

Incremental Value of Platelet-to-Hemoglobin Ratio beyond the R-Baux Score in Predicting Burn Mortality: Evidence from the Recovery Phase

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Introduction: Burn injuries trigger a systemic inflammatory response that increases mortality risk. The Revised Baux (R-Baux) score, a validated predictor of mortality at the time of admission, is determined by age, burn surface area, and the presence of inhalation injury. Nonetheless, it remains uncertain whether inflammatory indices derived from routine complete blood counts offer additional predictive value beyond this score.

Methods: This retrospective cohort study examined 238 burn patients admitted to a referral hospital in Surabaya, Indonesia, during the period from 2023 to 2024. Four inflammatory ratios derived from complete blood counts were assessed at three distinct phases of the clinical course: the initial phase (24-48 hours), the hyperinflammatory phase (3-7 days), and the recovery phase (beyond 7 days). For each phase, logistic regression models incorporating the R-Baux score alone were compared with each ratio using the likelihood-ratio test and a statistical test for comparing correlated receiver operating characteristic (ROC) curves.

Results: The overall mortality rate was 30.7%. The R-Baux score was a strong predictor of mortality on its own. Upon admission, none of the four ratios enhanced the predictive capability beyond that of the R-Baux score. During the hyperinflammatory phase, both the platelet-to-hemoglobin ratio (PHR) and neutrophil-to-lymphocyte ratio (NLR) contributed modestly yet significantly to prediction. In contrast, during the recovery phase, the PHR provided substantial incremental value, enhancing discrimination

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from good to excellent. The other ratios did not contribute value in any phase.

Conclusion: Inflammatory indices derived from complete blood count do not significantly surpass the R-Baux score at the time of admission. However, the PHR offers clinically valuable prognostic information, especially during the recovery phase of the clinical course.

Keywords: burns; platelet-to-hemoglobin ratio; R-Baux score; mortality prediction.

1. Introduction

Burn injuries are a serious trauma that significantly affects both individuals and public health systems. Clinically, burns may result in severe pain, extensive tissue damage, organ dysfunction, permanent disability, and often lead to mortality (1,2). Multiple factors, including delays in hospital admission, burn extent and depth, complications, underlying comorbidities, and the readiness of the healthcare system, influence patient outcomes (3,4).

Burns induce complex local and systemic responses. At the site of injury, inflammatory mediators initiate vasodilation and increased capillary permeability, resulting in significant fluid loss and subsequent hypovolemic shock (5). Systemically, burns lead to elevated neutrophil counts, platelet activation, and lymphocyte depletion, indicative of excessive inflammation and immune dysregulation. These processes increase vulnerability to infection, sepsis, and multiple organ failure. Concurrently, hypermetabolic and catabolic states impede wound healing and contribute to mortality (5,6).

Several clinical scoring systems are available to predict burn mortality using readily accessible admission variables. Among these, the Revised Baux (R-Baux) score is recognized as a reliable measure for predicting mortality across various burn populations. R-Baux score is calculated using the following formula: $R\text{-Baux score} = \text{age} + \text{percentage burn} + 17 \times (\text{inhalation injury, } 1 = \text{yes, } 0 = \text{no})$ (7). It is one of the most thoroughly validated tools for this purpose, consistently demonstrating high predictive accuracy across diverse burn populations (8). However, the R-

Baux score is fixed at admission and does not account for a patient's changing clinical trajectory. Other tools, such as the FLAMES score and inflammatory biomarkers like C-reactive protein and procalcitonin have also been investigated, though they may be impractical in resource-limited settings (9,10).

Integrating admission-based scores with supplementary laboratory markers has demonstrated potential. Combining inflammatory and coagulation parameters with clinical indices has been reported to improve early mortality prediction (11). This indicates that systemic inflammatory status may offer prognostic value beyond what is captured by admission variables. Simple hematological ratios derived from routine complete blood counts (CBC) such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), platelet-to-hemoglobin ratio (PHR), and systemic immune-inflammation index (SII), provide cost-effective, repeatable alternatives that can be measured serially throughout hospitalization, providing real-time insights into the progression of systemic inflammation (12–15). Several of these markers have individually shown associations with burn mortality (16,17). However, it remains untested whether any of these serially-measured indices offer predictive information beyond that provided by an established, validated admission score such as the R-Baux score, and at which stage of the clinical course such value might become apparent.

This study aimed to assess whether inflammatory indices derived from serial CBC, measured at three defined phases of the clinical course,

improve the prediction of in-hospital mortality beyond the R-Baux score in burn patients admitted to a tertiary referral hospital in Surabaya, Indonesia.

2. Materials and Methods

This retrospective cohort study examined secondary data from medical records of burn patients treated at the Burn Unit of Dr. Soetomo Hospital, Surabaya, Indonesia, between January 1, 2023, and December 31, 2024. The study employed total sampling. Inclusion criteria included acute burn injury and the availability of hematological data for at least the initial phase (24-48 hours) of hospitalization. Medical records that were incomplete or untraceable were excluded. Due to the requirement that hyperinflammatory-phase (3-7 days) and recovery-phase (more than 7 days) blood samples could only be obtained from patients whose clinical course extended sufficiently to reach those timepoints, the number of patients with data available for each subsequent phase was smaller than the full cohort: 238 patients had complete initial-phase data, 199 had hyperinflammatory-phase data, and 137 had recovery-phase data. All analyses used all patients with complete data for the relevant phase (available-case analysis).

Data collected included demographic characteristics (age, sex, comorbidities), clinical variables (burn extent, inhalation trauma), hematological parameters, and outcome (in-hospital mortality). Hematological data were obtained at three phases: initial (24-48 hours), hyperinflammatory (3-7 days), and recovery (more than 7 days). As later-phase data were only available for patients whose clinical course extended long enough for a corresponding blood draw, each phase-specific analysis used all patients with complete data for that phase: initial phase, $n=238$; hyperinflammatory phase, $n=198$; recovery phase, $n=137$.

Four inflammatory indices derived from hematology parameters were calculated: neutrophil-to-lymphocyte ratio (NLR), calculated by dividing the neutrophil count by the lymphocyte count; platelet-to-lymphocyte ratio

(PLR), obtained by dividing the platelet count by the lymphocyte count; platelet-to-hemoglobin ratio (PHR) derived by dividing the platelet count by the hemoglobin level in g/dL; and systemic immune-inflammation index (SII), which is calculated by multiplying neutrophil count by the platelet count and then dividing by the lymphocyte count. While the R-Baux score was calculated by summing the age in years, percentage total body surface area burned, and an additional 17 points if an inhalation injury was present (7).

Descriptive statistics were reported as medians with interquartile ranges due to the non-normal distribution of all continuous variables, as confirmed by the Shapiro-Wilk test. Group comparisons were conducted using the Mann-Whitney U test. For each biomarker and phase, receiver operating characteristic (ROC) curve analysis was employed to estimate the area under the curve (AUC) for predicting in-hospital mortality. The direction of discrimination indicating whether higher or lower values corresponded to greater risk, was determined a priori from the observed median differences between survivors and non-survivors and was applied consistently, rather than being selected post hoc to optimize the AUC. Pairwise comparisons of the AUC, both between biomarkers and between the R-Baux score alone versus the R-Baux score combined with each biomarker, used the DeLong's method for two correlated ROC curves (18). Incremental predictive value was evaluated by comparing nested logistic regression models (R-Baux score alone versus R-Baux score plus biomarker) using the likelihood-ratio test. A sensitivity analysis additionally adjusted the biomarker-mortality association for age, burn surface area, and comorbidity. Statistical significance was defined as $p < 0.05$.

Descriptive statistics, group comparisons, and logistic regression modeling, including the likelihood-ratio test for nested models, were performed using SPSS software 25.0 (IBM Corp.). Pairwise comparisons between correlated ROC curves were performed using Python (version 3.12.3; pandas 3.0.2, scikit-learn 1.8.0,

statsmodels 0.14.6, and scipy 1.17.1) (Python Software Foundation), as this method is not natively available in SPSS. All logistic regression results were independently cross-validated between the two software packages.

This study was approved by the Ethics Committee of Dr. Soetomo Hospital Surabaya (approval number 3621/108/4/IV/2025). As a retrospective analysis of de-identified secondary medical record data, the requirement for individual written informed consent was waived by the ethics committee, in accordance with national and institutional policies for retrospective record-based research. Patient privacy was preserved throughout data collection and analysis.

3. Results

3.1. Subject Characteristics

A total of 238 patients met inclusion criteria, including four infants under one year of age, all of whom survived. The overall mortality rate was 30.7% (73 out of 238). Non-survivors were notably older, exhibited larger burn sizes, had higher R-Baux scores, and experienced shorter length of stay compared to survivors. Fire was identified as the most prevalent cause of injury (56.3%), followed by scalds (24.4%) and electrical injury (10.9%). Inhalation trauma was present in 36.6% of patients. The characteristics of all subjects, including comparisons between survivors and non-survivors, are presented in Table 1.

Table 1. Subject characteristics, survivors versus non-survivors

Characteristic	Total (n=238)	Survivor (n=165)	Non-survivor (n=73)	p
Age, years, median (IQR)	32.0 (29.0)	27.0 (32.0)	43.0 (24.0)	<0.001
TBSA, %, median (IQR)	22.0 (35.8)	16.0 (26.0)	53.0 (46.0)	<0.001
Revised Baux Score, median (IQR)	65.0 (57.0)	51.0 (41.0)	109.0 (50.0)	<0.001
Length of stay, days, median (IQR)	12.0 (15.0)	15.0 (18.0)	8.0 (8.3)	<0.001
Inhalation injury, n (%)	87 (36.6)	39 (23.6)	48 (65.8)	<0.001
Sex (male), n (%)	157 (66.0)	113 (68.5)	44 (60.3)	0.278
Comorbidity, n (%)	80 (33.6)	52 (31.5)	28 (38.4)	0.378
NLR, initial, median (IQR)	6.63 (9.51)	5.69 (8.49)	10.13 (11.90)	<0.001
PLR, initial, median (IQR)	24.47 (31.43)	22.80 (27.24)	29.71 (47.85)	0.076
PHR, initial, median (IQR)	21.84 (16.16)	23.50 (16.85)	18.06 (16.20)	0.003
SII, initial, median (IQR)	1921 (2964)	1804 (2769)	2490 (4250)	0.037
NLR, hyperinflammatory	6.64 (7.67)	5.79 (6.25)	10.14 (10.20)	<0.001
PLR, hyperinflammatory	20.50 (23.56)	20.82 (22.76)	17.28 (24.40)	0.218
PHR, hyperinflammatory	22.06 (17.10)	25.14 (15.83)	15.13 (10.79)	<0.001
SII, hyperinflammatory	1526 (2071)	1555 (1997)	1428 (1858)	0.487
NLR, recovery	7.21 (7.73)	6.59 (6.49)	13.23 (14.78)	<0.001
PLR, recovery	38.00 (40.35)	40.80 (40.27)	28.33 (48.59)	0.085

PHR, recovery	37.72 (19.53)	42.40 (21.99)	24.41 (20.34)	<0.001
SII, recovery	3065 (3822)	3209 (3744)	2376 (4566)	0.239

IQR, interquartile range; TBSA, total body surface area; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PHR, platelet-to-hemoglobin ratio; SII, systemic immune-inflammation index.

3.2. Biomarker Performance by Clinical Phase

Table 2 and Figure 1 provide a summary of the AUC for each biomarker across the different phases. According to Table 1, the direction of discrimination indicates that lower PHR values

are consistently associated with mortality throughout all phases. In contrast, higher NLR values are associated with mortality across these phases. The PLR and SII demonstrate a pattern where higher values indicate risk only during the initial phase, with a reversal observed in the hyperinflammatory and recovery phases.

Table 2. Area under the ROC curve for each biomarker by clinical phase

Biomarker	Initial phase	Hyperinflammatory phase	Recovery phase
NLR	0.640	0.675	0.702
PLR	0.572	0.555*	0.598*
PHR	0.622*	0.748*	0.820*
SII	0.585	0.531*	0.567*

NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PHR, platelet-to-hemoglobin ratio; SII, systemic immune-inflammation index.

**Lower biomarker value was associated with increased mortality risk; all other values follow the higher-value-indicates-risk direction.*

Through the application of DeLong's method for pairwise comparison, it was found that the PHR demonstrated superior performance compared to PLR and SII during both the hyperinflammatory and recovery phases (all $p < 0.001$). However, no significant difference was observed between PHR and NLR across any phase ($p = 0.73, 0.21, 0.069$, respectively). Notably, PHR was the only

biomarker that maintained a consistent discrimination direction across all three phases. Meanwhile, the NLR was the only biomarker with a consistently higher-value-indicates-risk direction across the phases, whereas PLR and the SII exhibited a reversal in direction after the initial phase.

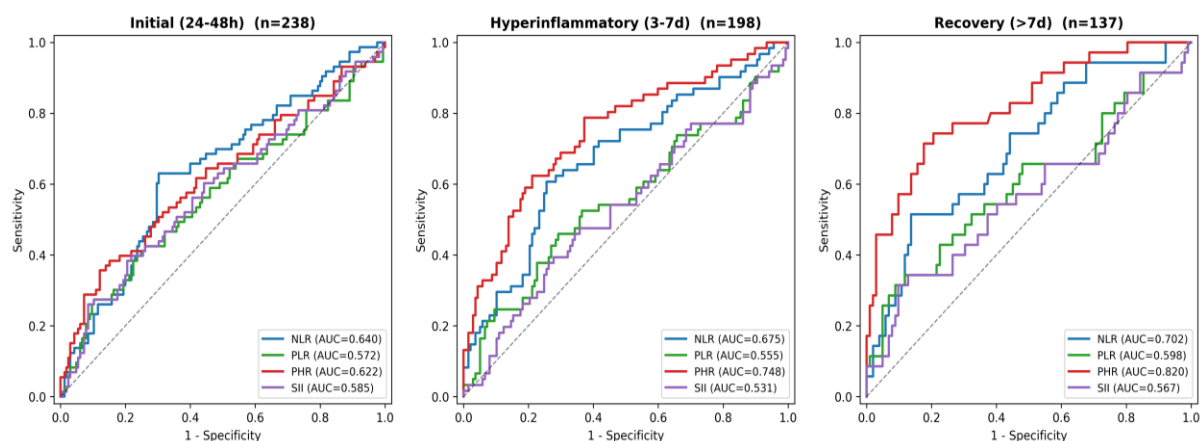


Figure 1. ROC curves for NLR, PLR, PHR, and SII by clinical phase.

3.3. Incremental Value Beyond the Revised Baux Score

The R-Baux score The R-Baux score independently demonstrated a strong predictive capability for in-hospital mortality (AUC = 0.868). Table 3 presents an analysis of whether the inclusion of each biomarker enhanced the predictive accuracy of the R-Baux Score, evaluated using the likelihood-ratio test and the method developed by DeLong et al. At the initial phase, none of the four biomarkers added significant predictive value. In the hyperinflammatory phase, both NLR and PHR provided modest yet statistically significant improvements by the likelihood-ratio test ($p=0.005$ and $p=0.02$, respectively), though neither achieved significance when evaluated using DeLong comparison.

The primary finding of this study indicates that during recovery phase, PHR contributed significant incremental value, improving discrimination from very good (AUC = 0.804, R-Baux score alone) to excellent (AUC = 0.895, combined model; likelihood-ratio $p<0.001$; DeLong $p=0.0096$). This finding remained significant after adjusting for age, burn surface area, and comorbidity (adjusted odds ratio 0.19 per 1 standard deviation increase, 95% confidence interval 0.09-0.41, $p<0.001$; Supplementary Table S2). The significance of this finding was maintained in a sensitivity analysis that excluded the four pediatric patients (AUC 0.801 to 0.894, DeLong $p=0.0096$). Comprehensive results for all phases and marker combinations are presents in Supplementary Table S1.

Table 3. Incremental predictive value of biomarkers beyond the R-Baux score

Phase	Marker	Revised Baux Score AUC	Combined AUC	LR-test p	DeLong p
Initial	all four	0.868	0.868-0.869	ns	ns
Hyperinflammatory	NLR	0.853	0.868	0.0045	0.214
Hyperinflammatory	PHR	0.853	0.861	0.023	0.436
Hyperinflammatory	PLR, SII	0.853	0.856	ns	ns
Recovery	PHR	0.804	0.895	<0.001*	0.0096*
Recovery	NLR, PLR, SII	0.804	0.805-0.812	ns	ns

AUC, area under the receiver operating characteristic curve; LR-test, likelihood-ratio test; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PHR, platelet-to-hemoglobin ratio; SII, systemic immune-inflammation index; ns, not significant ($p>0.05$). * $p<0.05$.

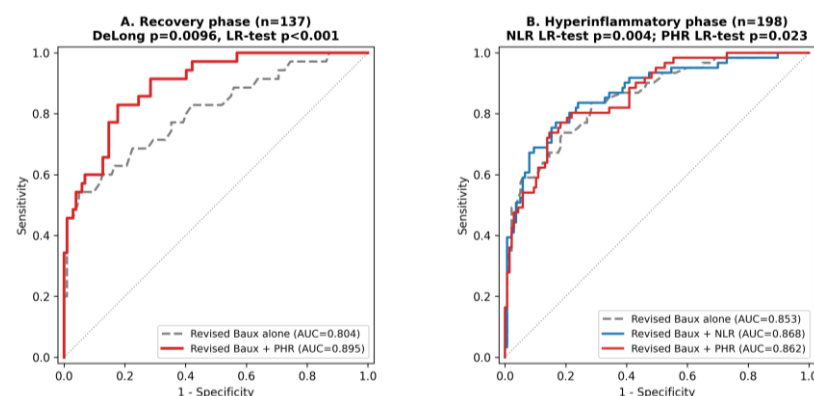


Figure 2. Incremental value beyond the R-Baux score:

- (a) recovery phase, R-Baux score alone versus combined with the platelet-to-hemoglobin ratio;
- (b) hyperinflammatory phase, R-Baux score alone versus combined with each of the two significant markers.

4. Discussion

This study investigated the potential of four CBC-derived inflammatory indices, measured serially across three phases of the clinical course, to improve the prediction of in-hospital mortality beyond the R-Baux score. The findings indicate a distinct and clinically relevant pattern rather than a uniform value across all four biomarkers. Specifically, no incremental value was observed at admission; the NLR demonstrated modest and phase-limited value, while the PHR exhibited substantial and robust value during the recovery phase.

At admission, no biomarker demonstrated an improvement over the R-Baux score. This outcome is unsurprising, as the R-Baux score already encompasses the primary early predictors of burn mortality, including age, burn extent, and inhalation injury. Inflammatory indices measured during the admission phase, before the systemic inflammatory response is fully established, seem to contribute little additional information beyond these three factors.

During the hyperinflammatory phase, NLR and PHR demonstrated a modest yet statistically significant improvement in likelihood-ratio. This likely reflects the peak neutrophil-driven and platelet-consumptive processes characteristic of this stage of injury (5,6). This timing is consistent with prior burn cohorts in which NLR's prognostic value was strongest around day 3–7 rather than at admission (19). However, neither ratio achieved significance on the DeLong comparison, suggesting a genuine but minor effect at this stage. Both associations remained statistically significant after adjustment for age, burn surface area, and comorbidity. NLR adjusted odds ratio 1.75 (95% confidence interval 1.10-2.78, $p=0.019$), and PLR adjusted odds ratio 0.52 (95% confidence interval 0.30-0.91, $p=0.023$), indicating these modest hyperinflammatory-phase signals are unlikely to be solely attributed to these measured confounders (Supplementary Table S2).

The primary finding of this study is the result observed during the recovery phase. This specificity to the recovery phase is interpreted as biologically coherent, as the PHR reflects the platelet count, an indicator of hemostasis and inflammation that may recover or not as the acute phase resolves, and the hemoglobin level, which reflecting perfusion, oxygen delivery, and the overall impact of the catabolic response. While specific evidence regarding PHR in burn cases is limited, analogous concepts have been explored in other medical domains. The combination of a platelet-derived inflammatory signal with hemoglobin-based perfusion status is proposed to offer prognostic insights that are not achievable by either measure independently. Notably, the platelet-to-hemoglobin ratio has been identified as an independent predictor of all-cause mortality in patients with acute coronary syndrome and diabetes mellitus (13). Since the R-Baux score is determined at the time of admission, it is unable to account for changes in a patient's condition over time. In contrast, the PHR, which can be measured serially from routine CBCs without additional costs, effectively captures this evolving information. This is particularly crucial for updating a clinician's risk assessment after the initial resuscitation period, as many patients who eventually die from their injuries do so as a result of complications, not the initial injuries.

An additional, unexpected finding concerns the direction of association across phases. The PHR maintained a consistent direction throughout the clinical course, as did NLR. However, PLR and SII both reversed direction after the initial phase. This reversal may reflect the transient, reactive nature of platelet and lymphocyte dynamics during burn recovery. Reactive thrombocytosis, a known characteristic of the healing phase in survivors, could potentially alter the early associations between platelet-based ratios and outcome once the acute phase resolves (20,21). This instability may partly explain why these two markers, despite achieving statistical significance in univariate comparisons at some phases, did not add incremental value beyond the R-Baux score. A marker with an unstable relationship to outcomes over time is unlikely to offer reliable independent prognostic information. In a recent meta-analysis involving 4,515 patients with severe burns, a significant correlation was identified between the admission PLR and short-term mortality (15). Our results do not contradict that correlation, as we did not evaluate the unadjusted relationship between

this ratio and outcomes. However, our findings suggest that while the platelet-to-lymphocyte ratio may initially signal risk at admission, it does not offer additional predictive value over the R-Baux score within our cohort. This distinction between correlation and incremental predictive value has not been extensively explored in the current literature on PLR.

These findings should be considered in the context of existing literature on individual biomarkers in burn injury, which has generally evaluated these markers in isolation rather than against an established prognostic benchmark (16,17,22). In a comprehensive study involving 2,758 patients in burn intensive care, researchers evaluated NLR, PLR, and SII indices as predictors of acute kidney injury, rather than mortality. The study found that these indices provided only moderate discrimination and were not compared against a validated mortality score (23). Our nested-model approach is designed to address this gap. While our findings do not challenge previous studies linking the NLR with burn outcomes, they offer a new perspective that an association with outcomes does not necessarily provide additional insights beyond those available at admission. We utilized the R-Baux score as the baseline comparator due to its status as the most validated mortality score in this context, with an AUC of 0.94 in an external burn intensive care cohort (24). Our results suggest that for three of the four markers tested, the prognostic information is entirely encompassed by the R-Baux score.

This study has several limitations. The retrospective, single-center design may introduce selection and information bias, and exclusive sampling from one hospital may limit generalizability. The recovery-phase analysis was necessarily limited to patients whose clinical course extended more than seven days. This subgroup exhibited a lower mortality rate compared to those excluded from this analysis (25.5% versus 37.6%). This observation aligns with the clinical reality that patients surviving the acute hyperinflammatory phase constitute a lower-risk population. Therefore, the recovery-phase PHR findings should be viewed as indicative of prognostic value conditional on surviving the initial critical period, rather than as predictors of early mortality. One methodological challenge involves the shared components among biomarkers. To mitigate this, we conducted an independent evaluation of each biomarker in comparison with the R-Baux score. This approach was chosen to prevent the occurrence of unstable coefficients that could arise from collinearity. Although confounding from unmeasured variables cannot be entirely ruled out, the incremental value of PHR remained consistent across confounder-adjusted models (Supplementary Table S2). To improve the reliability of results during the recovery phase, evaluating the effectiveness of the platelet-to-hemoglobin ratio in prospective, multicenter cohorts is essential.

5. Conclusion

Among four CBC-derived inflammatory indices, only PHR consistently demonstrated statistically significant incremental predictive value beyond the R-Baux score. This value was primarily observed in the recovery phase rather than at admission. NLR showed a limited signal confined to the hyperinflammatory phase, while PLR and SII did not significantly improve the R-Baux score at any stage of the clinical course, despite univariate significance at some phases. These findings suggest that routine, low-cost monitoring of the PHR after the acute resuscitation period may offer clinicians a practical method to update mortality risk stratification as a patient's clinical trajectory progresses. This approach would complement, rather than replace, the R-Baux score's assessment at the time of admission. Remaining gaps include external validation in prospective, multicenter cohorts and the evaluation of whether serial measurements of PHR rather than relying on single time-point measurements, improve prognostic accuracy.

Author Contributions

Conceptualization, A.P., I.D.S., and I.H.; methodology, A.P. and N.H.S.; software, N.H.S.; validation, A.P., I.D.S., and I.H.; formal analysis, A.P. and N.H.S.; investigation, A.P. and N.H.S.; resources, A.P.; data curation, A.P. and N.H.S.; original draft preparation, A.P. and N.H.S.; review and editing, A.P., I.D.S., and

I.H.; visualization, N.H.S.; supervision, I.D.S., and I.H.; project administration, A.P. All authors have read and agreed to the published version of the manuscript.

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Ethics Statement

This study was approved by the Ethics Committee of Dr. Soetomo Hospital Surabaya, approval number 3621/108/4/IV/2025.

Conflict of Interest

The authors declare that they have no conflict of interest.

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Supplementary Table S1

Full nested logistic regression results: incremental value of each biomarker beyond the R-Baux score

Phase	Marker	n	OR (per +1SD)	95% CI	Wald p	rBaux AUC	Combined AUC	LR-test p	DeLong p
Initial	NLR	238	1.16	0.86-1.58	0.330	0.868	0.869	0.347	0.6631
Initial	PLR	238	1.11	0.80-1.54	0.530	0.868	0.868	0.540	0.8855

Initial	PHR	238	1.22	0.86-1.73	0.267	0.868	0.869	0.252	0.8060
Initial	SII	238	1.11	0.80-1.54	0.529	0.868	0.868	0.540	0.9171
Hyperinflammatory	NLR	198	1.78	1.12-2.82	0.015	0.853	0.868	0.004*	0.2135
Hyperinflammatory	PLR	198	1.24	0.87-1.76	0.228	0.853	0.856	0.208	0.5725
Hyperinflammatory	PHR	199	0.55	0.32-0.95	0.032	0.853	0.861	0.023*	0.4355
Hyperinflammatory	SII	198	1.27	0.88-1.82	0.203	0.853	0.856	0.170	0.5920
Recovery	NLR	137	1.72	0.85-3.50	0.133	0.804	0.805	0.098	0.9042
Recovery	PLR	137	0.74	0.46-1.19	0.219	0.804	0.812	0.205	0.4109
Recovery	PHR	137	0.22	0.11-0.43	0.000	0.804	0.895	<0.001*	0.0096
Recovery	SII	137	0.80	0.51-1.28	0.359	0.804	0.809	0.351	0.4855

OR, odds ratio (per +1 SD increase in standardized biomarker value); CI, confidence interval; Wald *p*, significance of the biomarker coefficient in the combined model; LR-test *p*, likelihood-ratio test comparing the combined model to the R-Baux Score-only model; DeLong *p*, DeLong's test comparing the R-Baux-alone AUC to the combined-model AUC on the same patients.

Supplementary Table S2

Confounder-adjusted sensitivity analysis: biomarker associations with mortality adjusted for age, burn surface area, and comorbidity

Phase	Marker	n	Unadjusted OR (p)	Adjusted OR (95% CI, p)
Hyperinflammatory	NLR	198	2.20 (p<0.001)	1.75 (1.10-2.78, p=0.019)
Hyperinflammatory	PHR	199	0.29 (p<0.001)	0.52 (0.30-0.91, p=0.023)
Recovery	PHR	137	0.20 (p<0.001)	0.19 (0.09-0.41, p<0.001)

OR, odds ratio per +1 SD increase in standardized biomarker value; CI, confidence interval; NLR, neutrophil-to-lymphocyte ratio; PHR, platelet-to-hemoglobin ratio.

Supplementary Table S3

DeLong's Test Results for All ROC Curve Comparisons

Part A. Pairwise biomarker-vs-biomarker AUC comparisons, by phase (all 6 possible marker pairs)

Phase	Marker A	Marker B	n	AUC A vs B	z-statistic	p-value
Initial	NLR	PLR	238	0.640 vs 0.572	2.07	0.0388*
Initial	NLR	PHR	238	0.640 vs 0.622	0.35	0.7253
Initial	NLR	SII	238	0.640 vs 0.585	1.85	0.0645
Initial	PLR	PHR	238	0.572 vs 0.622	-0.69	0.4898
Initial	PLR	SII	238	0.572 vs 0.585	-2.53	0.0114*
Initial	PHR	SII	238	0.622 vs 0.585	0.53	0.5965

Hyperinflammatory	NLR	PLR	198	0.675 vs 0.555	1.47	0.1406
Hyperinflammatory	NLR	PHR	198	0.675 vs 0.748	-1.25	0.2098
Hyperinflammatory	NLR	SII	198	0.675 vs 0.531	1.73	0.0830
Hyperinflammatory	PLR	PHR	198	0.555 vs 0.748	-4.83	<0.001*
Hyperinflammatory	PLR	SII	198	0.555 vs 0.531	1.72	0.0858
Hyperinflammatory	PHR	SII	198	0.748 vs 0.531	5.03	<0.001*
Recovery	NLR	PLR	137	0.702 vs 0.598	1.01	0.3135
Recovery	NLR	PHR	137	0.702 vs 0.820	-1.82	0.0692
Recovery	NLR	SII	137	0.702 vs 0.567	1.29	0.1958
Recovery	PLR	PHR	137	0.598 vs 0.820	-3.83	<0.001*
Recovery	PLR	SII	137	0.598 vs 0.567	4.52	<0.001*
Recovery	PHR	SII	137	0.820 vs 0.567	4.29	<0.001*

AUC, area under the receiver operating characteristic curve. χ^2 -statistic and p -value from DeLong's test for two correlated ROC curves on the same patients. Bold/asterisk = $p < 0.05$. Direction of each biomarker (higher-value-indicates-risk or lower-value-indicates-risk) applied consistently with Table 2 of the main manuscript before comparison.

Part B. R-Baux Score alone versus R-Baux Score combined with each biomarker (recap of DeLong p column from Supplementary Table S1)

Phase	Marker	n	Revised Baux AUC	Combined AUC	DeLong p
Initial	NLR	238	0.868	0.869	0.6631
Initial	PLR	238	0.868	0.868	0.8855
Initial	PHR	238	0.868	0.869	0.8060
Initial	SII	238	0.868	0.868	0.9171
Hyperinflammatory	NLR	198	0.853	0.868	0.2135
Hyperinflammatory	PLR	198	0.853	0.856	0.5725
Hyperinflammatory	PHR	199	0.853	0.861	0.4355
Hyperinflammatory	SII	198	0.853	0.856	0.5920
Recovery	NLR	137	0.804	0.805	0.9042
Recovery	PLR	137	0.804	0.812	0.4109
Recovery	PHR	137	0.804	0.895	0.0096*
Recovery	SII	137	0.804	0.809	0.4855

AUC, area under the receiver operating characteristic curve. Bold/asterisk = $p < 0.05$.

Software note: All DeLong's test results in this file were computed in Python (version 3.12.3; pandas, scikit-learn, statsmodels, scipy) using a standard implementation of the DeLong, DeLong and Clarke-Pearson (1988) method, as this test is not natively available in SPSS.