Cancer Management and Research

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ORIGINAL RESEARCH

Ki-67 and Platelet-to-Lymphocyte Ratio (PLR) as Predictors of Progression-Free Survival in Metastatic Breast Cancer Receiving CDK4/6 Inhibitors: Clinical Implications

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Objective: We aimed to investigate the prognostic power of Platelet-to-Lymphocyte Ratio (PLR) and Ki-67 index in predicting progression-free survival (PFS) in patients with metastatic HR+/HER2- breast cancer treated with CDK4/6 inhibitors.

Materials and Methods: In this retrospective study, 121 patients who received CDK4/6 inhibitors (palbociclib, ribociclib) at the Department of Medical Oncology, Dokuz Eylul University, between January 2015 and January 2025, were analyzed. PLR was calculated using baseline complete blood count parameters. The optimal cut-off value of 168.37 was determined using ROC analysis, and patients were stratified into low and high PLR groups. Survival analyses were performed using the Kaplan-Meier method and Cox regression.

Results: A lower progression rate was observed in the high PLR group (p=0.01), although PLR was not identified as an independent prognostic factor for PFS in multivariate analysis. Patients with lung metastases showed a higher proportion of low PLR (p=0.04).Although PLR was associated with PFS in univariate analysis, it did not retain independent prognostic significance in multivariate analysis. Conversely, Ki-67 was significantly associated with shorter PFS in both univariate (p<0.001) and multivariate analyses (p=0.007).

Conclusion: While PLR did not independently predict survival outcomes, it may provide complementary information regarding metastatic distribution and disease behavior. Ki-67 remains a strong and independent prognostic marker for PFS in metastatic HR+/HER2- breast cancer. Our findings indicate that Ki-67 is the strongest and most consistent independent prognostic marker in this patient population.

Keywords: CDK4/6 inhibitors, platelet-to-lymphocyte ratio, Ki-67, metastatic breast cancer, progression-free survival

Introduction

Cyclin-dependent kinase 4 and 6 (CDK4/6) inhibitors, specifically palbociclib, ribociclib, and abemaciclib, achieve a significant increase in progression-free survival (PFS) and overall survival (OS) when combined with endocrine therapy for the treatment of metastatic hormone receptor-positive, HER2-negative breast cancer.Therefore, these agents are currently regarded as the standard of care in this clinical setting.[1]

Beyond tumor biology, the literature highlights the growing importance of systemic inflammatory biomarkers like the neutrophil-to-lymphocyte ratio (NLR), the platelet-to-lymphocyte ratio (PLR), the C-reactive protein-to-lymphocyte ratio (CLR), and the systemic immune-inflammation index (SII) for predicting outcomes in patients undergoing CDK4/6 inhibitor therapy.[2,3] Elevated baseline NLR values have repeatedly been identified as an independent predictor of inferior PFS and OS. Conversely, lower CLR levels have been associated with improved PFS and OS following CDK4/6 inhibitor treatment.[3]

A simple and inexpensive parameter, the PLR effectively signals immune activation and inflammation in the tumor microenvironment, leading to its established prognostic relevance in metastatic breast cancer.Several studies have demonstrated that higher PLR and NLR values are associated with shorter PFS and OS during CDK4/6 inhibitor therapy[4]. Similarly, composite indices incorporating monocyte-to-lymphocyte and neutrophil-to-lymphocyte ratios have been shown to predict therapeutic benefit from CDK4/6 inhibitors.[4] However, data directly evaluating the

https://doi.org/10.2147/CMAR.S584786
Received: 27 November 2025
Accepted: 23 February 2026
Published: 9 March 2026

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prognostic role of PLR in metastatic breast cancer patients treated with CDK4/6 inhibitors are limited and heterogeneous.

Moreover, elevated SII and pan-immune-inflammation value (PIV) levels have been linked to worse survival outcomes in meta-analyses.[5] Post-treatment inflammatory markers also appear prognostically relevant; a low NLR or a high absolute lymphocyte count (ALC) at the end of CDK4/6 inhibitor therapy has been associated with improved prognosis.[6,7]

A pivotal marker for tumor proliferation, the Ki-67 index distinguishes luminal A from luminal B subtypes, typically employing a 20% threshold in clinical settings. Furthermore, increased Ki-67 levels are consistently associated with decreased survival and aggressive tumor behavior.[8] Therefore, integrating Ki-67 assessment with inflammatory parameters may offer additional predictive value regarding treatment response and disease progression. Additionally, neutropenia the most frequently observed toxicity of CDK4/6 inhibitors has clinical significance due to its impact on treatment adherence and dose intensity. Exploring the relationship between inflammatory biomarkers and hematologic toxicities may aid in the development of more individualized treatment strategies.[9]

Despite increasing interest, studies evaluating the association between PLR and PFS in larger cohorts of metastatic breast cancer patients receiving CDK4/6 inhibitors remain limited. Furthermore, the prognostic performance of Ki-67, as well as its potential association with treatment-related hematologic toxicity, has not been fully elucidated in this population. The goal of this research was to retrospectively examine the link between PLR and PFS and to determine the prognostic importance of the Ki-67 index in metastatic breast cancer patients receiving CDK4/6 inhibitor therapy, providing new data to the field.

The novelty of this study lies in the combined evaluation of PLR and Ki-67 in a real-world cohort and in exploring their associations with progression-free survival, metastatic distribution, and hematologic toxicity within the same population.

Materials and Method

This study enrolled 121 patients diagnosed with histopathologically confirmed metastatic breast cancer who met specific criteria: receiving at least one cycle of a CDK4/6 inhibitor (either palbociclib or ribociclib) and having complete baseline blood count data available. Exclusion criteria included concurrent malignancy, inadequate laboratory results before starting CDK4/6 inhibitors, or incomplete follow-up information. Data collected from electronic medical records and patient files encompassed demographics (age, sex, menopausal status), tumor traits (histological subtype, receptor status, HER2 status, and metastasis sites), clinical factors (ECOG performance status, previous treatment lines), and treatment details (CDK4/6 inhibitor type, concomitant regimens, and duration).

To begin, the Platelet-to-Lymphocyte Ratio (PLR) was computed using baseline laboratory values. The cohort was subsequently divided into high and low PLR cohorts using an optimal threshold identified through receiver operating characteristic (ROC) curve analysis.The Ki-67 index was obtained from pathology reports of the primary tumor at the time of diagnosis.Progression-Free Survival (PFS) constituted the study's main outcome measure, representing the time elapsed between the commencement of the CDK4/6 inhibitor and the occurrence of radiologically confirmed disease progression or mortality due to any reason.

All statistical analyses were conducted using SPSS software (version 15; IBM Corp., Armonk, NY, USA). Categorical variables were compared using the chi-square test or Fisher's exact test, while continuous variables were expressed as mean ± standard deviation or median (minimum–maximum), as appropriate. Survival outcomes were evaluated using the Kaplan–Meier method, and differences between groups were assessed with the Log rank test. To determine whether PLR served as an independent prognostic factor, univariate and multivariate Cox proportional hazards regression analyses were performed. A p value of <0.05 was considered statistically significant.

Results

A total of 121 patients with metastatic breast cancer who were under treatment were included in the study. The median follow-up duration was 30 months (range: 3–108 months).The optimal cut-off value for PLR was determined as 168.37 using ROC curve analysis. The area under the ROC curve (AUC) for PLR was 0.64 (95% CI: 0.56–0.71), demonstrating moderate discriminatory ability, and the optimal cut-off value was 168.37.Accordingly, patients were categorized into two groups: low PLR (<168.37, n = 65) and high PLR (≥ 168.37, n = 56) (Table 1).

Table 1 Patient Characteristics According to PLR Groups

Parameter	Low PLR Group (<168.37) n (%)	High PLR Group (≥168.37) n (%)	Total n (%)	p-value
Age				0.59
<65	47 (55.0)	38 (45.0)	85 (70.3)	
≥65	18 (50.0)	18 (50.0)	36 (29.7)	
Survival status				0.49
Alive	40 (56.3)	31 (43.7)	71 (58.7)	
Dead	25 (50.0)	25 (50.0)	50 (41.3)	
CDK4/6 inhibitor				0.15
Palbociclib	34 (60.7)	22 (39.3)	56 (46.2)	
Ribociclib	31 (41.3)	34 (58.7)	75 (53.8)	
Combination partner				0.02
Letrozole	41 (63.0)	24 (37.0)	65 (53.7)	
Fulvestrant	24 (42.8)	32 (57.2)	56 (46.3)	
Progression status				0.01
Yes	45 (62.5)	27 (37.5)	72 (59.6)	
No	20 (40.8)	29 (59.2)	49 (40.4)	
Progesterone receptor (%)				0.22
<50%	35 (59.3)	24 (40.7)	59 (48.7)	
≥50%	30 (48.3)	32 (51.7)	62 (51.3)	
Estrogen receptor (%)				0.03
<50%	3 (25.0)	9 (75.0)	12 (10.0)	
≥50%	62 (56.8)	47 (43.2)	109 (90.0)	
Ki-67 index				0.96
<20%	31 (54.3)	26 (45.7)	57 (47.1)	
≥20%	34 (53.9)	29 (46.1)	63 (52.9)	
CDK4/6-related neutropenia				0.03
Grade 1	31 (65.9)	16 (34.1)	47 (38.8)	
Grade 2–4	34 (45.9)	40 (54.1)	74 (61.2)	
Lung metastasis				0.04
Yes	26 (66.6)	13 (33.4)	39 (32.2)	
No	39 (54.1)	43 (45.9)	72 (67.8)	

Abbreviations: PLR, platelet-to-lymphocyte ratio, CDK4/6, Cyclin-dependent kinase 4 and 6.

Demographic and Clinical Characteristics

No significant difference in age distribution (<65 vs ≥65 years) was found between the low and high PLR groups (p=0.59). Similarly, no significant difference was observed between the groups regarding survival status (alive vs

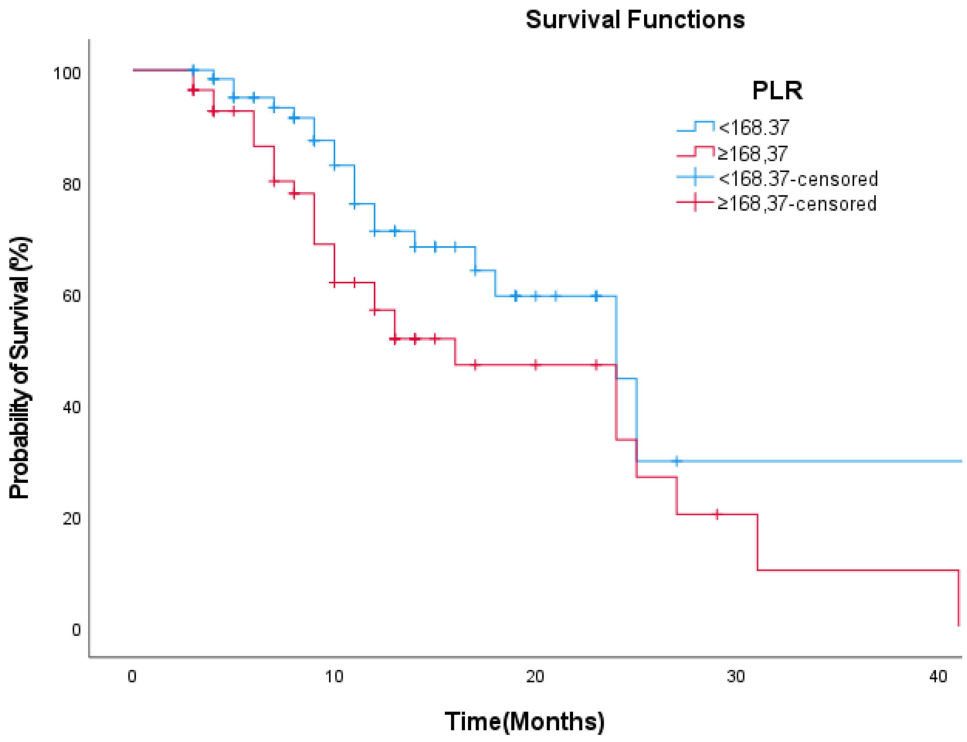


Figure 1 Kaplan–Meier curve for PFS according to PLR values.

deceased) (p=0.49). When evaluating the type of CDK4/6 inhibitor used, patients receiving palbociclib were more frequently observed in the low PLR group (%60.7), while patients using ribociclib were more frequently included in the high PLR group (%58.7); however, this difference was not statistically significant (p=0.15).

When evaluated in terms of combination therapy, patients treated with letrozole were more frequently represented in the low-PLR group (63%), whereas patients treated with fulvestrant were more common in the high-PLR group (57.2%). Among those who developed progression, the proportion of patients with low PLR was higher, and this difference was statistically significant (p = 0.01).

Analysis of receptor status revealed a significant association between lower estrogen receptor expression (<%50) and the high PLR group (p=0.03). However, progesterone receptor status showed no significant difference across the groups (p=0.22). Classification based on the Ki-67 index (<%20 vs.≥%20) also demonstrated no statistically significant distinction between the groups (p=0.96).

High PLR values were found to be associated with more advanced hematologic toxicity. Grade 2–4 neutropenia was significantly more frequent in the high-PLR group (p = 0.03). The higher proportion of low PLR among patients with lung metastases suggests that PLR does not show a direct positive relationship with lung metastasis (p = 0.04).

Progression-free survival was diminished among patients in the high PLR group, reflecting an inferior clinical result when benchmarked against the low PLR group. This PFS analysis was conducted using the Kaplan–Meier method, presented visually in Figure 1.

Cox Regression Analysis

Table 2 provides the summary of the Cox regression analysis, showing both univariate and multivariate results. Univariate analysis initially linked high Ki-67 expression (HR = 0.35, p < 0.001) and elevated PLR (≥168.37) (HR = 1.78, p = 0.053) with altered PFS risk. Crucially, in the multivariate model, high Ki-67 expression maintained its significance as an independent prognostic factor (HR = 0.36, p = 0.007), but the elevated PLR (≥168.37) lost its independent prognostic significance (HR = 1.40, p = 0.305).

Table 2 Univariate and Multivariate Cox Regression Analysis for PFS

Variable	Univariate HR (95% CI)	*p*-value	Multivariate HR (95% CI)	*p*-value
Age (≥65 vs <65)	1.39 (0.76–2.52)	0.283	0.73 (0.36–1.48)	0.388
Agent (Ribociclib vs Palbociclib)	1.24 (0.70–2.20)	0.469	1.55 (0.81–2.94)	0.185
Combination partner (Letrozole vs Fulvestrant)	0.72 (0.40–1.31)	0.283	0.63 (0.34–1.18)	0.152
ER ≥50%	0.56 (0.22–1.45)	0.232	0.35 (0.11–1.12)	0.076
PR ≥50%	1.73 (0.95–3.15)	0.071	1.46 (0.69–3.10)	0.323
High Ki-67	0.35 (0.19–0.65)	<0.001	0.36 (0.17–0.76)	0.007
Liver metastasis (yes vs no)	0.76 (0.41–1.40)	0.377	0.81 (0.42–1.59)	0.547
Lung metastasis (yes vs no)	0.51 (0.25–1.03)	0.062	0.49 (0.22–1.09)	0.081
PLR cut-off (≥168 vs <168)	1.78 (0.99–3.20)	0.053	1.40 (0.73–2.69)	0.305

Abbreviations: PFS, Progression free survival; PLR, platelet-to-lymphocyte ratio; ER, Estrogen receptor; PR, Progesteron receptor.

Discussion

In this retrospective study, we evaluated the prognostic value of PLR in 121 patients with metastatic hormone receptor–positive, HER2-negative breast cancer who were treated with CDK4/6 inhibitors. Our findings showed that progression-free survival was shorter in the high-PLR group, indicating poorer clinical outcomes compared with the low-PLR group; however, elevated PLR did not remain an independent prognostic factor in multivariate analysis. In our study, a discrepancy was observed between the distribution of progression events according to PLR and the time-dependent effect of PLR on PFS. This finding suggests that PLR may reflect the timing of progression rather than simply whether progression occurs. This suggests that the use of PLR as a standalone biomarker may be limited.[9,10]

CDK4/6 inhibitors have become the standard treatment option for this patient population by significantly prolonging survival outcomes. The PALOMA-3, MONALEESA, and MONARCH trials have consistently demonstrated clinically meaningful improvements in progression-free survival with these agents.[11,12] Nevertheless, identifying which patients are most likely to derive enhanced benefit remains a major clinical challenge.[13]

In recent years, inflammatory biomarkers have been increasingly investigated as potential predictors of treatment response. High NLR, recognized as one of the leading inflammatory markers, has been shown to correlate with worse PFS and OS in metastatic breast cancer patients receiving CDK4/6 inhibitors.[14] Likewise, accumulating data suggest that parameters such as CRP-to-lymphocyte ratio and PLR may have prognostic relevance in this setting.[4,15]

Moukas et al[4] demonstrated that monocyte-to-lymphocyte and neutrophil-to-lymphocyte ratios were significant predictors of benefit from CDK4/6 inhibitors. In a meta-analysis, Gong et al[16] reported that elevated PLR was associated with more advanced disease stage and poorer prognosis in breast cancer. Our results support these findings, indicating shorter PFS in the high-PLR group. While PLR did not retain independent significance in multivariate models, its potential role in providing preliminary prognostic insight during CDK4/6 inhibitor therapy may still be clinically valuable. Thus, although PLR cannot be considered a "gold-standard" biomarker, it may serve as a complementary parameter in clinical decision-making.

In our study, the Ki-67 proliferation index was a statistically significant predictor of PFS in both univariate and multivariate analyses. In contrast, Ki-67 was consistently associated with PFS in both univariate and multivariate analyses and emerged as the strongest independent prognostic marker in our study.This finding supports that Ki-67 retains its prognostic relevance not only in early-stage disease but also in the metastatic setting, particularly among patients treated with CDK4/6 inhibitors.This reinforces the prognostic value of Ki-67 not only as a marker of proliferative activity but also as an independent survival indicator.[8] As supported in the literature, this prognostic impact is particularly pronounced when the 20% cutoff is utilized.[17]

One notable finding was that low PLR values were more common among patients with lung metastases. Interestingly, this result contrasts with several earlier studies suggesting that systemic inflammatory burden may predispose to lung

metastatic spread.[18] Therefore, our data indicate that PLR is unlikely to be directly associated with lung metastasis, and caution should be taken when interpreting PLR as a surrogate marker for metastatic patterns.

Additionally, significant differences in neutropenia grades between PLR groups represent another important observation. This suggests a potential link between inflammatory biomarkers and treatment tolerability.[11] Neutropenia remains one of the most common adverse effects of CDK4/6 inhibitors and can affect dose intensity as well as treatment continuity. Understanding how PLR relates to the development of hematologic toxicities may help guide more individualized therapy strategies in the future.

Overall, although PLR did not emerge as an independent predictor of survival in multivariate analysis, it may still provide complementary prognostic information. In contrast, Ki-67 clearly stands out as a strong and independent prognostic marker. The associations observed between PLR, metastatic distribution, and hematologic toxicity also suggest that PLR should be interpreted within a broader clinical context.[19]

Given the retrospective design of this study, the results should be interpreted with caution, as residual confounding and unmeasured variables may have influenced the observed associations, and causal inferences cannot be definitively established.

Conclusion

In this study, elevated PLR was associated with poorer progression-free outcomes but did not retain independent prognostic significance, indicating that PLR alone may have limited utility as a robust biomarker. In contrast, Ki-67 consistently emerged as a strong and independent predictor of survival, underscoring its importance in risk stratification. Additionally, the observed associations between PLR, metastatic distribution, and treatment-related hematologic toxicity suggest that PLR may still provide meaningful complementary insight when interpreted alongside established clinical and pathological parameters. Integrating inflammation-based indices such as PLR with proliferative markers like Ki-67 may enhance prognostic assessment and help guide more personalized therapeutic decision-making in metastatic HR +/HER2- breast cancer.

Data Sharing Statement

The dataset linked to this research cannot be shared publicly, strictly adhering to national patient privacy and confidentiality rules. Nonetheless, anonymized data can be supplied if a suitable request is made and subsequently approved by the authors and the ethics committee.

Institutional Review Board Statement

The Clinical Research Ethics Committee of the Faculty of Medicine, Dokuz Eylul University, granted ethical clearance for the study (Decision No: 2022/32-06, Dated: 12.10.2022).This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Institutional Clinical Research Ethics Committee, and written informed consent was obtained from all participants.

Informed Consent Statement

Since the study design was retrospective and non-interventional, the requirement for obtaining patient informed consent was officially waived by the relevant ethics committee.

Funding

No external financial support was allocated to this research project.

Disclosure

The authors affirm that they have no competing interests to disclose.

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