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The impact of monocyte to HDL ratio and Prognostic Nutritional Index on survival in stage III colorectal cancer patients

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ABSTRACT

Aims: This study explores how systemic inflammatory and nutritional indicators, specifically the monocyte-to-high-density lipoprotein ratio (MHR) and Prognostic Nutritional Index (PNI), influence clinical outcomes in stage III colorectal cancer (CRC) patients.

Methods: A retrospective review of 109 individuals was conducted. ROC curve analysis was employed to determine the optimal cut-off values of MHR and PNI for predicting mortality. Survival outcomes, including overall survival (OS) and disease-free survival (DFS), were evaluated using Kaplan-Meier estimates and compared with log-rank tests. Cox regression was utilized to pinpoint factors independently associated with DFS.

Results: The findings revealed significantly lower OS among patients not undergoing adjuvant chemotherapy (45.6 vs. 82.2 months; p=0.002). Additionally, diminished MHR (<0.37) and PNI (<46.8) levels were linked to poorer OS (p=0.041 and p=0.003, respectively). While low PNI was also associated with reduced DFS (p=0.021), MHR did not significantly impact DFS (p=0.42). Both MHR (AUC: 0.643) and PNI (AUC: 0.657) demonstrated moderate predictive capabilities for mortality. Importantly, perineural invasion surfaced as an independent negative prognostic factor for DFS (HR: 2.36; p=0.038).

Conclusion: In conclusion, pre-treatment MHR and PNI values serve as accessible and low-cost indicators that may assist in prognostic stratification in stage III CRC management.

Keywords: Monocyte-to-HDL ratio, Prognostic Nutritional Index, colorectal cancer, stage III, survival, prognostic markers

INTRODUCTION

Colorectal cancer (CRC) ranks among the most prevalent malignancies globally, with high incidence and mortality rates across both developed and developing nations. According to GLOBOCAN 2024 estimates, it is the third most commonly diagnosed cancer and the second leading cause of cancer-related death worldwide.[1] In Turkiye, CRC represents 12.7% of all malignancies, with age-standardized incidence rates of 23.6/100,000 in males and 16.2/100,000 in females, reflecting unique regional patterns that warrant population-specific investigations.[2-4]

Stage III CRC defined by regional lymph node involvement without distant metastasis per AJCC 8th edition criteria[5] occupies a critical therapeutic window. While surgical resection offers potential curability, occult micrometastases drive recurrence rates exceeding 30% despite adjuvant chemotherapy.[6] Current 5-year survival rates plateau around 60-65%, underscoring limitations of conventional TNM staging and highlighting the urgent need for refined prognostic tools.[7] Heterogeneity in treatment response remains poorly explained by histopathology alone, as tumors with identical staging may exhibit divergent biological behaviors influenced by host inflammatory responses and nutritional status.[8]

Systemic inflammation constitutes the seventh hallmark of cancer, fostering a tumor-permissive microenvironment through multiple pathways: angiogenesis induction, DNA damage acceleration, and immune evasion.[8] Circulating immune cells serve as quantifiable sentinels of this process, with neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) extensively validated as prognostic indicators.[9] More recently, the monocyte-to-HDL cholesterol ratio (MHR) has emerged as a superior biomarker by integrating pro-tumorigenic and cardioprotective mechanisms. Monocytes promote metastasis via matrix metalloproteinase secretion and immunosuppressive cytokine production (e.g., IL-10, TGF-β),[10] while HDL cholesterol exerts anti inflammatory effects through endothelial protection and oxidized lipid clearance.[11] Elevated MHR thus signifies disrupted homeostasis favoring tumor progression, with meta-analyses confirming its prognostic value across gastrointestinal malignancies.[12]

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Nutritional compromise frequently parallels cancer associated inflammation, creating a vicious cycle that accelerates cachexia and immunosuppression. The Prognostic Nutritional Index (PNI) calculated from serum albumin and lymphocyte counts quantifies this dual insult. Albumin synthesis suppression reflects hepatic inflammatory signaling (IL-6 mediated), while lymphopenia indicates adaptive immune impairment.[13,14] Multiple studies demonstrate PNI's predictive power, with thresholds <45 reducing 5-year survival by 30-40% in gastric and CRCs.[15,16] Notably, PNI's prognostic independence from body mass index makes it particularly valuable in obese populations where traditional nutritional assessments fail.[14]

Given this biological rationale, both MHR and PNI offer promising insight into the tumor microenvironment and host-tumor interactions. Despite their growing use in clinical research, few studies have examined their combined prognostic value specifically in patients with stage III colorectal adenocarcinoma, where treatment decisions hinge on accurate survival predictions. Therefore, identifying whether these simple biomarkers are capable of predicting survival and recurrence may have significant implications for clinical practice.

This study aims to investigate the prognostic significance of preoperative MHR and PNI levels in patients with stage III colorectal adenocarcinoma. By examining their association with overall survival (OS) and disease-free survival (DFS), we seek to determine whether these cost-effective indices can supplement conventional prognostic tools and contribute to more personalized treatment approaches.

METHODS

This study was approved by the Van Training and Research Hospital Non-interventional Clinical Researches Ethics Committee (Date: 04.07.2025, Decision No: GOKAEK/2025-05-11) and conducted in accordance with the Declaration of Helsinki. Informed consent was waived due to the retrospective nature.

This retrospective cohort study included 109 patients who were diagnosed with stage III colorectal adenocarcinoma and underwent surgical resection between 2015 and 2022 at a single tertiary care center. Inclusion criteria required histopathologically confirmed stage III disease. Given the sample size (n=109), multivariate analyses were limited to ≤5 covariates to avoid overfitting. Patients with chronic inflammatory diseases, active infections, hematologic disorders, second malignancies, or missing data were excluded.

Comprehensive clinical, pathological, and demographic data were retrieved from electronic medical records. Collected parameters included patient age, sex, tumor location, surgical urgency (elective vs. emergency), histological grade, T stage, lymphovascular and perineural invasion status, number of harvested lymph nodes, number of metastatic lymph nodes, and details of adjuvant chemotherapy regimen administered (Table 1).

Table 1. Patients demographic characteristics

	n	%
Gender		
Female	45	41.3
Male	64	58.7
T stage		
T2	12	11
T3	65	59.6
T4	32	29.4
Histologic grade		
Grade 1	37	33.9
Grade 2	60	55.1
Grade 3	12	11
Lymphovascular invasion	60	55.0
Perineural invasion	50	45.9
Tumor localisation		
Rectum	27	24.8
Right colon	40	36.7
Left colon	42	38.5
Emergency operation status	22	20.2
Adjuvant chemotherapy	95	87.2
Adjuvant chemotherapy (n=95)		
Capox	40	39.6
Folfox	41	40.6
Capecitabine	14	13.8
Relapse	43	39.4
Mortality	30	27.5
PNI		
Low	31	28.4
High	78	71.6
MHR		
Low	46	42.2
High	63	57.8
	Median±SD	**Median (min-max)**
Age	62.2±13.4	63 (40-84)
Harvested lymph nodes	19.6±8.3	18 (4-46)
Positive lymph nodes	5.61±3.9	5 (1-20)
PNI	48.9±3.9	49.4 (40-56.9)
MHR	0.44±0.17	0.4 (0.01-0.89)
Positive lymph nodes rates	0.29±0.17	0.26 (0.04-0.83)

PNI: Prognostic Nutritional Index, MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein, SD: Standard deviation, Min: Minimum, Max: Maximum

Blood samples were collected after a 12-hour fasting period. In patients undergoing elective surgery, samples were obtained within 7 days prior to the operation. For those undergoing emergency procedures, preoperative laboratory tests were drawn within 24 hours of hospital admission, as part of the routine workup. Serum HDL cholesterol and absolute monocyte counts were measured using automated spectrophotometry within 2 hours of collection.

Albumin levels were quantified via bromocresol green method. The PNI was calculated using the formula: 10×serum albumin (g/dl)+0.005×total lymphocyte count (per mm^3).[14] The MHR was derived by dividing absolute monocyte count by HDL cholesterol level.[12] All laboratory values used in the calculations were obtained from preoperative blood samples.

Statistical Analysis

Data analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as medians±standard deviations or medians with ranges, depending on distribution. The Kolmogorov-Smirnov test was used to assess normality. Group comparisons were conducted using Chi-square or Fisher's exact tests for categorical variables.

Receiver operating characteristic (ROC) curve analysis was utilized to identify optimal cut-off values for MHR and PNI based on their ability to predict OS. Kaplan-Meier survival curves were generated to assess OS and disease-free survival (DFS), with statistical significance evaluated using the log-rank test. DFS was defined as the time from surgery to the first documented tumor recurrence or radiologic evidence of relapse. Variables that reached significance in univariate analysis were further examined in a multivariate Cox proportional hazards model to determine independent predictors of DFS. A two-tailed p-value <0.05 was considered statistically significant throughout the analysis. Multivariate Cox regression model included the following variables that were significant in univariate analysis: histologic grade, perineural invasion, emergency operation status, age group, and PNI level. Due to the limited number of death events (n=30), OS was not analyzed in multivariate fashion to avoid statistical overfitting.

RESULTS

A total of 109 patients with stage III colorectal adenocarcinoma were included in the study. The median age of the cohort was 62.2±13.4 years (medain: 63), and the gender distribution comprised 64 males (58.7%) and 45 females (41.3%).

In terms of tumor T staging, 11% of patients were classified as T2, 59.6% as T3, and 29.4% as T4. Regarding histologic differentiation, 33.9% of tumors were grade 1, 55.1% were grade 2, and 11% were grade 3. Lymphovascular invasion (LVI) was identified in 60 patients (55%), while perineural invasion was present in 50 patients (45.9%).

Tumor localization was as follows: rectum in 24.8% of cases, right colon in 36.7%, and left colon in 38.5%. Surgeries were performed electively in 87 patients (79.8%), whereas 22 (20.2%) underwent emergency procedures. Adjuvant chemotherapy was administered to 95 patients (87.2%), with the following regimens recorded: CAPOX (40 patients), FOLFOX (41 patients), and oral capecitabine (14 patients) (Table 1).

During the follow-up period, 43 patients (39.4%) experienced disease recurrence, and 30 (27.5%) died. The median number of harvested lymph nodes was 19.6±8.3, with a median of 18. The average number of positive nodes was 5.61±3.9, yielding a median lymph node ratio of 0.26.

Laboratory-based indicators revealed an average MHR of 0.44±0.17 (median: 0.40), with 42.2% of patients having values <0.37. The mean PNI was calculated at 48.9±3.9 (median: 49.4), and 28.4% of the cohort had PNI values below 46.8 (Table 2). ROC analysis, performed using OS as the endpoint, identified the cut-off points for MHR and PNI as <0.37 and <46.8, respectively.

Table 2. Examination of ROC curve test for the ability of MHR and PNI values to predict mortality

	MHR	PNI
Cut-Off	<0.37	<46.8
AUC (95% CI)	0.643 (0.545-0.732)	0.657 (0.560-0.745)
Sensitivity (95% CI)	60 (40.6-77.3)	53.33 (34.3-71.7)
Specificity (95% CI)	63.29 (51.7-73.9)	75.95 (65-84.9)
PPV (95% CI)	38.3 (29.1-48.4)	45.7 (33.5-58.5)
NPV (95% CI)	80.6 (72.3-86.9)	81.1 (74.1-86.5)
p	0.017*	0.009**

*p<0.05, ROC curve test, ROC: Receiver operating characteristic, MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein, PNI: Prognostic Nutritional Index, AUC: Area under curve, CI: Confidence interval, PPV: Positive predictive value, NPV: Negative predictive value

The AUC values were 0.643 (95% CI: 0.545-0.732; p=0.017) for MHR and 0.657 (95% CI: 0.560-0.745; p=0.009) for PNI, suggesting moderate prognostic utility (Figure 1).

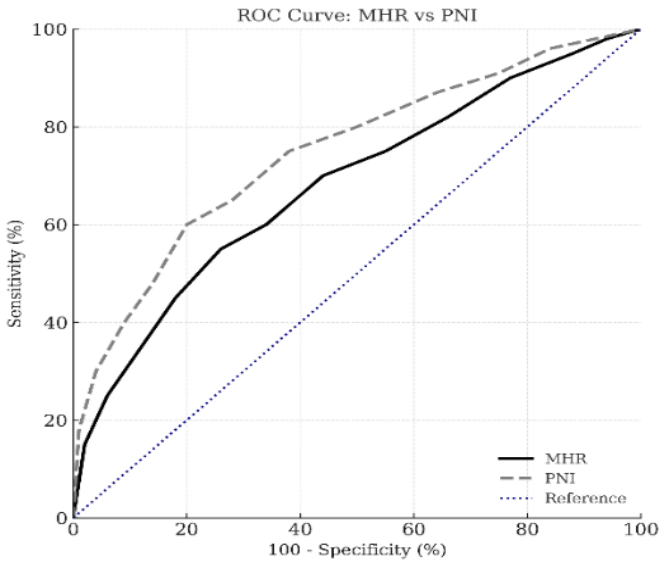

Figure 1. Examination of the mortaity prediction ability of MHR and PNI values using the ROC curve test

*The diagnostic performance of MHR and PNI values in predicting mortality among the included patients was evaluated using the ROC curve test. According to the analysis results, the cut-off values for MHR and PNI were determined to be <0.37 and <46.8, respectively. MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein, PNI: Prognostic Nutritional Index, ROC: Receiver operating characteristic

Survival analysis demonstrated significantly reduced OS among patients who did not receive adjuvant chemotherapy (45.6 months vs. 82.2 months; p=0.002) ant they had higher Charlson Comorbidity Index scores (median: 6.2 vs. 3.1; p=0.01) (Figure 2A). Patients with PNI <46.8 had markedly lower OS (65.3 months vs. 85.1 months; p=0.003) (Figure 2B). Likewise, individuals with MHR <0.37 had inferior OS

compared to those with higher values (71.7 vs. 84.4 months; p=0.041) (Figure 2C). No significant OS differences were observed for gender (p=0.842), tumor location (p=0.466), LVI (p=0.323), or perineural invasion (p=0.285) (Table 3).

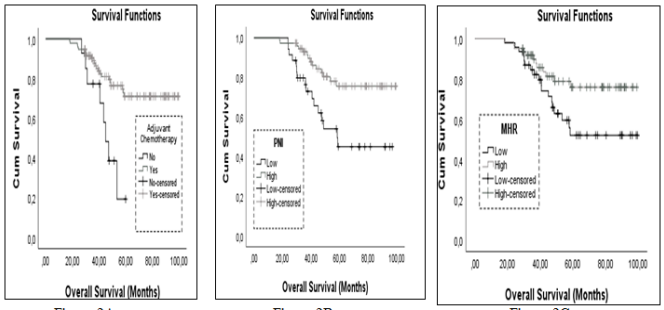

Figure 2A Figure 2B Figure 2C

Figure 2. Kaplan-Meier curves for OS stratified by adjuvant chemotherapy status, PNI, and MHR

OS: Overall survival, PNI: Prognostic Nutritional Index, MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein

Table 3. Differences between the median survival times of patients and variables

	Median	SE	95% CI Min	95% CI Max	p
OS (months)	78.9	3.1	72.8	85.1	
Gender					
Female	77.9	4.5	69.0	86.8	0.842
Male	78.6	4.1	70.5	86.6	
Adjuvant chemotherapy					
No	45.6	3.1	39.5	51.8	0.002**
Yes	82.2	3.2	76.0	88.5	
MHR (<0.37)					
Low	71.7	4.9	62.0	81.4	0.041*
High	84.4	3.8	77.1	91.8	
PNI (<46.8)					
Low	65.3	5.6	54.4	76.2	0.003**
High	85.1	3.4	78.4	91.8	

*p<0.05, **p<0.01, Kaplan meier, Log rank test, SE: Standard error, CI: Confidence interval, Min: Minimum, Max: Maximum, OS: Overall survival, MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein, PNI: Prognostic Nutritional Index

The median disease-free survival (DFS) for the cohort was 16.8±1.0 months (Table 4). Univariate analysis indicated significantly worse DFS in patients with grade 3 tumors (p=0.008), presence of perineural invasion (p=0.043) (Figure 3A), emergency surgery (p=0.013) (Figure 3B), and low nutritional PNI values (p=0.021) (Figure 3C). No significant DFS differences was observed for MHR (Figure 3D). Age >65 years (p=0.040) (Figure 3E).

Multivariate Cox regression analysis revealed that only perineural invasion remained an independent predictor of shortened DFS (HR: 2.361; p=0.038), while other variables did not retain statistical significance (Table 5). Multivariate analysis for OS was not performed due to the limited number of death events (n=30), which may reduce statistical power and increase the risk of overfitting. The median follow-up time for surviving patients was 42 months (range: 18-96 months), calculated using the reverse Kaplan-Meier method.

Table 4. Differences between the variables and the median disease-free survival time of the patients

	Median	SE	95% CI Min	95% CI Max	p
DFS (months)	16.8	1.0	14.8	18.8	
Gender					
Female	16.9	1.7	13.7	20.2	0.703
Male	16.6	1.2	14.2	19.0	
Hystologic grade					
Grade 1	17.6	1.3	15.0	20.2	0.008**
Grade 2	17.5	1.8	14.0	21.0	
Grade 3	10.7	1.7	7.4	13.9	
Perineural invasion					
No	17.7	1.5	14.8	20.6	0.043*
Yes	15.5	1.2	13.1	17.8	
Emergency operation status					
No	17.9	1.2	15.6	20.2	0.013*
Yes	13.0	1.7	9.8	16.3	
PNI (<46.8)					
Low	11.4	1.2	9.2	16.7	0.021*
High	20.5	1.4	17.7	23.3	
Age					
65 and under	17.8	1.3	15.2	20.4	0.040*
65 upper	14.8	1.4	12.1	17.5	

*p<0.05, **p<0.01, Kaplan meier, Log rank test, SE: Standard error, CI: Confidence interval, Min: Minimum, Max: Maximum, DFS: Disease-free survival, PNI: Prognostic Nutritional Index

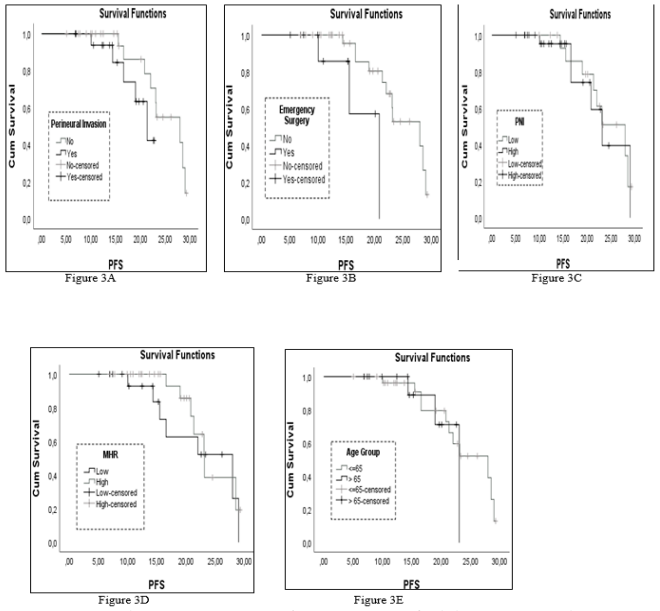

Figure 3A Figure 3B Figure 3C Figure 3D Figure 3E

Figure 3. Kaplan-Meier curves for DFS stratified by perineural invasion, surgery urgency, PNI, MHR, and age group

DFS: Disease-free survival, PNI: Prognostic Nutritional Index, MHR: Monocyte-to-HDL cholesterol ratio, HDL: High-density lipoprotein

Table 5. Examining the factors affecting the median disease-free survival time

		Hazard ratio	95% CI		
			Min	Max	
Hystologic grade					
Grade 1					0.134
Grade 2	-1.297	0.273	0.076	0.980	0.046*
Grade 3	-1.066	0.344	0.091	1.307	0.117
Perineural invasion	0.859	2.361	1.050	5.308	0.038*
Emergency operation	0.659	1.933	0.734	5.094	0.182
PNI (<46.8)					
Low	0.742	2.100	0.954	4.642	0.067
High					
Age					
65 and under					
65 upper	0.688	1.989	0.829	4.772	0.124

*p<0.05, **p<0.01, Multivariate Cox regresyon test, **Perineural invasion emerged as the only independent predictor of worse DFS (HR: 2.361; p=0.038), surpassing other variables in prognostic significance. DFS: Disease-free survival, CI: Confidence interval, Min: Minimum, Max: Maximum, PNI: Prognostic Nutritional Index

DISCUSSION

This study provides evidence supporting the prognostic utility of the MHR and PNI in patients with stage III CRC. Our findings demonstrate that lower MHR and PNI values are significantly associated with poorer OS and DFS, reinforcing the role of systemic inflammation and nutritional status in cancer progression.[14-16,19]

The considerable survival benefit observed with adjuvant chemotherapy reaffirms current clinical guidelines.[6] The discrepancy in OS between treated and untreated patients (82.2 vs. 45.6 months; p=0.002) is consistent with the IDEA collaboration and molecular subtype studies demonstrating survival benefit particularly in CMS2 and CMS4 subtypes.[6,17] The observed OS difference in the non-chemotherapy group may be confounded by higher comorbidity burden.

The prognostic impact of PNI aligns with previous reports. A Japanese prospective study reported 5-year OS below 58% for patients with PNI <45, compared to 78% for those with higher scores.[16] In our cohort, a similar pattern was observed-patients with PNI <46.8 had a median OS of 65.3 months, while those with higher values reached 85.1 months (p=0.003), underscoring the importance of nutritional resilience in CRC prognosis. Given that PNI predicted both OS and DFS, its role as a modifiable risk factor through nutritional interventions deserves attention. Early nutritional support, particularly in patients with borderline PNI values, may improve treatment tolerance and reduce recurrence.

Likewise, our data support the relevance of MHR as a marker of tumor-promoting inflammation. Monocytes promote tumor progression via angiogenesis and immune suppression,[10] while HDL exerts anti-inflammatory effects by inhibiting monocyte adhesion.[11] This mechanistic balance explains MHR's prognostic value. In our analysis, patients with MHR <0.37 experienced inferior OS (71.7 months vs. 84.4 months; p=0.041), corroborating findings from Korean retrospective cohorts and recent meta-analyses.[19]

Interestingly, MHR was significantly associated with OS but not DFS. This discrepancy may reflect the long-term systemic impact of chronic inflammation on survival, rather than short-term recurrence risk. Monocyte-driven mechanisms, such as immune exhaustion and vascular remodeling, may accelerate mortality in the absence of direct tumor progression.

Pathological perineural invasion, a classic histopathologic feature, was identified as an independent predictor of DFS (HR: 2.36; p=0.038), consistent with previous findings by Ishii et al.[17] This supports the hypothesis that perineural invasion reflects more aggressive tumor biology with greater potential for recurrence. Additionally, reduced DFS in patients undergoing emergency surgery highlights the need for early detection and optimized surgical protocols.[12]

The high rate of recurrence and death in our cohort may partially stem from the symptomatic presentation of most cases. As evidenced in European registry studies, patients diagnosed through screening had 5-year OS rates exceeding 83%, compared to only 57.5% among symptomatic patients,[22] suggesting that delayed diagnosis contributes to poorer outcomes.

In conclusion, low PNI and high MHR reflect malnutrition and systemic inflammation, directly impairing OS/DFS. These accessible biomarkers (requiring routine blood tests) offer clinical utility for risk stratification. The 45.6-month OS without adjuvant chemotherapy mirrors IDEA trial data,[6] emphasizing non-compliance as a modifiable risk factor. As the sole independent DFS predictor in multivariate analysis, presence of perineural invasion signifies aggressive tumor biology and neural spread-aligning with Ishii et al.'s[17] findings. Reduced DFS in older patients and emergent surgeries highlights needs for geriatric oncology protocols and early interventions.[12]

Our findings suggest that lower MHR and PNI values may identify high-risk patients who could benefit from more intensive monitoring and tailored therapeutic strategies. Given their accessibility and cost-effectiveness, MHR and PNI could be incorporated into standard risk stratification algorithms alongside conventional clinical and pathological parameters. Future prospective studies with larger, multicenter cohorts are warranted to confirm these observations and further validate their prognostic value. Our study is among the few to explore MHR and PNI in a Turkish cohort. Regional dietary habits, access to screening programs, and inflammatory profiles may differ from Western populations, thereby supporting the value of population-specific analyses.

Limitations

This study has several limitations. First, the relatively small sample size (n=109) may limit the statistical power of subgroup analyses and increase the risk of errors. Although multivariate analysis was restricted to ≤5 covariates to avoid overfitting, larger prospective cohorts are needed to validate our findings. In addition, the cut-off values identified via ROC analysis were not validated in an independent external

cohort, which limits their generalizability and may introduce overfitting bias.

CONCLUSION

While our findings are compelling, several limitations warrant consideration. The retrospective design introduces potential selection bias, and the lack of molecular data limits insights into tumor biology. Nonetheless, the inclusion of practical, low-cost markers such as MHR and PNI enhances the study's clinical applicability. Future prospective and multicenter studies are needed to validate these findings and further refine prognostic stratification models. In summary, this study highlights the clinical relevance of preoperative MHR and PNI as prognostic indicators in patients with stage III CRC. Both markers, derived from routine blood tests, were significantly associated with survival outcomes, offering insight into the interplay between systemic inflammation, nutritional status, and tumor progression.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Van Training and Research Hospital Non-interventional Clinical Researches Ethics Committee (Date: 04.07.2025, Decision No: GOKAEK/2025-05-11).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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