariable Cox proportional hazards regression: variables with a right-skewed distribution (systemic immune-inflammation index-SII, CALLY, LDH, CEA, CA 19-9) were natural

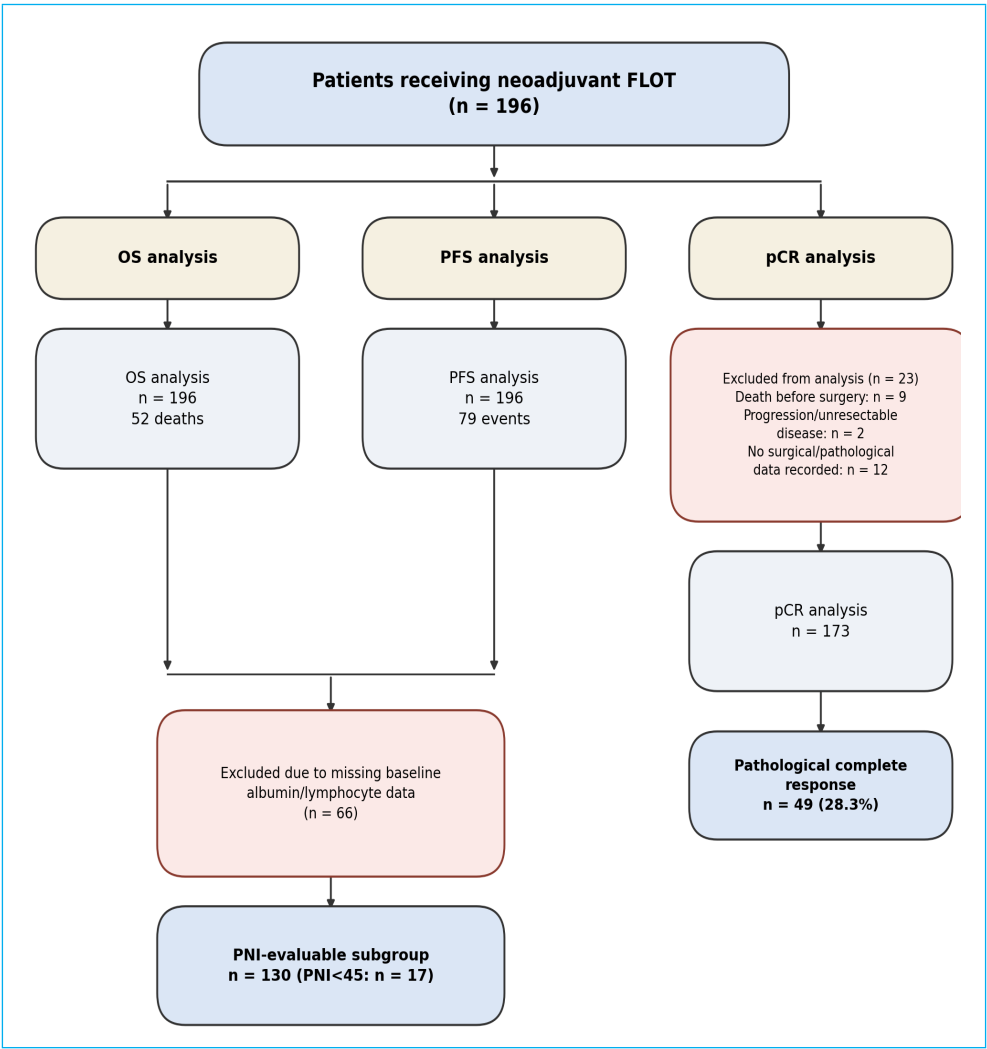


Figure 1. Patient flow diagram and analysis subsets.

log-transformed before modeling. PNI was additionally analyzed categorically using the 45 cut-off. The Benjamini–Hochberg FDR correction was applied across the 22 tests spanning eleven markers and two endpoints, with $q < 0.05$ considered significant.^[14] For PNI, a multivariable Cox model adjusted for age, pathological complete response, and treatment schedule (total neoadjuvant/perioperative) was constructed. Median follow-up was calculated using the reverse Kaplan–Meier method. Analyses were performed in Python (v3.12) using the lifelines and scipy libraries; a two-sided $p < 0.05$ was considered statistically significant.

Results

Patient Characteristics

The 196 patients enrolled had a median age of 62 years (range 23–83). Among the 186 patients with a known treatment schedule, 133 (71.5%) were treated with the total neoadjuvant strategy and 53 (28.5%) with the perioper-

ative strategy; the median number of neoadjuvant FLOT cycles was 8. Among the 130 patients with available histological subtype data, 80 (61.5%) had signet-ring cell histology, 40 (30.8%) intestinal type, and 10 (7.7%) diffuse type. Among the 173 patients who underwent surgery with an available pathological evaluation, 49 (28.3%) achieved a pathological complete response (Fig. 1). The demographic and clinicopathological characteristics of the patients are summarized in Table 1, and pretreatment laboratory and index values are in Table 2.

Survival Outcomes

Median follow-up was 30.9 months, during which 52 patients (26.5%) died. Median OS for the whole cohort was 86.4 months; the 24-month OS rate was 77.8% and the 36-month OS rate was 72.2%. In the PFS analysis set ($n=196$), 79 events were observed and median PFS was 53.6 months.

Table 1. Demographic and clinicopathological characteristics of patients (n=196)

Characteristic	Value
Age, median (range), years	62 (23–83)
Period of diagnosis	2016–2025
Treatment schedule, n (%) ^a	
Total neoadjuvant (8 cycles → surgery)	133 (71.5)
Perioperative (4 cycles → surgery → 4 cycles)	53 (28.5)
Number of neoadjuvant FLOT cycles, median	8
Histological subtype, n (%) ^b	
Signet-ring cell	80 (61.5)
Intestinal	40 (30.8)
Diffuse	10 (7.7)
Pathological complete response (ypT0N0), n/N (%)	49/173 (28.3)
Follow-up, median, months	30.9
Death, n (%)	52 (26.5)

^aTreatment schedule was available for 186 patients. ^bHistological subtype was available for 130 patients.

Table 2. Pretreatment laboratory values and indices

Marker	n	Median (IQR)
Albumin, g/dL	130	4.22 (3.97–4.51)
Lymphocytes, /mm ³	154	1850 (1400–2400)
CRP, mg/L	129	4.8 (1.7–13.5)
LDH, U/L	96	162.5 (149.8–208.3)
CEA, ng/mL	131	2.0 (1.3–4.0)
CA 19-9, U/mL	130	15.3 (7.0–32.6)
PNI	130	52.5 (47.5–55.5)
NLR	154	2.33 (1.56–3.47)
PLR	154	141.3 (100.5–196.6)
SII, ×10 ³	154	600 (378–1004)
CAR	117	1.19 (0.45–3.74)
HALP	130	0.33 (0.23–0.56)
CALLY	117	0.17 (0.05–0.42)

IQR: Interquartile range; CRP: C-reactive protein; LDH: Lactate dehydrogenase; CEA: Carcinoembryonic antigen; CA: Carbohydrate antigen; PNI: Prognostic nutritional index; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; SII: Systemic immune-inflammation index; CAR: C-reactive protein-to-albumin ratio; HALP: Hemoglobin, albumin, lymphocyte, and platelet; CALLY: C-reactive protein-albumin-lymphocyte index.

PNI and Survival

PNI could be calculated in 130 patients with available pretreatment albumin and lymphocyte values (median 52.5; IQR 47.5–55.5); 17 patients (13.1%) had PNI<45. Patients with PNI<45 had significantly shorter OS: median OS was

18.0 months in the low-PNI group versus not reached in the high-PNI group; 24-month OS rates were 48% and 85%, respectively (log-rank $p=0.001$; Fig. 2). On univariable Cox analysis, PNI<45 was associated with an HR of 3.39 (95% CI 1.55–7.40; $p=0.002$), and this association retained its significance after FDR correction across 22 tests ($q=0.047$). In the multivariable model adjusted for age, pathological complete response, and treatment schedule ($n=122$; 31 events), PNI<45 remained independently associated with shorter OS (HR 3.75; 95% CI 1.53–9.19; $p=0.004$; Table 3). For PFS, the association was directionally consistent but of borderline significance: median PFS was 13.1 months in the low-PNI group versus 53.9 months in the high-PNI group (24-month PFS 42% vs. 71%; log-rank $p=0.065$; HR 1.90; 95% CI 0.95–3.79; $p=0.070$; Fig. 3).

Inflammatory Indices and Survival

None of neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), SII, CAR, HALP, CALLY, CRP, LDH, CEA, or CA 19-9 showed a significant association with OS or PFS after FDR correction (all $q\geq 0.32$; Table 4). Uncorrected p -values for CAR (OS) and CA 19-9 (PFS) were 0.046 and 0.043, respectively, but effect sizes were negligible (HR per unit 1.06 and 1.19) and significance was lost after correction for multiple comparisons ($q=0.322$ for both).

Discussion

In this cohort of patients with gastric/GEJ adenocarcinoma receiving neoadjuvant FLOT, eleven markers derived from routine pretreatment blood tests were tested simultaneously against two survival endpoints, with correction for multiple comparisons. Our main finding is twofold: first, none of the classical systemic inflammatory indices widely proposed in the literature as prognostic (NLR, PLR, SII, CAR, HALP, CALLY) was associated with OS or PFS in this cohort; second, PNI<45, representing the nutritional axis, remained an independent predictor of shorter overall survival in both univariable analysis and models adjusted for age, pathological response, and treatment schedule.

The PNI finding is consistent with meta-analytic data from series of patients undergoing gastric cancer surgery; in the meta-analysis by Yang et al.^[10], low PNI was associated with shorter OS. Our study suggests that this association may also hold in the context of contemporary, intensive neoadjuvant FLOT therapy. The decline in 24-month survival to 48% in the low-PNI group indicates that the effect is also of clinically meaningful magnitude. Given that albumin, one of the two components of PNI, serves both as a negative acute-phase reactant of chronic inflammation and as a marker of nutritional reserve, the observed signal is likely to derive substantially from the albumin axis; indeed, this

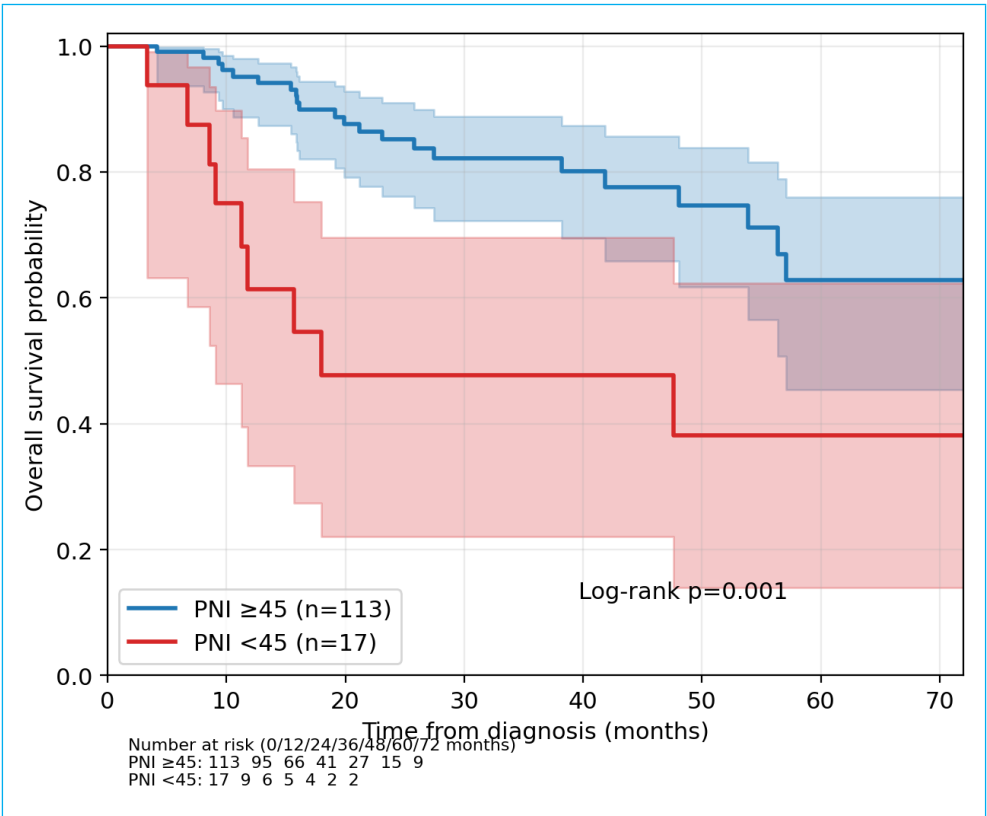


Figure 2. Kaplan–Meier curves for overall survival according to PNI category.

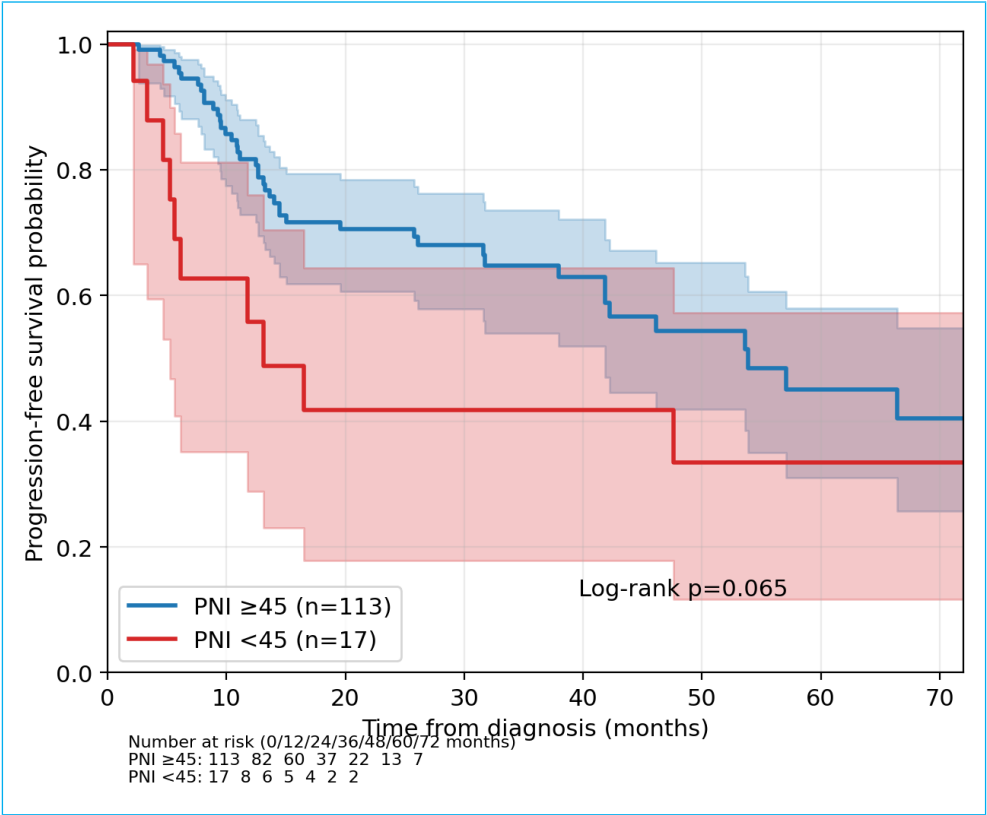


Figure 3. Kaplan–Meier curves for progression-free survival according to PNI category.

Table 3. Multivariable Cox regression model for overall survival (n=122; 31 events)

Variable	HR (95% CI)	p
PNI <45 (vs. ≥45)	3.75 (1.53–9.19)	0.004
Age (years)	0.98 (0.95–1.01)	0.242
Pathological complete response (yes vs. no)	0.43 (0.15–1.25)	0.123
Treatment schedule (perioperative vs. total neoadjuvant)	0.57 (0.23–1.43)	0.232

HR: Hazard ratio; CI: Confidence interval; PNI: Prognostic nutritional index.

interpretation is supported by the fact that the purely inflammatory ratios that do not include albumin (NLR, PLR, SII) were uniformly null. From a practical standpoint, this finding aligns with current guideline recommendations emphasizing the importance of nutritional screening be-

fore neoadjuvant treatment and early nutritional intervention where indicated^[15]; however, whether nutritional intervention would alter survival in this patient group can only be answered by prospective studies.

The uniformly negative findings for the inflammatory indices may at first appear to conflict with the broader literature but can be explained by several mechanisms. First, much of the evidence supporting the prognostic value of these indices derives from metastatic or stage-heterogeneous cohorts; in a curative-intent neoadjuvant population with relatively good performance status, the baseline inflammatory burden may be confined to a narrow range, reducing discriminative power. Second, in small series testing a single index, the higher likelihood of publication of positive findings (publication bias) and data-driven selection of cut-offs may have inflated effect estimates in the literature.^[8] In our study, the use of pre-specified cut-offs and

Table 4. Association of markers with overall survival and progression-free survival (univariable Cox regression)

Marker	Endpoint	n	Events	HR (95% CI)	p	FDR q
PNI (<45 vs. ≥45)	OS	130	32	3.39 (1.55–7.40)	0.002	0.047
PNI (<45 vs. ≥45)	PFS	130	53	1.90 (0.95–3.79)	0.070	0.322
NLR	OS	154	37	1.06 (0.97–1.15)	0.219	0.603
NLR	PFS	154	60	1.05 (0.98–1.13)	0.180	0.566
PLR	OS	154	37	1.00 (1.00–1.00)	0.853	0.912
PLR	PFS	154	60	1.00 (1.00–1.00)	0.711	0.912
SII (log)	OS	154	37	1.07 (0.71–1.61)	0.753	0.912
SII (log)	PFS	154	60	1.09 (0.79–1.50)	0.612	0.903
CAR	OS	117	28	1.06 (1.00–1.11)	0.046	0.322
CAR	PFS	117	47	1.04 (1.00–1.08)	0.073	0.322
HALP	OS	130	32	0.50 (0.11–2.19)	0.354	0.779
HALP	PFS	130	53	0.69 (0.23–2.07)	0.511	0.864
CALLY (log)	OS	117	28	1.10 (0.25–4.77)	0.902	0.912
CALLY (log)	PFS	117	47	1.45 (0.51–4.13)	0.491	0.864
CRP	OS	129	32	1.01 (0.99–1.03)	0.412	0.823
CRP	PFS	129	52	1.00 (0.99–1.02)	0.678	0.912
LDH (log)	OS	96	23	1.15 (0.28–4.68)	0.849	0.912
LDH (log)	PFS	96	38	0.32 (0.07–1.42)	0.133	0.489
CEA (log)	OS	131	34	1.02 (0.66–1.58)	0.912	0.912
CEA (log)	PFS	131	54	1.09 (0.79–1.50)	0.616	0.903
CA 19-9 (log)	OS	130	32	1.13 (0.91–1.41)	0.269	0.657
CA 19-9 (log)	PFS	130	52	1.19 (1.01–1.41)	0.043	0.322

OS: Overall survival; PFS: Progression-free survival; HR: Hazard ratio; CI: Confidence interval; FDR: False discovery rate (Benjamini–Hochberg); PNI: Prognostic nutritional index; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; SII: Systemic immune-inflammation index; CAR: C-reactive protein-to-albumin ratio; HALP: Hemoglobin, albumin, lymphocyte, and platelet; CALLY: C-reactive protein-albumin-lymphocyte index; CRP: C-reactive protein; LDH: Lactate dehydrogenase; CEA: Carcinoembryonic antigen; CA: Carbohydrate antigen; PNI was modeled categorically (<45 vs. ≥45); all other markers were modeled as continuous variables. Variables marked (log) were natural log-transformed.

FDR control was intended to minimize such biases. Third, given the moderate number of events in our cohort, small true effects cannot be excluded; however, our results suggest that at least large, clinically decision-changing effects are not present in this context.

The pCR rate of 28.3% in our cohort is higher than both the 16% reported for the FLOT arm in the FLOT4-AIO phase 2 data and the 19% reported in the durvalumab+FLOT arm of the MATTERHORN trial.^[4,5] This difference may partly reflect the predominance of the total neoadjuvant strategy (71.5%) in our cohort and the fact that pathological evaluation was performed locally.

Limitations of our study include its retrospective, single-center design; the reduced number of patients in whom indices could be calculated due to missing laboratory data (130/196 for PNI); the small size of the low-PNI group ($n=17$; 9 deaths) and the resulting wide confidence intervals; incomplete histological subtype and clinical stage data; and the lack of central pathological review. In addition, the borderline significance of the PFS signal warrants re-evaluation as the number of events increases. For these reasons, the PNI finding should be interpreted as hypothesis-generating rather than confirmatory. The strengths of the study, on the other hand, include a relatively homogeneous and contemporary cohort treated with a single regimen (FLOT), a mature median follow-up of 30.9 months, pre-specified cut-offs, and transparent reporting of the multiple-comparison correction.

Conclusion

In patients with gastric/GEJ adenocarcinoma receiving neoadjuvant FLOT, none of the classical systemic inflammatory indices was prognostic, whereas pretreatment PNI <45 was identified as an independent predictor of shorter overall survival. PNI, which is cost-free and can be calculated from routinely available data, may contribute to pretreatment risk stratification and to the design of nutritional intervention studies in this patient population; this finding requires validation in larger, prospective cohorts.

Disclosures

Ethics Committee Approval: The study was approved by the Koç University Ethics Committee (no: 2026.395.IRB2.225, date: 10.08.2026).

Informed Consent: Not applicable.

Conflicts of Interest: The authors declared no conflict of interest.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: The authors declared that during the preparation of this manuscript, the authors used Claude (Anthropic, PBC) to improve the language and readability of the text

and to assist with English-language translation. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Authorship Contributions: Concept — FS, ND; Design – ND, FS; Supervision – FS, ŞG, DeT; Data Collection or Processing –ND, ÇİK, BK, FK, TBG, DeT, ŞL, ŞG, DiT, FS; Analysis or Interpretation – ND, FS; Literature Search – ND, ÇİK, BK, FK, TBG, DeT, ŞL, ŞG, DiT, FS; Writing – ND; Critical Review – ND, ÇİK, BK, FK, TBG, DeT, ŞL, ŞG, DiT, FS.

Peer-review: Externally peer-reviewed.

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