e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 434-439

Original Research Article

Role of Mean Platelet Volume as a Prognostic Marker in Carcinoma Stomach

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Received: 01-08-2025 / Revised: 15-09-2025 / Accepted: 21-10-2025

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Conflict of interest: Nil

Abstract

Background: Platelet activation and inflammatory responses play a key role in tumor progression and metastasis. Mean platelet volume (MPV), a routinely available hematologic parameter, reflects platelet activation and has been proposed as a potential biomarker in several malignancies. This study aimed to evaluate the prognostic significance of MPV in patients with carcinoma stomach.

Material and Methods: A prospective observational study was conducted on 150 patients with histologically confirmed, treatment-naïve gastric carcinoma. Baseline MPV was measured before initiation of therapy using an automated hematology analyzer. Receiver operating characteristic (ROC) curve analysis identified the optimal MPV cut-off for predicting 2-year overall survival (OS). Patients were categorized into low (≤9.5 fL) and high (>9.5 fL) MPV groups. Survival analysis was performed using Kaplan–Meier curves and Cox proportional hazards regression, adjusting for age, ECOG status, tumor stage, platelet count, and neutrophil-to-lymphocyte ratio (NLR).

Results: The mean age of the cohort was 58.4 ± 11.2 years, with 65.3% males. The median MPV was 9.7 fL (IQR 8.9–10.6). ROC analysis yielded an AUC of 0.68 (95% CI 0.60–0.76) with an optimal cut-off of 9.5 fL for 2-year OS. Patients with low MPV (≤9.5 fL) had a significantly lower 2-year OS (54.1%) compared with those with high MPV (>9.5 fL, 73.8%) ($p = 0.004$). In multivariable Cox regression, high MPV remained an independent predictor of improved survival (adjusted HR = 0.56, 95% CI 0.35–0.89, $p = 0.014$), after adjusting for disease stage and performance status.

Conclusion: A lower pre-treatment MPV is associated with advanced disease and poorer survival in gastric carcinoma. MPV may serve as a simple, cost-effective, and independent prognostic biomarker for risk stratification in these patients.

Keywords: Mean Platelet Volume; Gastric Carcinoma; Prognostic Biomarker; Survival Analysis; Platelet Indices.

Introduction

Gastric carcinoma continues to be a major global health challenge: despite declining incidence in some regions, it remains one of the leading causes of cancer mortality worldwide, particularly in lower- and middle-income countries. Early diagnosis and effective prognostic stratification are essential to improving outcomes, yet reliable, low-cost biomarkers suitable for widespread use remain limited. Platelets are increasingly recognized as active participants in cancer biology, not merely in hemostasis. They contribute to tumour progression, angiogenesis, immune evasion and metastasis, through secretion of growth factors, cytokines, and interactions with tumour cells and the tumour microenvironment. Accordingly, platelet indices derived from routine complete blood count

(CBC)—including mean platelet volume (MPV)—have attracted interest as potential biomarkers in solid tumours [1,2]. MPV reflects the average size of circulating platelets and serves as a surrogate for platelet activation: larger platelets tend to be metabolically and enzymatically more active, with greater pro-thrombotic potential [3]. In oncologic settings, elevated MPV has been associated with worse outcomes in several tumour types including gastric, colorectal, lung and breast cancers [4,5]. Specifically in gastric cancer, early evidence suggests that higher preoperative MPV correlates with deeper tumour invasion, lymph node metastasis and reduced survival [6].

However, findings in gastric carcinoma are heterogeneous: some studies reported no significant association between MPV and tumour stage or prognosis, and cut-offs for MPV vary widely across cohorts [7]. Despite these inconsistencies, the advantages of MPV—its low cost, wide availability, and minimal added burden—make it an attractive candidate for prognostic assessment.

Given the need for validated prognostic biomarkers in gastric carcinoma and the promising but inconsistent existing data on MPV, this study was designed to evaluate the prognostic significance of pre-treatment MPV in patients with carcinoma of the stomach. We hypothesised that higher baseline MPV values would be independently associated with adverse survival outcomes, even when controlling for established clinicopathologic variables.

Material and Methods

Study design and setting: This prospective observational cohort study was conducted at a tertiary care teaching hospital in India. The study aimed to evaluate whether pre-treatment mean platelet volume (MPV) predicts clinicopathologic outcomes and survival in patients with histologically confirmed carcinoma of the stomach.

Sample size and sampling: A total of 150 consecutive patients with newly diagnosed, treatment-naïve gastric carcinoma who met eligibility criteria were enrolled. The sample size was selected to provide adequate power for survival analyses and multivariable modeling of MPV alongside established prognostic covariates (T stage, nodal status, performance status), assuming approximately 40–50 outcome events during follow-up. Consecutive sampling of eligible patients presenting to the oncology clinic and surgical services was used.

Inclusion criteria

- Age ≥18 years.
- Histologically confirmed adenocarcinoma of the stomach.
- Treatment-naïve at time of baseline blood sampling (no prior chemotherapy, radiotherapy, or major surgery for the current malignancy).
- Planned for curative-intent surgical resection or systemic therapy as per tumor board decision.
- Provided written informed consent for participation and follow-up.

Exclusion criteria

- Active infection or inflammatory disease causing potential acute platelet activation.
- Use of antiplatelet agents (aspirin, clopidogrel) or anticoagulants within 14 days prior to baseline sample.
- Known hematologic disorders affecting platelet indices (e.g., immune thrombocytopenia, myeloproliferative neoplasm).
- Recent major bleeding or blood transfusion within 14 days prior to sampling.
- Severe hepatic impairment or end-stage renal disease (requiring dialysis).

Clinical data collection and follow-up: Baseline demographic data (age, sex), comorbidities, performance status (ECOG), tumor location, histologic grade, and clinical stage (based on contrast-enhanced CT and endoscopy) were recorded. Treatment modality (surgery, adjuvant or neoadjuvant chemotherapy, palliative therapy) and dates of intervention were documented. Patients were followed for a minimum of 24 months or until death, whichever occurred first. Outcomes included overall survival (OS; time from diagnosis to death from any cause) and disease-free survival (DFS; time from curative-intent surgery to documented recurrence or death). Date and site of recurrence and any adjuvant treatments were recorded.

Blood sampling and laboratory methods: Peripheral venous blood was drawn at baseline (prior to any treatment) using standard phlebotomy technique. Samples for complete blood count (CBC) were collected into K2-EDTA tubes and processed within 2 hours of collection to minimize pre-analytical variation in platelet indices. MPV and other CBC parameters were measured using an automated hematology analyzer according to manufacturer instructions. Calibration and internal quality controls were maintained throughout the study period. MPV values were reported in femtoliters (fL). If multiple baseline samples were inadvertently obtained, the earliest pre-treatment value was used for analysis.

Definition of MPV groups and cut-off selection: MPV was analyzed both as a continuous variable and as a categorical variable. An optimal dichotomous cut-off for MPV to predict 2-year overall survival was determined by receiver operating characteristic (ROC) curve analysis using Youden's index. We also examined tertiles and quartiles of MPV in sensitivity analyses to evaluate dose–response relationships.

Additional laboratory and pathological variables: Baseline hemoglobin, platelet count, neutrophil-to-lymphocyte ratio (NLR), and C-reactive protein (when available) were recorded. Pathology reports provided tumor differentiation, lymphovascular invasion, and pathologic TNM stage for surgical cases. For patients receiving neoadjuvant therapy, pre-treatment biopsies and post-treatment resection pathology were documented as available.

Statistical Analysis: Continuous variables are presented as mean ± standard deviation or median (interquartile range) depending on distribution; categorical variables are presented as counts and percentages. Baseline differences between MPV groups were compared using Student's t-test or Mann–Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Survival analysis was performed using Kaplan–Meier curves and the log-rank test to compare OS and DFS between MPV-defined groups. Cox proportional hazards regression was used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for the association between MPV and outcomes. Multivariable models adjusted for pre-specified covariates: age, sex, ECOG performance status, clinical/pathologic stage (T and N), tumor grade, and baseline platelet count.

The proportional hazards assumption was tested by Schoenfeld residuals. Missing data were handled by complete-case analysis for primary models; sensitivity analyses using multiple imputation were planned if missingness exceeded 5%.For ROC analysis, area under the curve (AUC) with 95% CI was reported; optimal MPV threshold was derived from Youden index with sensitivity and specificity. A two-sided p-value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 26.0 (IBM Corp.) and R version 4.x (survival and pROC packages).

Results

A total of 150 patients with histologically confirmed carcinoma stomach were enrolled. The mean age was 58.4 ± 11.2 years, with males comprising 65.3% of the study population. Most patients (74.7%) had an ECOG performance status of 0–1. Regarding stage distribution, 40% were in stages I–II, 37.3% in stage III, and 22.7% in stage IV disease. The distal stomach (antrum and pylorus) was the most frequent tumor site (58.7%). Histopathologically, moderately differentiated adenocarcinoma was the predominant type (52%). The mean baseline hemoglobin level was 11.2 ± 1.8 g/dL, and the mean platelet count was 260 ± 75 $\times 10^3/\mu L$. The median neutrophil-to-lymphocyte ratio (NLR) was 3.1 (IQR 2.1–5.0). The median follow-up period was 26 months (range, 3–36 months) (Table 1).

The mean MPV in the cohort was 9.8 ± 1.2 fL, with a median of 9.7 fL (IQR 8.9–10.6). ROC curve analysis for prediction of 2-year overall survival identified an optimal MPV cut-off of 9.5 fL (Youden index = 0.30), which yielded a sensitivity of 72% and a specificity of 58% (AUC = 0.68; 95% CI, 0.60–0.76) (Table 3). Based on this threshold, 85 patients (56.7%) were classified as having low MPV (≤9.5 fL), while 65 patients (43.3%) had high MPV (>9.5 fL) (Table 2).

At baseline, patients with low MPV had a higher proportion of advanced disease (71.8% vs 44.6%, $p < 0.05$) and elevated inflammatory indices (median NLR 3.6 vs 2.6). Mean platelet counts were comparable between groups (268 $\times 10^3/\mu L$ vs 249 $\times 10^3/\mu L$).

During the follow-up period, 56 deaths (37.3%) occurred. The overall 2-year survival rate was 62.0%. Kaplan–Meier analysis revealed significantly poorer survival among patients with low MPV compared to those with high MPV. The 2-year overall survival was 54.1% in the low MPV group versus 73.8% in the high MPV group (log-rank p = 0.004). The median overall survival for low MPV patients was 28 months, whereas the median survival was not reached for the high MPV group (Table 4).

On univariable analysis, low MPV, poor performance status, higher stage, and elevated NLR were significantly associated with reduced overall survival. In the multivariable Cox model, high MPV (>9.5 fL) remained independently associated with better survival (adjusted HR = 0.56, 95% CI 0.35–0.89, p = 0.014). Advanced disease stage (Stage III–IV) and poor performance status (ECOG ≥2) were the strongest adverse predictors (HR = 3.45 and 2.10, respectively, both $p < 0.01$). Elevated NLR (≥3) was also associated with poorer outcome (HR = 1.75, p = 0.018) (Table 5). Platelet count and age were not significant predictors in the adjusted model.

Table 1: Baseline demographic, clinical and laboratory characteristics (n = 150)

Variable	Value
Age (years), mean ± SD	58.4 ± 11.2
Sex, n (%)	
— Male	98 (65.3)
— Female	52 (34.7)
ECOG performance status, n (%)	
— 0–1	112 (74.7)
— ≥2	38 (25.3)
Clinical stage (AJCC), n (%)	
— I–II	60 (40.0)
— III	56 (37.3)
— IV	34 (22.7)
Tumor location, n (%)	
— Distal (antrum/pylorus)	88 (58.7)
— Proximal (cardia/body)	62 (41.3)
Histologic grade, n (%)	
— Well differentiated	30 (20.0)
— Moderately differentiated	78 (52.0)
— Poorly differentiated	42 (28.0)
Baseline hemoglobin (g/dL), mean ± SD	11.2 ± 1.8
Baseline platelet count ($\times10^3/\mu L$), mean ± SD	260 ± 75
Neutrophil-to-lymphocyte ratio (NLR), median (IQR)	3.1 (2.1–5.0)
Follow-up, median months (range)	26 (3–36)

Table 2: MPV distribution and grouping (n = 150)

Metric	Value
MPV (fL), mean ± SD	9.8 ± 1.2
MPV (fL), median (IQR)	9.7 (8.9–10.6)
MPV cut-off by Youden index (ROC) for 2-yr OS	9.5 fL
MPV groups (dichotomized)	
— Low MPV (≤ 9.5 fL), n (%)	85 (56.7)
— High MPV (> 9.5 fL), n (%)	65 (43.3)

Baseline comparison (selected variables) by MPV group:

Variable	Low MPV ≤9.5 (n = 85)	High MPV >9.5 (n = 65)
Age, mean ± SD (yrs)	58.7 ± 11.4	57.9 ± 10.9
ECOG ≥2, n (%)	24 (28.2)	14 (21.5)
Stage III–IV, n (%)	61 (71.8)	29 (44.6)
Baseline Hb (g/dL), mean ± SD	10.9 ± 1.9	11.7 ± 1.6
Platelets ($\times10^3/\mu L$), mean ± SD	268 ± 78	249 ± 70
NLR median (IQR)	3.6 (2.4–5.6)	2.6 (1.9–3.7)

Table 3: ROC analysis for MPV predicting 2-year overall survival (OS)

Metric	Value
Outcome evaluated	2-year overall survival
AUC (95% CI)	0.68 (0.60 – 0.76)
Optimal cut-off (Youden index)	MPV = 9.5 fL
Sensitivity at cut-off	72%
Specificity at cut-off	58%
Youden index	0.30

Table 4: Kaplan–Meier 2-year overall survival by MPV group

Group	n	2-yr survivors, n (%)	2-yr OS (%)	Median OS (months)	Log-rank p
Overall cohort	150	93	62.0	Not reached (median follow-up 26 mo)	—
Low MPV (≤9.5 fL)	85	46	54.1	28.0	0.004 (vs high MPV)
High MPV (>9.5 fL)	65	48	73.8	Not reached	

[Number of deaths by group | Low MPV: 39 /85 (45.9%); High MPV: 17 /65 (26.2%); Total deaths: 56 /150 (37.3%)]

Table 5: Multivariable Cox proportional hazards model for overall survival (n = 150)

Variable	Adjusted HR	95% CI	p-value
High MPV (>9.5 fL) (ref = Low MPV)	0.56	0.35 – 0.89	0.014
Age (per 10-yr increase)	1.12	0.90 – 1.40	0.30
ECOG ≥2 (ref = 0–1)	2.10	1.30 – 3.40	0.002
Stage III–IV (ref = I–II)	3.45	2.10 – 5.70	<0.001
Platelet count (per 100 ×10³/µL)	1.05	0.90 – 1.22	0.55
NLR ≥3 (ref <3)	1.75	1.10 – 2.80	0.018

Discussion

In this prospective cohort of 150 patients with treatment-naïve gastric carcinoma, a lower baseline MPV (≤9.5 fL) was associated with more advanced stage, higher inflammatory indices and significantly worse overall survival. These observations add to a growing but heterogeneous body of literature that implicates platelet size and function as clinically meaningful correlates of tumour biology and outcome.

Biologically, MPV is considered a surrogate of platelet activation and reactivity: larger platelets contain more dense granules and pro-thrombotic/pro-inflammatory mediators and are functionally more active than smaller platelets. Platelets interact with tumour cells and the tumour microenvironment to promote angiogenesis, shield circulating tumour cells from immune clearance, and facilitate metastatic seeding; such mechanistic links support the plausibility of platelet indices as prognostic biomarkers in solid cancers. The conceptual framework linking platelet activity to cancer progression has been reviewed in detail [8].

Clinical investigations of MPV in gastric cancer and other solid tumours have produced variable results. Some studies and composite platelet indices (for example MPV/platelet count ratio) have demonstrated independent prognostic value and improved model discrimination when combined with conventional clinicopathologic parameters, supporting MPV's potential utility as part of multivariable prognostic tools. For instance, preoperative combined indices incorporating MPV have been proposed to augment postoperative prognostic models in resectable gastric cancer [9].

At the same time, aggregate analyses and meta-analyses report heterogeneity in the direction and magnitude of associations between MPV and survival across tumour types and cohorts. In gastric cancer specifically, pooled data indicate a statistically significant association between MPV and outcomes in some analyses, but effect estimates and optimal cut-offs have varied substantially between studies, reflecting differences in patient mix, disease stage, assay methodology and timing of measurement. Such heterogeneity underscores the need for careful interpretation and contextualization of single-centre findings. [10] Pre-analytical and analytical factors substantially influence MPV values and likely contribute to between-study variability. The choice of anticoagulant (EDTA vs citrate), the time interval between venipuncture and measurement, and the hematology analyzer model and lab calibration procedures can all alter MPV measurements; standardisation of sample handling and reporting is therefore essential if MPV is to be translated into clinical decision-making. Several methodologic studies and reviews document these sources of variance and recommend strict, reproducible protocols for MPV measurement. [11,12]

Our study's finding that lower MPV was associated with worse outcome contrasts with reports that higher MPV predicts adverse prognosis in some gastric cancer series; this apparent inconsistency may reflect differences in cohort composition (e.g., proportions of early versus advanced disease), the interplay of systemic inflammation and bone marrow response, or the use of combined indices (MPV/PC) rather than MPV alone. Recent reports indicate that composite measures such as MPV/platelet count or MPV combined with inflammatory markers may provide greater prognostic accuracy than MPV alone and warrant exploration in future validation cohorts. [13,14]

Strengths of the present work include a prospective design, standardized baseline sampling prior to any therapy, and multivariable survival modelling that adjusted for established prognostic covariates (stage, performance status, NLR). Limitations deserve emphasis: this single-centre cohort may not capture the full biological or therapeutic heterogeneity of gastric cancer populations elsewhere; the MPV cut-off identified by ROC in our sample requires external validation; and although we minimized pre-analytic variability by processing samples promptly, residual analytic differences inherent to automated analyzers cannot be fully excluded.

Conclusion

The present study demonstrates that mean platelet volume (MPV) has a significant prognostic association in patients with carcinoma stomach. Given that MPV is a routinely available, cost-effective hematological parameter, it may be integrated into pre-treatment risk assessment models to help identify high-risk patients who could benefit from closer monitoring and aggressive management. However, larger multicentric and prospective studies are warranted to validate its predictive reliability and to establish standardized MPV cut-off values for clinical application in gastric carcinoma.

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