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Prognostic Value of the Neutrophil-To-Lymphocyte Ratio (Nlr) in Hepatocellular Carcinoma Patients Treated With Atezolizumab Plus Bevacizumab: Updated Real-World Data From Vietnam

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Abstract

Background

Atezolizumab plus bevacizumab is the standard-of-care systemic regimen for advanced hepatocellular carcinoma (HCC). Accessible prognostic indicators are valuable in real-world practice, particularly in populations with a high burden of hepatitis B virus (HBV).

Objective

To evaluate the prognostic value of pretreatment neutrophil-to-lymphocyte ratio (NLR) and determine a pragmatic, clinically interpretable threshold for risk stratification.

Materials and methods

This retrospective, single-center study included 233 patients with advanced HCC treated with atezolizumab plus bevacizumab at the Vietnam National Cancer Hospital (February 2020 – April 2026; follow-up censored June 17, 2026). Candidate NLR thresholds were evaluated through Kaplan-Meier survival curves, multivariable Cox proportional hazards models, and internal validation techniques.

Results

Median patient age was 59 years; 91.4% were male, 72.5% had HBV etiology, and 31.3% met real-world trial-ineligibility criteria (IMbrave150-OUT). Median baseline NLR was 2.34 (IQR: 1.71–3.73). Continuous NLR was associated with overall survival (OS) and progression-free survival (PFS). Among candidate thresholds, NLR ≥ 3.6 provided optimal risk separation, significantly stratifying median OS (15.64 vs. 10.02 months; $p < 0.001$) and median PFS (4.34 vs. 3.25 months; $p = 0.039$). In the final multivariable Cox model (n = 210), NLR ≥ 3.6 remained an independent prognostic factor for both OS (adjusted HR 1.73; 95% CI, 1.13–2.66; $p = 0.012$) and PFS (adjusted HR 1.50; 95% CI, 1.05–2.14; $p = 0.025$).

Conclusion

Pretreatment NLR ≥ 3.6 serves as a simple, independent adjunctive biomarker for risk stratification in real-world patients with HCC receiving atezolizumab plus bevacizumab, including those ineligible for pivotal clinical trials. External validation in prospective cohorts is warranted.

1. Introduction

Hepatocellular carcinoma (HCC) is one of the most common cancers worldwide and the fourth leading cause of cancer-related deaths in Vietnam, where hepatitis B virus (HBV) infection is highly endemic [1]. Many patients are diagnosed at advanced stage (Barcelona C stage), when curative treatment is no longer feasible, for which systemic therapy serves as the primary treatment strategy [2, 3]. Atezolizumab, an anti-PD-L1 antibody, combined with bevacizumab, an anti-VEGF antibody, improved survival compared with sorafenib in IMbrave150 and became a standard option for unresectable HCC [6, 7]. Real-world outcomes are heterogeneous because clinical populations often include patients with impaired liver function, higher tumor burden, portal vein tumor thrombosis, extrahepatic spread, or prior systemic therapy [8–11].

In this context, simple, accessible, and low-cost prognostic biomarkers are urgently needed to refine clinical risk stratification and guide patient monitoring. The pretreatment neutrophil-to-lymphocyte ratio (NLR)—a routine blood-derived marker reflecting the balance between systemic inflammation and host immune response—has been broadly associated with survival outcomes in solid tumors and immune-checkpoint inhibitor therapies [12–16]. Nevertheless, two key gaps remain: the optimal NLR threshold in real-world cohorts receiving atezolizumab plus bevacizumab remains uncertain—with arbitrary cutoffs typically ranging from 3 to 5 [15–17]—and evidence from HBV-predominant Southeast Asian populations is scarce.

To address these limitations, This study aimed to evaluate the prognostic value of pretreatment NLR for overall survival (OS) and progression-free survival (PFS) in patients with advanced HCC receiving atezolizumab plus bevacizumab in real-world clinical practice, and to establish a clinically interpretable threshold for risk stratification.

2. Materials and methods

2.1. Study design and participants

This retrospective single-center study used medical records and longitudinal follow-up data from the Vietnam National Cancer Hospital. Eligible patients met the following criteria: (1) clinically or histologically diagnosed with HCC according to Vietnamese Ministry of Health criteria, (which align with AASLD/EASL criteria); (2) received at least one cycle of atezolizumab plus bevacizumab between February 2020 and April 2026, and (3) had complete baseline routine blood counts obtained within 14 days prior to treatment initiation. Follow-up was censored on 17 June 2026. Exclusion criteria included: (1) missing baseline NLR or survival outcomes; (2) active acute infections, hematologic disorders, or systemic corticosteroid therapy at sampling that could significantly alter leukocyte counts; and (3) secondary malignancies.

2.2. Variables and endpoints

Collected variables included demographics, viral etiology, Child-Pugh class, ALBI grade, ECOG performance status, BCLC stage, AFP, tumor burden, portal vein tumor thrombosis (PVTT/Vp

classification), extrahepatic spread, prior treatments. Pretreatment NLR was calculated as the absolute neutrophil count divided by the absolute lymphocyte count. Tumor response and disease progression were evaluated using dynamic CT or MRI scans approximately every 6–9 weeks according to modified RECIST (mRECIST) for HCC. PFS was defined from treatment initiation to clinical/radiologic progression or death, whichever occurred first. OS was defined from treatment initiation to death from any cause or last follow-up.

Patients were classified as IMbrave150-OUT if they met at least one real-world clinical criterion that would have rendered them ineligible for the landmark IMbrave150 trial. These criteria included: ECOG performance status ≥ 2, Child-Pugh class B or C or prior systemic therapy.

2.3. NLR cutoff selection and statistical analysis

Continuous variables were summarized as medians with interquartile ranges (IQR) and compared using the Mann-Whitney U test. Categorical variables were expressed as frequencies (%) and compared using the Chi-square or Fisher's exact test. Candidate prognostic variables were screened via univariable Cox proportional hazards regression for OS and PFS.

To determine a clinically interpretable NLR cutoff, candidate thresholds were evaluated using Kaplan-Meier survival curves with log-rank tests and exploring multivariable Cox regression models incorporating clinically relevant covariates. The final selected model included NLR cutoff, ECOG PS ≥ 1, ALBI grade 2–3, AFP ≥ 400 ng/mL, and Vp3–4. Child-Pugh class and ALBI grade were not combined in the same model to avoid collinearity regarding hepatic reserve. Complete-case multivariable Cox regression was performed. The non-linear continuous relationship between NLR and survival outcomes was evaluated using restricted cubic spline (RCS) Cox models with 3 knots. Internal stability of the selected model was evaluated using 500 bootstrap resamples. All statistical analyses were conducted using R software (version 4.3.2; R Foundation for Statistical Computing). A two-tailed p-value < 0.05 was considered statistically significant.

2.4. Ethics

This non-interventional study used de-identified data and was conducted in accordance with the Declaration of Helsinki. The study was approved by the Institutional Ethics Committee of Hanoi Medical University (Approval No. 452/QĐ-ĐHY, dated 20 February 2025). Patient informed consent was waived due to the retrospective nature of the study and the use of anonymized data.

3. Results

3.1. Study population and univariable screening

The NLR analysis cohort included 233 patients, with 125 death events and 181 progression/death events. Median age was 59 years (IQR, 48–65), 91.4% were male, 72.5% had HBV etiology, 84.5% were Child-Pugh A, and 92.3% were BCLC C. IMbrave150-OUT criteria were met by 31.3%. Median

pretreatment NLR was 2.34 (IQR, 1.71–3.73). Univariable screening showed that NLR as a continuous linear variable was associated with OS and PFS. ECOG PS, Child-Pugh B/C, AFP ≥ 400 ng/mL, tumor size ≥ 10 cm, PVTT/Vp > 0, Vp3-4, Vp4, and IMbrave150-OUT showed statistical significance or prognostic trend in at least one endpoint. The corresponding univariable hazard ratios are summarized in Table 1.

Table 1
Univariable screening of candidate prognostic variables for OS and PFS.

Variable	N analyzed	OS HR (95% CI); p	PFS HR (95% CI); p
NLR, per 1-unit increase	233	1.10 (1.03–1.16); 0.002	1.08 (1.01–1.15); 0.020
Age, per year	233	0.99 (0.98–1.01); 0.307	0.99 (0.98–1.01); 0.295
Male sex	233	1.20 (0.66–2.19); 0.543	0.78 (0.47–1.29); 0.338
HBV infection	233	1.25 (0.86–1.84); 0.242	1.21 (0.87–1.67); 0.255
HCV infection	225	0.80 (0.33–1.96); 0.623	0.96 (0.49–1.90); 0.917
ECOG PS ≥ 1	224	1.58 (1.09–2.29); 0.015	1.07 (0.79–1.46); 0.659
Child-Pugh B/C	233	1.87 (1.23–2.85); 0.004	1.60 (1.10–2.33); 0.014
ALBI grade 2–3	221	1.13 (0.79–1.62); 0.486	1.14 (0.85–1.54); 0.370
AFP ≥ 400 ng/mL	230	1.69 (1.17–2.45); 0.005	1.28 (0.95–1.72); 0.107
Tumor max ≥ 10 cm	212	1.54 (1.06–2.23); 0.024	1.37 (0.99–1.88); 0.054
Multiple tumors	212	1.35 (0.88–2.07); 0.169	1.17 (0.82–1.66); 0.393
PVTT/Vp > 0	233	2.45 (1.24–4.83); 0.010	1.73 (1.14–2.61); 0.009
Vp3–4	233	1.35 (0.94–1.93); 0.102	1.36 (1.01–1.83); 0.045
Vp4	233	1.67 (1.06–2.61); 0.026	1.37 (0.93-2.00); 0.107
Extrahepatic spread	233	0.97 (0.63–1.50); 0.897	1.29 (0.90–1.86); 0.162
Prior systemic therapy	233	1.16 (0.73–1.82); 0.532	1.08 (0.75–1.58); 0.671
IMbrave150-OUT	233	1.48 (1.03–2.14); 0.036	1.31 (0.96–1.78); 0.087

Cox analyses used all patients with available data for each variable; therefore, N varied by covariate completeness. The largest Cox models included all 233 patients.

3.2. NLR cutoff exploration by Kaplan-Meier analysis

Lower NLR thresholds, including 1.68 and the cohort median of 2.34, successfully stratified OS but failed to achieve clear stratification for PFS. Conversely, thresholds ranging from 3.0 to 3.6 provided consistent separation for both OS and PFS. Among these, an NLR threshold of ≥ 3.6 demonstrated the most robust OS stratification while maintaining statistical separation for PFS and preserving a clinically meaningful

high-risk subgroup size. **(detailed median survival and 12-month survival rates across candidate thresholds are provided in Supplementary Table S1)**

3.3. Multivariable model exploration and final cutoff selection

Exploration of multiple clinically grounded multivariable Cox models demonstrated that the prognostic independence of NLR was sensitive to covariate selection (**(Supplementary Table S2**). Comprehensive models incorporating extensive tumor burden metrics or Child-Pugh class (Models 1–4) failed to identify a single NLR cutoff that was independently predictive of both OS and PFS. Replacing any-grade PVTT with advanced thrombosis (Vp3–4) and utilizing ALBI grade instead of Child-Pugh class substantially improved model stability.

In the final selected model (Model 5: incorporating ECOG PS ≥ 1, ALBI grade 2–3, AFP ≥ 400 ng/mL, and Vp3–4), NLR cutoffs ranging from 3.3 to 3.6 maintained independent prognostic value for both endpoints. As detailed in Table 2, NLR ≥ 3.6 provided the optimal balance of statistical significance and hazard separation, remaining independently associated with inferior OS (adjusted HR 1.73; 95% CI, 1.13–2.66; p = 0.012) and shorter PFS (adjusted HR 1.50; 95% CI, 1.05–2.14; p = 0.025). Lower thresholds (< 3.0) and higher thresholds (≥ 4.0) lost statistical independence for one or both outcomes after multivariable adjustment.

Table 2
Multivariable Evaluation of NLR Cutoffs

NLR cutoff	n low/high	Adjusted OS HR (95% CI); p	Adjusted PFS HR (95% CI); p
2.0	82/151	1.33 (0.90–1.99); 0.157	1.16 (0.84–1.59); 0.369
2.5	123/110	1.35 (0.92–1.99); 0.122	1.26 (0.92–1.73); 0.144
3.0	146/87	1.48 (1.00-2.20); 0.051	1.37 (0.99–1.89); 0.059
3.3	160/73	1.53 (1.02–2.30); 0.041	1.42 (1.01–1.99); 0.044
3.5	167/66	1.54 (1.01–2.37); 0.046	1.46 (1.02–2.08); 0.039
3.6	170/63	1.73 (1.13–2.66); 0.012	1.50 (1.05–2.14); 0.025
3.7	173/60	1.88 (1.24–2.87); 0.003	1.43 (1.00-2.04); 0.050
4.0	184/49	1.66 (1.05–2.62); 0.031	1.33 (0.90–1.96); 0.147
5.0	203/30	1.53 (0.91–2.56); 0.111	1.45 (0.91–2.31); 0.115

Final model: NLR cutoff + ECOG PS ≥ 1 + ALBI grade 2–3 + AFP ≥ 400 ng/mL + Vp3-4. Adjusted HRs were estimated in complete-case models; the final model included 210 patients with 116 death events and 168 progression/death events.

3.4. Baseline and treatment characteristics according to NLR cutoff 3.6

After selection of the NLR ≥ 3.6 threshold, 170 patients were classified as NLR < 3.6 and 63 as NLR ≥ 3.6. The high-NLR group had more frequent ECOG PS ≥ 1 and IMbrave150-OUT status, and showed higher proportions of Child-Pugh B/C and AFP ≥ 400 ng/mL, although not all comparisons reached statistical significance. Treatment exposure was broadly similar between groups. DCR in the ITT population and subsequent regorafenib use were lower in the NLR ≥ 3.6 group; ORR did not differ clearly between groups. Baseline characteristics and response data are summarized in Table 3.

Table 3
Baseline characteristics and response according to NLR cutoff 3.6

Characteristic	NLR < 3.6 (n = 170)	NLR ≥ 3.6 (n = 63)	p-value
Child-Pugh B/C	21/170 (12.4%)	15/63 (23.8%)	0.052
ECOG PS ≥ 1	51/162 (31.5%)	29/62 (46.8%)	0.048
AFP ≥ 400 ng/mL	90/168 (53.6%)	41/62 (66.1%)	0.120
Tumor max, cm	6.8 [4.5–11.0]	7.8 [5.7–12.0]	0.072
HBV infection	119/170 (70.0%)	50/63 (79.4%)	0.209
ALBI grade 2–3	66/160 (41.2%)	31/61 (50.8%)	0.259
IMbrave150-OUT	46/170 (27.1%)	27/63 (42.9%)	0.032
Vp3–4	85/170 (50.0%)	27/63 (42.9%)	0.411
Extrahepatic spread	121/170 (71.2%)	51/63 (81.0%)	0.180
Male sex	158/170 (92.9%)	55/63 (87.3%)	0.271
HCV infection	11/163 (6.7%)	2/62 (3.2%)	0.523
Multiple tumors	115/154 (74.7%)	45/58 (77.6%)	0.795
Prior systemic therapy	28/170 (16.5%)	15/63 (23.8%)	0.275
First-line systemic Atezo/Bev	121/170 (71.2%)	45/63 (71.4%)	1.000
Prior TACE	44/170 (25.9%)	17/63 (27.0%)	0.998
Prior resection	39/170 (22.9%)	9/63 (14.3%)	0.205
Prior sorafenib	12/170 (7.1%)	9/63 (14.3%)	0.146
Prior lenvatinib	16/170 (9.4%)	6/63 (9.5%)	1.000
Response evaluable	141/170 (82.9%)	44/63 (69.8%)	0.044
ORR, ITT	37/170 (21.8%)	10/63 (15.9%)	0.363
DCR, ITT	75/170 (44.1%)	16/63 (25.4%)	0.010
Post-Atezo/Bev active treatment	78/124 (62.9%)	20/44 (45.5%)	0.051
Post-Atezo/Bev lenvatinib	28/134 (20.9%)	5/54 (9.3%)	0.092
Post-Atezo/Bev regorafenib	15/134 (11.2%)	1/54 (1.9%)	0.043
ORR, evaluable	37/141 (26.2%)	10/44 (22.7%)	0.788
DCR, evaluable	75/141 (53.2%)	16/44 (36.4%)	0.076

3.5. Survival outcomes and final Cox model

In the overall cohort, median OS was 13.44 months and median PFS was 4.11 months. Patients with NLR < 3.6 had longer OS than those with NLR ≥ 3.6 (15.64 vs. 10.02 months; log-rank p < 0.001). PFS was also longer in the low-NLR group (4.34 vs. 3.25 months; p = 0.039). These survival outcomes are summarized in Table 4.

Table 4
Survival outcomes according to NLR cutoff 3.6

Outcome	NLR group	N	Events	Median, months	12-month rate	24-month rate	p-value
OS	NLR < 3.6	170	84	15.64	58.4%	32.0%	< 0.001
OS	NLR ≥ 3.6	63	41	10.02	43.2%	13.8%	
PFS	NLR < 3.6	170	129	4.34	24.8%	10.6%	0.039
PFS	NLR ≥ 3.6	63	52	3.25	17.0%	3.6%	

In the final Cox model, NLR ≥ 3.6 remained associated with shorter OS and PFS after adjustment. AFP ≥ 400 ng/mL and Vp3-4 were also associated with inferior OS, while Vp3-4 showed an adverse trend for PFS. The final multivariable Cox model is presented in Table 5.

Table 5
Final multivariable Cox model for OS and PFS

Variable	Adjusted OS HR (95% CI); p	Adjusted PFS HR (95% CI); p
NLR ≥ 3.6	1.73 (1.13–2.66); 0.012	1.50 (1.05–2.14); 0.025
ECOG PS ≥ 1	1.30 (0.87–1.94); 0.201	0.90 (0.65–1.26); 0.548
ALBI grade 2–3	0.95 (0.65–1.40); 0.809	1.06 (0.78–1.44); 0.717
AFP ≥ 400 ng/mL	1.50 (1.01–2.22); 0.043	1.15 (0.84–1.59); 0.373
Vp3–4	1.54 (1.05–2.25); 0.028	1.34 (0.98–1.82); 0.069

Complete-case n = 210 for OS and PFS in the final model; death events = 116 and progression/death events = 168.

Kaplan-Meier curves of OS and PFS according to the NLR cutoff of 3.6 are shown in Fig. 1.

3.6. Restricted cubic spline analysis and bootstrap internal validation

Adjusted restricted cubic spline Cox analysis did not show a clear non-linear association between continuous NLR and OS or PFS. For OS, n/events = 210/116, the overall p-value was 0.335, and the non-

linearity p-value was 0.931. For PFS, n/events = 210/168, the overall p-value was 0.026, and the non-linearity p-value was 0.205.

In 500 bootstrap resamples of the selected NLR ≥ 3.6 model, the direction of the NLR effect remained consistently adverse. For OS, the observed aHR was 1.73 (95% CI, 1.13–2.66; p = 0.012), the bootstrap median HR was 1.72 (1.16–2.81), and 66.2% of bootstrap samples had p < 0.05. For PFS, the observed aHR was 1.50 (95% CI, 1.05–2.14; p = 0.025), the bootstrap median HR was 1.50 (1.03–2.18), and 62.0% of bootstrap samples had p < 0.05. The optimism-corrected C-indices were 0.599 for OS and 0.548 for PFS.

4. Discussion

This study supports the prognostic value of pretreatment NLR in patients with HCC treated with atezolizumab plus bevacizumab. The analysis was designed to avoid relying on a single unadjusted cutoff. Candidate clinical predictors were first screened, then NLR thresholds were examined in Kaplan-Meier analysis and in several Cox models. Through this process, NLR ≥ 3.6 emerged as a pragmatic threshold: it separated OS and PFS, retained significance in the selected multivariable model, and preserved an interpretable high-risk subgroup.

The biological plausibility of NLR as a prognostic marker is consistent with prior evidence. Neutrophils may contribute to tumor progression through angiogenesis, invasion, metastasis, and immunosuppression, whereas lymphocytes are central to antitumor immune activity [12–14]. A higher NLR may therefore reflect a pro-tumor inflammatory state and reduced antitumor immune reserve. International studies of atezolizumab plus bevacizumab in HCC have used NLR thresholds around 3 to 5 and generally show worse outcomes in patients with higher NLR [15, 16]. The selected threshold of 3.6 falls within this range. Importantly, while most pivotal trials and Western cohorts primarily involve hepatitis C or non-viral etiologies, our findings extend the prognostic utility of NLR ≥ 3.6 specifically to a Southeast Asian real-world population heavily dominated by chronic HBV infection, where background hepatic inflammation is uniquely active.

The selected threshold should be interpreted cautiously. RCS analysis did not identify a statistically clear non-linear dose-response pattern, suggesting that 3.6 should not be viewed as a biological inflection point. Rather, it is a risk-stratification threshold supported by its performance in the selected model. Bootstrap analysis showed a consistent adverse direction of effect, but the optimism-corrected C-indices were modest. Thus, NLR can support clinical risk stratification but does not constitute a standalone prediction model.

Baseline comparisons showed that patients with NLR ≥ 3.6 had poorer performance status and were more often classified as IMbrave150-OUT. These patterns suggest that NLR may capture a combination of systemic inflammation, liver reserve, tumor biology, and ability to continue therapy. This is particularly meaningful in real-world practice, as high NLR effectively flags fragile patients—such as those with borderline hepatic function or ECOG PS ≥ 1—who face a heightened risk of early treatment failure and

intolerance. The lower DCR and lower use of post-progression regorafenib in the high-NLR group further support the interpretation that NLR reflects vulnerability beyond a simple blood count ratio.

This study has limitations. First, its retrospective single-center design may introduce selection bias, information bias, and residual confounding. Second, cutoff selection was performed within the same dataset, so data dependency remains possible. Third, PFS and response evaluation in routine practice may be influenced by imaging intervals and clinical decision-making. Fourth, due to the retrospective nature of real-world data, explicit details on maximum tumor size and tumor multifocality were unavailable in a small subset of patients (n = 21) with diffuse or poorly demarcated lesions, leading to their exclusion from secondary subgroup analyses involving tumor burden. Fifth, other inflammatory markers such as CRP, PLR, SII, or DCP were not comprehensively available. External validation in independent cohorts is required before broad clinical application.

5. Conclusions

In real-world clinical practice, baseline NLR serves as an accessible prognostic biomarker for patients with advanced HCC receiving atezolizumab plus bevacizumab. An NLR threshold of ≥ 3.6 effectively stratifies both OS and PFS, offering an independent, clinically interpretable marker for identifying high-risk individuals—particularly those with poorer performance status or beyond trial eligibility criteria. While internal validation supports the consistency of this threshold, external validation in prospective, independent cohorts is warranted to confirm its utility for personalized patient management.

Abbreviations

UTBMTBG/HCC	Ung thư biểu mô tế bào gan / Hepatocellular carcinoma
Atezo/Bev	Atezolizumab plus bevacizumab
NLR	Neutrophil-to-lymphocyte ratio
OS	Overall survival
PFS	Progression-free survival
ORR	Objective response rate
DCR	Disease control rate
AFP	Alpha-fetoprotein
HBV	Hepatitis B virus
HCV	Hepatitis C virus
PVTT	Portal vein tumor thrombosis
BCLC	Barcelona Clinic Liver Cancer
ALBI	Albumin-bilirubin grade
ECOG PS	Eastern Cooperative Oncology Group performance status

Declarations

Author Contributions

Conceptualization, T.T.H.N., T.Y.L. and V.Q.L.; methodology, T.T.H.N., T.A.P. and H.T.V.; formal analysis, T.T.H.N., X.D.N. and Q.H.D.; investigation, T.A.P., H.T.V., Q.H.C., T.Y.L., X.D.N., H.A.N. and Q.H.D.; data curation, T.A.P., X.D.N., H.A.N. and Q.H.D.; writing—original draft preparation, T.A.P. and T.T.H.N.; writing—review and editing, T.T.H.N., T.Y.L., H.T.V., Q.H.C. and V.Q.L.; visualization, T.T.H.N., X.D.N. and Q.H.D.; supervision, T.Y.L. and V.Q.L. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Ethics Committee of Hanoi Medical University (Approval No. 452/QĐ-ĐHY, 20 February 2025).

Informed Consent Statement

The requirement for informed consent was waived due to the retrospective design of the study and the use of de-identified data. This waiver should be confirmed according to the final ethics approval document.

Data Availability Statement

The datasets generated during and/or analyzed during the current study are not publicly available due to patient privacy and institutional regulations but are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

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Figures

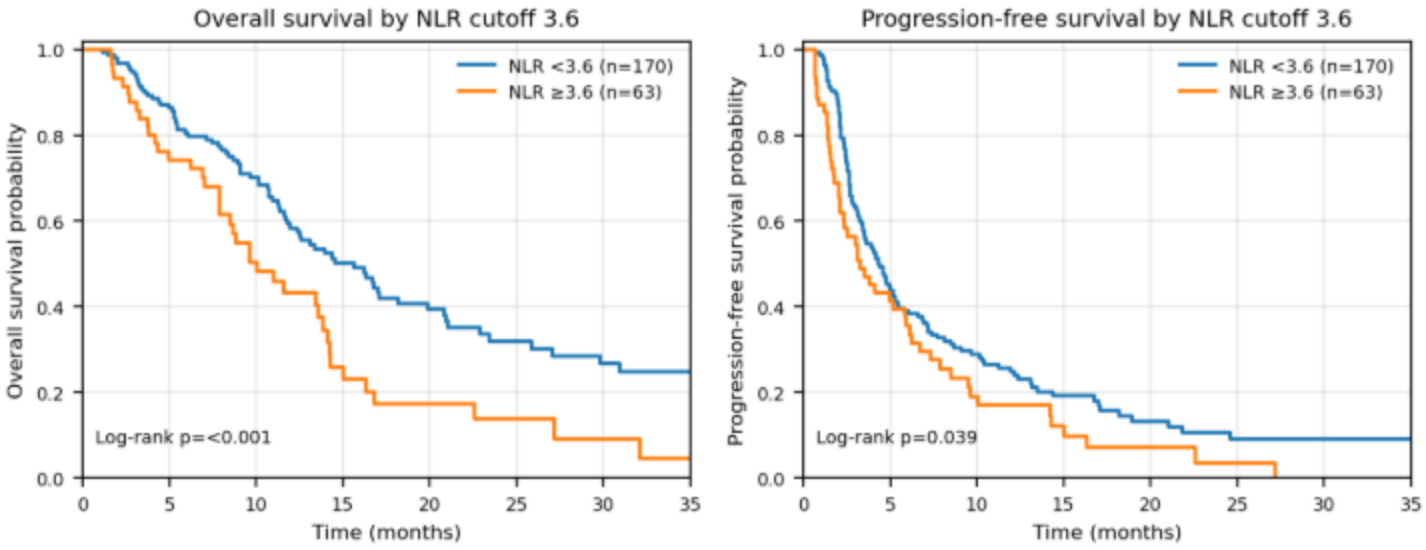


Figure 1

Kaplan-Meier curves of OS and PFS according to NLR cutoff 3.6

Supplementary Files

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- SupplementaryTableS123.docx