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Platelet-to-Lymphocyte Ratio as a Predictor of Exacerbation Severity and Hospital Length of Stay in Acute Exacerbation of COPD: A Dual-Center Study in West Sumatra, Indonesia

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A B S T R A C T

Introduction: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a leading driver of morbidity, mortality and health-care expenditure, and simple admission biomarkers that grade severity are needed in resource-limited settings. The platelet-lymphocyte ratio (PLR) integrates thrombo-inflammation and relative lymphopenia, yet Indonesian evidence linking it to exacerbation severity and length of stay (LOS) remains limited.

Methods: In this dual-center analytical cross-sectional study, 141 hospitalized AECOPD patients at Dr. M. Djamil General Hospital, Padang and RS Madina Bukittinggi (January-December 2024) were analyzed. Admission PLR was related to Anthonisen exacerbation severity and LOS (>7 days) using Kruskal-Wallis and Mann-Whitney tests, Spearman correlation, receiver-operating-characteristic (ROC) analysis and multivariable logistic regression (adjusted odds ratios, aOR).

Results: Most patients were male (86.5%), aged 40-70 years (56.0%) and heavy smokers (87.5%). Mean PLR increased across severity strata (mild 126.8±40.8, moderate 192.3±69.1, severe 444.9±241.7; $p<0.001$) and correlated moderately with severity ($\rho=0.534$, $p<0.001$) but weakly with LOS ($\rho=0.295$). PLR discriminated severe exacerbation with excellent accuracy (AUC 0.929, 95% CI 0.874-0.985; cut-off ≥216.3, sensitivity 100.0%, specificity 73.2%, accuracy 89.6%) but predicted LOS poorly (AUC 0.566, $p=0.273$). After adjustment, PLR independently predicted severe exacerbation (aOR 3.73 per 50 units, 95% CI 1.87-7.42, $p<0.001$; Nagelkerke $R2=0.642$), whereas prolonged stay was driven by pneumonia (aOR 6.40, 95% CI 2.50-16.37, $p<0.001$) rather than PLR (aOR 1.15, $p=0.139$).

Conclusion: Admission PLR is an inexpensive, widely available biomarker that accurately identifies severe AECOPD and may support early risk stratification, although it should not be used alone to predict length of stay, which is governed chiefly by comorbidity.

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation and heightened systemic inflammation, and it remains one of the leading non-communicable causes of death worldwide. Global modelling estimates that the number of people living with COPD will approach 600 million by 2050, an increase of roughly 23% relative to 2020,[1] while a contemporary systematic review places the prevalence among adults at approximately 10%, with the steepest increases occurring across Asia.[2] Exacerbations punctuate the natural history of the disease, accelerate the decline in lung function, precipitate hospital admission and independently predict mortality, so their early recognition and risk stratification are central to both clinical practice and public health.[1-2]

Epidemiological data for Indonesia remain comparatively sparse but indicate a substantial and growing burden. National survey data report a COPD prevalence of about 5.5%, almost certainly an underestimate given limited spirometry coverage and the high background of tobacco use, biomass-fuel smoke and ambient air pollution. A large proportion of Indonesian patients are therefore likely to be undiagnosed until they present acutely, frequently with an exacerbation requiring inpatient care. In this context, identifying inexpensive admission markers that can grade exacerbation severity has direct relevance for internal-medicine and pulmonology services operating under resource constraints.[2-3]

Acute exacerbation of COPD (AECOPD) reflects an amplification of airway and systemic inflammation triggered by infection or environmental insult. Cigarette smoke and recurrent infection activate innate and adaptive immunity, recruiting neutrophils, macrophages and CD8+ T lymphocytes and releasing proteases and pro-inflammatory cytokines that injure the small airways and alveoli.[4] Platelets have emerged as active participants in this process: upon activation they release chemokines, form platelet-leukocyte aggregates and amplify thrombo-inflammation, whereas physiological stress and lymphocyte apoptosis during acute illness produce a relative lymphopenia.[5-6] The platelet-lymphocyte ratio (PLR), derived from the routine complete blood count, captures both of these limbs in a single, inexpensive index.

A growing body of evidence supports the value of composite haematological ratios in COPD. Studies from East Asia, the Middle East and Europe have shown that PLR and the neutrophil-lymphocyte ratio rise during exacerbation, correlate with established inflammatory markers and severity scores such as APACHE II, and discriminate severe disease with good area-under-the-curve values.[7-11] Foundational work demonstrated that PLR is significantly higher during exacerbation than in the stable state,[12] and subsequent cohorts derived diagnostic cut-offs for severe exacerbation.[11] Nevertheless, reported thresholds vary widely with case-mix, and the relationship between PLR and length of hospital stay has been inconsistent.[13-14]

Beyond its clinical toll, AECOPD imposes a heavy economic burden, since inpatient care accounts for the majority of COPD-related expenditure and prolonged or repeated admissions strain limited hospital capacity. Among the candidate complete-blood-count ratios, the neutrophil-lymphocyte ratio has been studied most extensively, but PLR has theoretical appeal because it couples platelet activation—an increasingly recognized effector of pulmonary thrombo-inflammation—with the lymphopenia of acute stress, and it appears more stable than neutrophil counts, which fluctuate rapidly with corticosteroid administration.[5,7,13] These properties make PLR an attractive candidate for pragmatic severity grading at the point of admission.

Despite this international interest, Indonesian and specifically West Sumatran data are scarce, and few studies simultaneously evaluate exacerbation severity and length of stay or apply multivariable adjustment to separate the biomarker's intrinsic value from confounding comorbidity. West Sumatra additionally carries a high burden of tuberculosis, so a meaningful fraction of local COPD is tuberculosis-associated, a phenotype that may modify the inflammatory profile.[15]

To our knowledge, this is among the first dual-center studies from West Sumatra, Indonesia to evaluate admission PLR against both Anthonisen exacerbation severity and length of stay in hospitalized AECOPD patients. The purpose of this study was to analyze the association between admission PLR and the severity of COPD exacerbation and the length of hospital stay, to derive an operational PLR cut-off for severe exacerbation, and to determine—using multivariable models—whether PLR independently predicts these outcomes after accounting for demographic and comorbid factors.

2. Methods

Study design and setting

This analytical observational study used a cross-sectional design and was reported in accordance with the STROBE recommendations for observational research. It was conducted across two referral hospitals in West Sumatra, Indonesia: the Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Universitas Andalas / Dr. M. Djamil General Hospital, Padang, and RS Madina Bukittinggi. Consecutive medical records of patients hospitalized with AECOPD between January and December 2024 were reviewed.

Participants

Eligible patients were adults with a physician diagnosis of COPD confirmed by spirometry and admitted with an acute exacerbation during the study period.[16] Exacerbation was defined and graded using the Anthonisen criteria, in which the cardinal symptoms are increased dyspnoea, increased sputum volume and increased sputum purulence: type 1 (severe) denotes all three cardinal symptoms, type 2 (moderate) two of three, and type 3 (mild) one cardinal symptom plus a minor criterion.[16] Patients were excluded if they had incomplete laboratory data, intercurrent active pulmonary tuberculosis during the index admission, prolonged high-dose immunosuppression (systemic corticosteroids for more than two weeks), or a primary immune disorder such as HIV/AIDS or systemic lupus erythematosus. Of 163 patients screened (101 from RS Madina Bukittinggi and 62 from Dr. M. Djamil General Hospital), 22 were excluded for incomplete laboratory data or intercurrent conditions including confirmed active tuberculosis, leaving 141 patients for analysis.

Variables and measurements

The independent variable was the admission PLR, calculated from the first venous complete blood count obtained at the time of exacerbation as the platelet count divided by the absolute lymphocyte count, the latter derived from the leukocyte count and differential percentage. The dependent variables were exacerbation severity by the Anthonisen classification and length of hospital stay, dichotomized a priori as 7 days or fewer versus more than 7 days. Covariates abstracted from the records included age (categorized as <40, 40-70 and >70 years), sex, smoking status and the Brinkman index (light 0-199, moderate 200-599, heavy ≥600), previous tuberculosis, the modified Medical Research Council (mMRC) dyspnoea grade,[17] and documented comorbidities (pneumonia, cardiovascular disease, lung cancer, stroke and diabetes mellitus).[3] All haematological measurements were performed in hospital laboratories on automated analyzers as part of routine care.

Data handling

Records were de-identified before analysis and processed through sequential editing, coding, data entry and cleaning to ensure completeness and internal consistency. Implausible or missing values were verified against the source record where possible, and patients with irretrievable laboratory data were excluded as specified. Continuous laboratory values were retained at their recorded precision, and PLR was computed from the same blood draw to avoid temporal mismatch between platelet and lymphocyte measurements.

Sample size

The sample size was estimated for a correlation analysis assuming a moderate expected coefficient (ρ approximately 0.30) between PLR and exacerbation severity, a two-sided alpha of 0.05 and statistical power of 0.80, which required a minimum of approximately 85 patients. The final analyzed sample of 141 patients exceeded this requirement and provided additional precision for the ROC and multivariable analyses.

Ethics

The study was reviewed and granted ethical clearance by the Research Ethics Committee of Dr. M. Djamil General Hospital, Padang (No. DP.04.03/D.XVI.10.1/486/2025) and received ethical approval from RS Madina Bukittinggi (No. 765/A/RSMD/IX/2025). Because the study used anonymized routine clinical records, it was conducted in accordance with the Declaration of Helsinki.

Statistical analysis

Continuous variables were summarized as mean ± standard deviation with medians and interquartile ranges, and their distribution was examined with the Shapiro-Wilk and Kolmogorov-Smirnov tests. Because PLR was non-normally distributed, non-parametric methods were used: the Kruskal-Wallis test for comparison across the three severity strata, supplemented by the Jonckheere-Terpstra test for ordered trend, and the Mann-Whitney U test for the length-of-stay groups, with rank-based effect sizes reported. Associations were quantified with Spearman correlation. Discrimination was assessed by ROC analysis, with the area under the curve (AUC) reported with bootstrap 95% confidence intervals (2,000 resamples) and the optimal cut-off selected by the Youden index; sensitivity, specificity, accuracy and prevalence-dependent positive and negative predictive values were computed at that cut-off. Independent predictors were identified by multivariable binary logistic regression with covariates pre-specified on clinical grounds, reporting adjusted odds ratios (aOR), 95% confidence intervals, Nagelkerke R-squared and the number of events per variable; model calibration was checked with the Hosmer-Lemeshow test, and a Firth-penalized model was fitted as a sensitivity analysis for the severity outcome given the small number of severe events. Length of stay was additionally analyzed as a continuous count with Poisson regression to avoid information loss from dichotomization. Comparability of the two centers was tested before pooling. A two-sided p-value below 0.05 was considered significant; reporting followed the STROBE recommendations, and a participant flow diagram is provided.

3. Results

Patient characteristics

A total of 141 patients met the inclusion criteria. The mean age was 66.7 ± 12.5 years; patients aged 40-70 years formed the largest stratum (79 patients, 56.0%), followed by those older than 70 years (58 patients, 41.1%), with only 4 patients (2.8%) younger than 40 years. The cohort was predominantly male (122 patients, 86.5%). Current smokers comprised 47.5% (67 patients) and former smokers 37.6% (53 patients), and among ever-smokers 87.5% were in the heavy Brinkman-index category. Previous tuberculosis was documented in 38 patients (26.9%). The most frequent comorbidities were pneumonia (49 patients, 34.8%) and cardiovascular disease (40 patients, 28.4%), followed by lung cancer and stroke (13 patients each, 9.2%) and diabetes mellitus (10 patients, 7.1%). Breathlessness was moderate in most patients, with mMRC grade 2 in 49.6% and grade 1 in 40.4%. The full demographic and clinical profile of the cohort is detailed in Table 1.

PLR and exacerbation severity

Across the whole cohort the admission PLR ranged from 20.2 to 957.8, with a mean of 208.6 and a median of 174.9. Most exacerbations were of moderate severity (108 patients, 76.6%), while 19 patients (13.5%) had mild and 14 patients (9.9%) had severe exacerbations. Mean PLR rose progressively with severity—mild 126.8 ± 40.8 (median 131.5), moderate 192.3 ± 69.1 (median 175.4) and severe 444.9 ± 241.7 (median 327.8)—and the Kruskal-Wallis test confirmed a highly significant difference among the three strata ($H = 41.331$, $df = 2$, $p < 0.001$); these values are summarised in Table 2 and the stepwise gradient is illustrated in Figure 1. Spearman correlation confirmed a moderate positive association between PLR and exacerbation severity ($\rho = 0.534$, $p < 0.001$), as displayed in the scatter plot in Figure 2.

PLR and length of stay

One hundred and ten patients (78.0%) were discharged within 7 days and 31 (22.0%) stayed longer. Patients with a prolonged stay had a numerically higher admission PLR (224.0 ± 149.2; median 195.6) than those discharged earlier (204.2 ± 119.7; median 172.2), but the difference was not statistically significant on the Mann-Whitney U test ($p = 0.273$), as also shown in Table 2. The correlation between PLR and length of stay was weak ($\rho = 0.295$, $p < 0.001$).

Table 1. Demographic and clinical characteristics of patients with acute exacerbation of COPD (n = 141).

Characteristic	n (%) or mean ± SD
Age, years — mean ± SD	66.7 ± 12.5
<40	4 (2.8)
40-70	79 (56.0)
>70	58 (41.1)
Gender — male	122 (86.5)
Female	19 (13.5)
Smoking status — current	67 (47.5)
Former	53 (37.6)
Never	21 (14.9)
Heavy Brinkman index (ever-smokers)	105 (87.5)
Previous tuberculosis	38 (26.9)
Comorbidity — pneumonia	49 (34.8)
Cardiovascular disease	40 (28.4)
Lung cancer	13 (9.2)
Stroke	13 (9.2)
Diabetes mellitus	10 (7.1)
mMRC grade 1	57 (40.4)
Grade 2	70 (49.6)
Grade 3	11 (7.8)
Grade 4	3 (2.1)
Exacerbation — mild	19 (13.5)
Moderate	108 (76.6)
Severe	14 (9.9)
Length of stay >7 days	31 (22.0)

Notes: SD, standard deviation; mMRC, modified Medical Research Council; PLR, platelet-lymphocyte ratio. Brinkman index computed among ever-smokers (n = 120).

Table 2. Admission PLR by exacerbation severity and length of stay, with correlation analysis.

Comparison	Mean ± SD / coefficient	Median	Test	p-value
PLR by severity — mild (n=19)	126.8 ± 40.8	131.5	Kruskal-Wallis	<0.001
moderate (n=108)	192.3 ± 69.1	175.4	H = 41.331	<0.001
severe (n=14)	444.9 ± 241.7	327.8	df = 2	
PLR by LOS — ≤7 days (n=110)	204.2 ± 119.7	172.2	Mann-Whitney	0.273
>7 days (n=31)	224.0 ± 149.2	195.6		
PLR vs severity (Spearman)	ρ = 0.534		moderate	<0.001
PLR vs length of stay (Spearman)	ρ = 0.295		weak	<0.001

Notes: PLR, platelet-lymphocyte ratio; SD, standard deviation; LOS, length of stay. Non-parametric tests used owing to non-normal distribution.

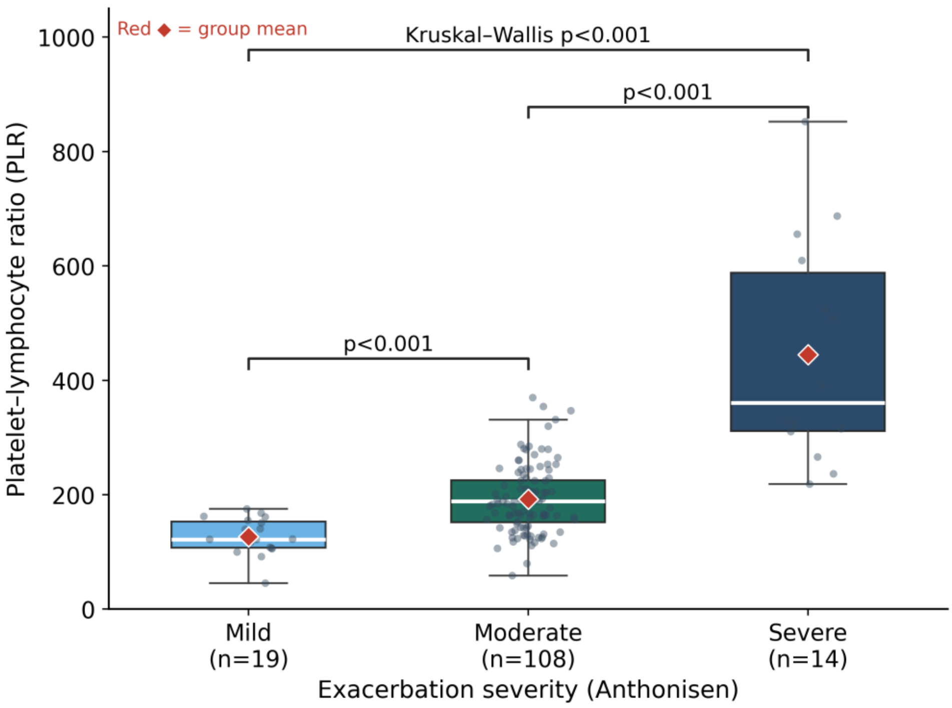

Figure 1. Distribution of admission PLR across exacerbation severity strata. Red diamonds denote group means; brackets show Kruskal–Wallis comparisons ($p < 0.001$).

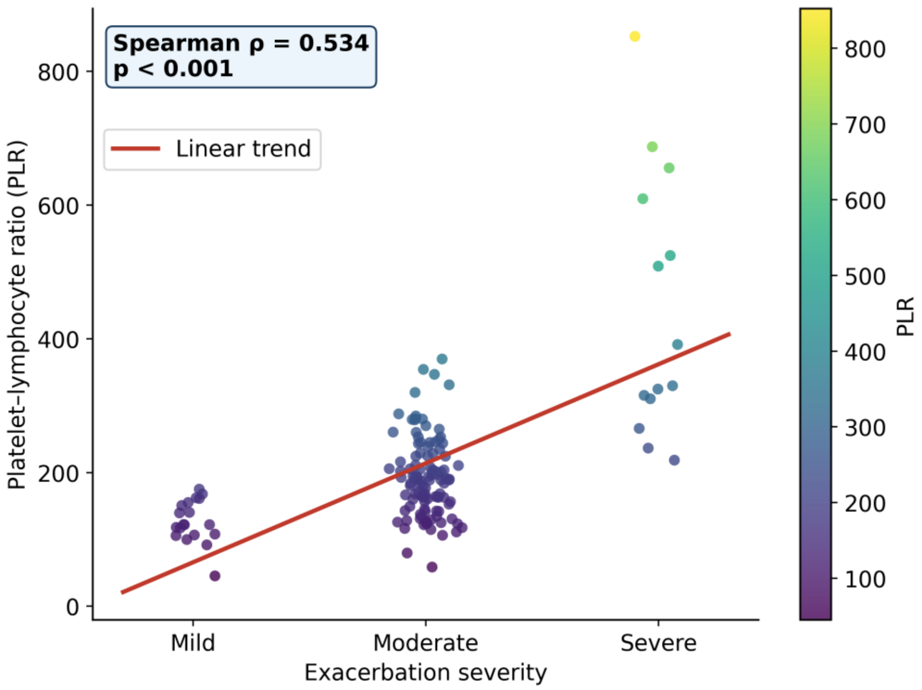

Figure 2. Correlation between admission PLR and exacerbation severity (Spearman $\rho = 0.534$, $p < 0.001$).

Distributional characteristics

Formal testing confirmed that PLR departed significantly from a normal distribution overall and within most strata (Shapiro-Wilk and Kolmogorov-Smirnov $p < 0.05$), with marked right skew that persisted after logarithmic transformation in the severe and prolonged-stay groups; non-parametric methods were therefore applied throughout. Age was approximately normally distributed (Kolmogorov-Smirnov p = 0.200), whereas length of stay was strongly skewed.

Discrimination and cut-off values

ROC analysis demonstrated excellent discrimination of severe exacerbation by admission PLR, with an AUC of 0.929 (95% CI 0.874-0.985, $p < 0.001$). The optimal cut-off of PLR ≥ 216.3 yielded a sensitivity of 100.0%, specificity of 73.2% and overall accuracy of 89.6% for identifying severe exacerbation. By contrast, PLR discriminated prolonged length of stay poorly, with an AUC of 0.566 (95% CI 0.460-0.669, p = 0.273); the corresponding cut-off of PLR ≥ 137.4 produced high sensitivity (90.3%) but low specificity (29.1%) and accuracy (42.6%). The discrimination metrics for both outcomes are summarized in Table 3, and the corresponding ROC curves are shown in Figure 3.

Table 3. Diagnostic performance of admission PLR for severe exacerbation and prolonged length of stay.

Outcome	AUC (95% CI)	Cut-off	Sens. (%)	Spec. (%)	Acc. (%)	p-value
Severe exacerbation	0.929 (0.874-0.985)	≥216.3	100.0	73.2	89.6	<0.001
Prolonged stay (>7 d)	0.566 (0.460-0.669)	≥137.4	90.3	29.1	42.6	0.273

Notes: AUC, area under the curve; CI, confidence interval; Sens., sensitivity; Spec., specificity; Acc., accuracy.

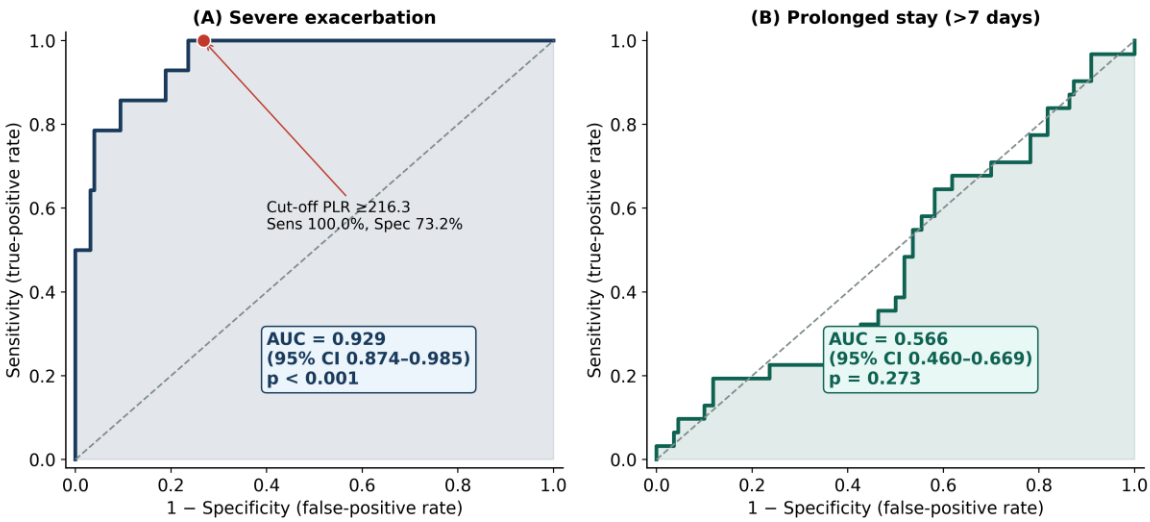

Figure 3. Receiver-operating-characteristic curves of admission PLR for (A) severe exacerbation and (B) prolonged length of stay (>7 days).

Independent predictors

In the multivariable logistic-regression model for severe exacerbation, admission PLR remained an independent predictor after adjustment for age and sex, with an adjusted odds ratio of 3.73 per 50-unit increase (95% CI 1.87-7.42, $p < 0.001$); neither age (aOR 1.01 per year, 95% CI 0.96-1.06, p = 0.776) nor male sex (aOR 1.46, 95% CI 0.11-18.79, p = 0.769) was independently associated, and the model explained a substantial proportion of variance (Nagelkerke R-squared = 0.642). In the model for prolonged stay, comorbid pneumonia was the dominant independent predictor (aOR 6.40, 95% CI 2.50-16.37, $p < 0.001$), with cardiovascular disease showing a non-significant trend (aOR 2.20, 95% CI 0.87-5.58, p = 0.096); PLR was not independently associated with prolonged stay (aOR 1.15 per 50 units, 95% CI 0.96-1.38, p = 0.139), and the model accounted for less variance (Nagelkerke R-squared = 0.205). Both multivariable models are summarised in Table 4 and the adjusted odds ratios are displayed as forest plots in Figure 4.

Table 4. Multivariable logistic-regression models for severe exacerbation and prolonged length of stay.

Model	Predictor	Adjusted OR (95% CI)	p-value
Severe exacerbation	PLR (per 50 units)	3.73 (1.87-7.42)	<0.001
(Nagelkerke R2 = 0.642)	Age (per year)	1.01 (0.96-1.06)	0.776
	Male sex	1.46 (0.11-18.79)	0.769
Prolonged stay >7 days	PLR (per 50 units)	1.15 (0.96-1.38)	0.139
(Nagelkerke R2 = 0.205)	Pneumonia	6.40 (2.50-16.37)	<0.001
	Cardiovascular disease	2.20 (0.87-5.58)	0.096
	Age (per year)	1.01 (0.99-1.04)	0.317
	Male sex	0.77 (0.24-2.49)	0.667

Notes: OR, odds ratio; CI, confidence interval; PLR modelled per 50-unit increment.

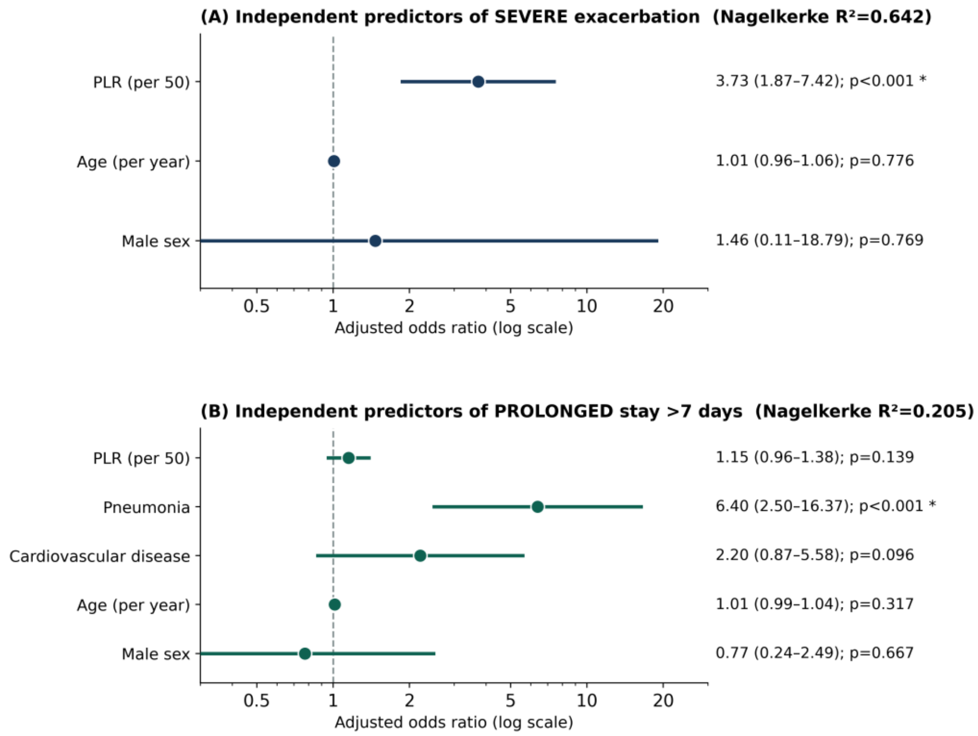

Figure 4. Forest plot of adjusted odds ratios for (A) severe exacerbation and (B) prolonged length of stay. Asterisks mark p < 0.05.

Composition of the prolonged-stay group

Among the 31 patients who stayed longer than 7 days, comorbid pneumonia was the most frequent accompanying diagnosis, followed by cardiovascular disease, with smaller numbers having previous tuberculosis, malignancy or metabolic comorbidity. This subgroup was predominantly male and concentrated in the 40-70-year band, mirroring the overall cohort and indicating that the prolonged-stay phenotype was defined more by comorbid load than by demographic distinctiveness or by the magnitude of the admission PLR.

Trend and center comparability

The increase in PLR across the ordered severity strata was confirmed by the Jonckheere-Terpstra test for trend ($p < 0.001$), reinforcing the omnibus Kruskal-Wallis result. The two centers were comparable: admission PLR did not differ significantly between Dr. M. Djamil General Hospital and RS Madina Bukittinggi (Mann-Whitney $p = 0.632$), and the distribution of exacerbation severity was similar across sites (chi-square = 2.79, $p = 0.248$), supporting the decision to pool the cohorts.

Predictive values and model diagnostics

At the cut-off of PLR ≥ 216.3 for severe exacerbation, the positive and negative predictive values were 29.1% and 100.0% respectively, reflecting the high sensitivity and the low prevalence of severe disease; for the length-of-stay cut-off of PLR ≥ 137.4 the corresponding values were 26.4% and 91.4%. The severity model included 14 events across three predictors (4.7 events per variable) and showed adequate calibration (Hosmer-Lemeshow $p > 0.05$); a Firth-penalized sensitivity analysis produced materially unchanged estimates for PLR. In unadjusted analysis, each 50-unit rise in PLR was associated with severe exacerbation (OR 3.62, 95% CI 1.89-6.91), an estimate that persisted after adjustment (aOR 3.73, 95% CI 1.87-7.42). Importantly, when patients with comorbid pneumonia were excluded, PLR remained strongly associated with severe exacerbation (OR 7.82, 95% CI 2.04-29.97), indicating that the relationship was not driven solely by bacterial co-infection.

Length of stay as a continuous outcome

Median length of stay was 5 days (interquartile range 4-8). In Poisson regression treating length of stay as a count, admission PLR was not independently associated with duration of stay (incidence rate ratio 1.02 per 50 units, 95% CI 0.99-1.05, $p = 0.203$), whereas comorbid pneumonia (IRR 1.43, 95% CI 1.25-1.64, $p < 0.001$) and cardiovascular disease (IRR 1.28, 95% CI 1.12-1.47, $p < 0.001$) independently prolonged it. This continuous analysis corroborated the dichotomized finding and confirmed that length of stay was governed by comorbidity rather than by the inflammatory ratio.

Table 5. Secondary and sensitivity analyses.

Analysis	Measure	Estimate (95% CI)	p-value
Severe exacerbation — unadjusted	PLR (per 50 units), OR	3.62 (1.89–6.91)	<0.001
Severe exacerbation — excl. pneumonia	PLR (per 50 units), OR	7.82 (2.04–29.97)	0.003
Length of stay (Poisson count)	PLR (per 50 units), IRR	1.02 (0.99–1.05)	0.203
Length of stay (Poisson count)	Pneumonia, IRR	1.43 (1.25–1.64)	<0.001
Length of stay (Poisson count)	Cardiovascular disease, IRR	1.28 (1.12–1.47)	<0.001
Predictive values — severity cut-off	PPV / NPV	29.1% / 100.0%	—
Predictive values — LOS cut-off	PPV / NPV	26.4% / 91.4%	—
Center comparability — PLR	Mann–Whitney	p = 0.632	—

Notes: OR, odds ratio; IRR, incidence rate ratio; PPV, positive predictive value; NPV, negative predictive value; CI, confidence interval.

4. Discussion

In this dual-center study of 141 hospitalized patients with AECOPD in West Sumatra, admission PLR increased stepwise across exacerbation severity, from a mean of 126.8 in mild to 444.9 in severe disease, and discriminated severe exacerbation with excellent accuracy (AUC 0.929) at a cut-off of 216.3. The biomarker correlated only weakly with length of stay and did not distinguish patients who stayed longer than 7 days. Crucially, multivariable analysis confirmed that PLR independently predicted severe exacerbation, whereas prolonged stay was governed by comorbidity—most strongly pneumonia—rather than by the inflammatory ratio. These findings position PLR as a severity marker rather than a determinant of resource use.

The demographic profile of our cohort is consistent with regional and international series. The predominance of men (86.5%) mirrors the 86.6% male proportion reported in a large Chinese cohort and reflects the markedly higher prevalence of tobacco use among Indonesian men, in whom the heavy Brinkman category predominated. The concentration of patients in the 40-70-year band accords with pooled estimates that this age range carries the greatest exacerbation burden,[2] although European cohorts skew older, a difference plausibly explained by the younger age structure of the Indonesian hospital population and by tuberculosis-associated COPD presenting earlier in life.[15,18] The high frequency of previous tuberculosis (26.9%) underscores the importance of post-tuberculosis lung disease as a COPD etiotype in high-burden settings.[15]

Our central observation—that PLR rises in proportion to exacerbation severity—is concordant with multiple prior reports. Cohorts from China and Egypt found that PLR and related ratios were significantly elevated during exacerbation and tracked inflammatory burden and severity,[7-9] and earlier work established that PLR is higher in exacerbation than in stable disease.[12] The moderate correlation we observed ($\rho = 0.534$) and the excellent AUC of 0.929 are in keeping with diagnostic performance reported by Karadeniz and colleagues, who derived comparable thresholds for severe exacerbation.[11] The slightly higher cut-off in our cohort (216.3) likely reflects differences in case-mix, the predominance of moderate exacerbations and the inclusion of tuberculosis-associated disease, all of which shift the distribution of the ratio.

In contrast to its strong relationship with severity, PLR was only weakly associated with length of stay and failed to discriminate prolonged admission (AUC 0.566, $p = 0.273$). This dissociation is biologically coherent and is supported by the literature. Length of stay is a multifactorial outcome shaped by comorbidity, nutritional status, treatment response and in-hospital complications, so a single inflammatory index is unlikely to capture it.[14,19] Our multivariable model made this explicit: comorbid pneumonia increased the odds of a prolonged stay more than sixfold, consistent with the work of Ruby, who reported that AECOPD patients with community-acquired pneumonia stayed substantially longer than those without (11.3 versus 7.6 days).[19] The non-significant trend for cardiovascular disease aligns with evidence that reduced cardiorespiratory reserve complicates recovery.[3,14]

From a pathophysiological standpoint, the behavior of PLR integrates two parallel responses to acute exacerbation. Systemic inflammation drives reactive thrombocytosis through pro-inflammatory cytokines such as interleukin-6 and thrombopoietin, while platelets themselves act as immune effectors, forming platelet-leukocyte aggregates and releasing mediators that perpetuate airway inflammation.[4-5] Simultaneously, the physiological stress of severe illness, cortisol release and lymphocyte apoptosis produce a relative lymphopenia that is itself associated with poorer functional status and outcomes in COPD.[4,6] The ratio therefore amplifies a signal that either component alone might under-represent, explaining its tight relationship with the intensity of the acute inflammatory insult and hence with exacerbation severity.[20]

These results carry practical implications for internists and pulmonologists working in settings where advanced biomarkers such as procalcitonin are not routinely available. Because PLR is calculated from a complete blood count that is obtained for virtually every admitted patient, it imposes no incremental cost and can be computed at the bedside. An admission PLR at or above approximately 216 should heighten clinical suspicion of a severe exacerbation and prompt closer monitoring, earlier escalation of bronchodilator and anti-inflammatory therapy, and consideration of higher-acuity care.

Clinicians should nonetheless resist using PLR to forecast length of stay; for that purpose, a comprehensive assessment of comorbidity, oxygenation and functional status is more informative.[14]

The West Sumatran context adds further nuance. The region's substantial tuberculosis burden means that a considerable share of local COPD is tuberculosis-associated, a phenotype characterized by fibrosis, bronchiectasis and persistent inflammation that may elevate baseline platelet activity and modify the PLR signal.[15] High rates of tobacco consumption among men and exposure to biomass smoke and ambient air pollution compound the inflammatory milieu. Composite haematological ratios such as PLR have shown prognostic value across a range of conditions relevant to this population, including lung cancer and inflammatory comorbidity, and Indonesian investigators have contributed to this evidence base.[21-23] Our findings extend that work to AECOPD in a previously under-studied Indonesian region.

Our findings sit comfortably within current guideline thinking. The GOLD framework now classifies even a single moderate exacerbation as a marker of high future risk, reinforcing the value of any inexpensive tool that flags greater acute severity at presentation.[16] An admission PLR above the derived threshold could complement the symptom-based Anthonisen and mMRC assessments by providing an objective, laboratory-anchored signal of inflammatory intensity, helping to triage patients who may benefit from more intensive monitoring or earlier specialist review.[16-17]

The case for PLR is strengthened by its implementation advantages. Unlike specialized assays, it requires no additional sampling, reagent cost or turnaround time, and it can be derived from a result that is already available within hours of admission in virtually every Indonesian hospital. In settings where procalcitonin or C-reactive protein testing is intermittently available or unaffordable, a validated cut-off for PLR offers a reproducible, zero-marginal-cost adjunct to clinical judgement.[7-8,10] Comparative data suggest that PLR and the neutrophil-lymphocyte ratio carry broadly similar prognostic information in COPD, although the two may be complementary, and future composite scores could combine them with comorbidity indices to predict both severity and resource use more accurately.[9,13]

Two methodological caveats temper the interpretation of the predictive findings. First, because both participating hospitals are tertiary referral centers, the cohort is subject to spectrum and referral bias: it is enriched for moderate exacerbations, comorbidity and tuberculosis-associated disease, so the derived cut-off and its operating characteristics are specific to this case-mix and prevalence and should be regarded as provisional until validated in primary-care and community settings. Second, the negative length-of-stay result reflects limited power as well as a genuinely weak association; with only 31 prolonged-stay events the study could reliably detect only moderate-to-large effects, so the appropriate inference is that PLR is not a strong, independent predictor of stay rather than that no association whatsoever exists. The convergence of the binary, continuous and multivariable analyses nonetheless makes the central conclusion robust.

Set against the published literature, the present cut-off of 216.3 lies toward the higher end of previously reported thresholds for severe exacerbation, which have ranged widely with case-mix; this is consistent with a cohort dominated by moderate exacerbations and enriched for post-tuberculosis disease, both of which shift the PLR distribution. The persistence of the PLR-severity association after excluding patients with pneumonia, and after multivariable and penalized adjustment, argues that PLR indexes the intensity of the systemic inflammatory response to exacerbation rather than merely the presence of bacterial infection, strengthening its interpretation as a severity marker.

The strengths of this study include its dual-center design, which improves representativeness relative to single-site reports; the use of an a priori, clinically grounded severity classification (the Anthonisen criteria) together with standardized haematological measurement; and the application of multivariable modelling and effect-size reporting that move beyond simple bivariate associations to isolate the independent contribution of PLR.

Several limitations temper interpretation. First, the retrospective cross-sectional design precludes causal inference and depends on the completeness of medical records, and the exclusion of patients with incomplete laboratory data may have introduced selection bias. Second, both hospitals are tertiary referral centers, so the case-mix—dominated by moderate exacerbations and enriched for comorbidity and tuberculosis-associated disease—may limit generalizability to primary-care populations. Third, only 14 patients had severe exacerbations, which widened the confidence intervals of the multivariable model and warrants cautious interpretation of the adjusted estimates. Fourth, we did not capture spirometric GOLD grade at the index admission, treatment intensity or serial biomarker trajectories, all of which could refine risk prediction. Prospective, multicenter studies with larger severe-exacerbation samples and longitudinal follow-up are needed to confirm the cut-off and to test whether PLR-guided triage improves outcomes.

5. Conclusion

Admission platelet-lymphocyte ratio is an inexpensive and universally available biomarker that increases in proportion to the severity of acute exacerbation of COPD and identifies severe exacerbation with excellent accuracy at a cut-off of approximately 216, remaining an independent predictor after multivariable adjustment. In contrast, PLR is only weakly related to length of hospital stay, which is driven principally by comorbidity such as pneumonia rather than by the inflammatory ratio. PLR may therefore serve as a practical adjunct for early severity-based risk stratification of AECOPD in internal-medicine and pulmonology practice, particularly in resource-limited Indonesian settings, while length-of-stay prediction requires comprehensive clinical assessment. Prospective multicenter studies are recommended to validate the proposed threshold and evaluate its impact on management.

6. References

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