

The Prognostic Value of the Lactate-to-Albumin Ratio in Predicting Mortality in the Pediatric Intensive Care Unit

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ABSTRACT

Purpose: To evaluate the prognostic value of admission lactate-to-albumin ratio (LAR) for mortality in a large pediatric intensive care unit (PICU) cohort, compare it with lactate, albumin, Pediatric risk of mortality-3 (PRISM-3), and Pediatric logistic organ dysfunction-2 (PELOD-2) scores, and assess its performance across diagnostic subgroups and association with intubation.

Methods: This retrospective cohort included 7,317 patients aged 1 month–18 years admitted to a tertiary PICU between September 2019 and April 2025 with admission lactate and albumin measurements. ROC analysis, logistic regression, DeLong tests, and analyses of 10 diagnostic subgroups with Bonferroni correction were performed.

Results: Mortality was 10.39% (760/7,317). LAR was higher in non-survivors than survivors (median 0.90 vs. 0.47; $p<0.001$) and predicted mortality with an AUC of 0.743 (cutoff 0.70; sensitivity 63.3%; specificity 74.9%). It outperformed lactate (AUC 0.691) but not albumin (AUC 0.748). Each 1-unit increase in LAR increased mortality odds 2.86-fold (95% CI 2.60–3.14); this association remained after adjustment for age, sex, and PRISM-3 (adjusted OR 2.07) or PELOD-2 (adjusted OR 2.12). LAR was associated with mortality in all subgroups (OR 1.96–5.67), remaining significant after Bonferroni correction in nine, and was modestly higher in intubated survivors ($p<0.001$). PRISM-3 and PELOD-2 outperformed LAR (AUC 0.897 and 0.889; both $p<0.001$), although adding LAR increased their AUCs to 0.910 and 0.906.

Conclusion: Admission LAR is a rapid, independent predictor of PICU mortality that outperforms lactate and remains informative across diagnoses. Although not a substitute for PRISM-3 or PELOD-2, it may complement these scores in early risk stratification.

Keywords: Lactate/albumin ratio; PRISM-3; PELOD-2; pediatric intensive care unit; mortality; biomarker

73 **What Is Known**

- 74 - Lactate and albumin are associated with outcomes in critically ill children.
- 75 - The lactate-to-albumin ratio has been proposed as a prognostic marker in critically ill patients.
- 76 - Studies evaluating LAR in heterogeneous, broad-spectrum pediatric populations remain limited.

77 **What is New**

- 78 - In 7,317 patients, LAR was associated with mortality after adjustment for PRISM-3 or PELOD-2.
- 79 - The lactate-to-albumin ratio discriminated mortality better than lactate; superiority over albumin was not
- 80 demonstrated.

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82 **List of Abbreviations**

83 **AUC:** Area under the curve

84 **CI:** Confidence interval

85 **IQR:** Interquartile range

86 **LAR:** Lactate-to-albumin ratio

87 **OR:** Odds ratio

88 **PELOD-2:** Pediatric logistic organ dysfunction-2

89 **PICU:** Pediatric intensive care unit

90 **PRISM-3:** Pediatric risk of mortality-3

91 **ROC:** Receiver operating characteristic

92 **SD:** Standard deviation

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INTRODUCTION

Predicting mortality in pediatric intensive care patients is critical for guiding triage and resource allocation, but conventional severity scores rely on numerous parameters, require time-consuming calculation, and respond slowly to dynamic changes in a patient's condition. This has motivated the search for simpler biomarkers that are rapid to obtain, inexpensive, and can reliably predict mortality risk at an early stage of PICU admission.

Lactate is a well-established indicator of tissue hypoxia and impaired perfusion, rising in shock, systemic inflammation, trauma, and infection, and is a recognized marker of poor prognosis in critically ill children [1]; it is also a diagnostic criterion for septic shock in the Phoenix sepsis score [2]. Admission lactate ≥ 4 mmol/L and poor lactate clearance within 6–24 hours are both associated with increased pediatric mortality [3,4], though lactate's specificity is limited by its sensitivity to liver function, drugs, and metabolic disease [5]. Serum albumin, similarly, falls in critical illness as a reflection of inflammation, nutritional status, and capillary permeability, and is independently associated with poor prognosis [6,7].

The lactate/albumin ratio (LAR) combines both parameters and has shown superior prognostic performance compared with either alone in critically ill patients. However, large, diagnostically heterogeneous pediatric cohorts evaluating LAR remain scarce, and its relationship with clinical severity indicators such as mechanical ventilation need has been little studied [6,7].

This study evaluates the prognostic value of admission LAR for predicting PICU mortality in a large, heterogeneous tertiary-center cohort, determines its optimal threshold and superiority over lactate and albumin alone, and examines its performance across admission-diagnosis subgroups and its association with the need for intubation.

METHODS

Study Design

This retrospective observational study was conducted at the 32-bed Pediatric Intensive Care Unit (PICU) of Ankara Bilkent City Hospital, a tertiary referral center. The study protocol was approved by the local ethics committee (Decision No: TABED 1/1619/2025) in accordance with the Declaration of Helsinki; as a retrospective review of existing records, individual informed consent was waived.

Study Population and Data Collection

Electronic medical records of all patients aged 1 month to 18 years admitted to the PICU between September 2019 and April 2025 were reviewed (N = 8,481). Patients with missing admission lactate or albumin, metabolic disease, or nephrotic syndrome were excluded; 7,317 patients met the study criteria (Figure 1).

Inclusion criteria:

1- Between 1 month and 18 years of age

2- Patients in whom lactate and albumin can be detected in hospitalized blood samples

Exclusion criteria:

1- Patients for whom lactate and albumin levels in blood samples cannot be determined

2- Patients diagnosed with metabolic diseases

3- Patients with nephrotic syndrome

Demographic data (age, sex), primary admission diagnosis, mechanical ventilation (intubation) status, and outcome (survivor/non-survivor) were retrospectively recorded. Admission diagnoses were categorized into 10 groups: neurological, hematological, infectious, cardiovascular, endocrine, trauma/surgical, musculoskeletal, oncological, respiratory, and gastrointestinal. PRISM-3 and PELOD-2 scores were calculated using the worst values recorded during the first 24 hours after PICU admission. Complete data for both scores were available for all 7,317 patients.

Laboratory Measurements and LAR Calculation

Venous or arterial blood samples drawn within the first hour of admission were used for laboratory analysis. Serum lactate (mmol/L) and albumin (g/dL) were measured by standard methods, and the Lactate-Albumin Ratio (LAR) was calculated as the admission lactate level divided by the admission albumin level.

Patients were divided into survivors (n=6,557) and non-survivors (n=760). Lactate, albumin, and LAR were compared between groups, and ROC analysis was used to determine the optimal mortality cutoff for each; associations with length of intensive care unit stay were also evaluated.

Statistical Analysis

SPSS 25.0 (SPSS Inc., Chicago, IL) was used for analysis. Normality was assessed with the Kolmogorov-Smirnov test. Categorical variables were expressed as frequencies/percentages, and continuous variables as mean $\pm$ SD or median (IQR), depending on distribution.

Survivors and non-survivors were compared using the Mann-Whitney U test (continuous variables) and Chi-square test (categorical variables). ROC curve analysis evaluated LAR's prognostic performance for mortality; AUC was calculated and the optimal cutoff determined by the Youden Index. Univariate and multivariate logistic regression identified independent mortality risk factors, with ORs and 95% CIs calculated per 1-unit LAR increase; ROC-AUC values were compared pairwise using the DeLong method. The multivariate model was extended to include admission diagnosis category alongside age and sex; PRISM-3 and PELOD-2 were each entered in separate models with age and sex — rather than together, given their high collinearity ($r = 0.91$) — and mechanical ventilation status was excluded as a covariate, since it was a near-deterministic correlate of death that produced quasi-complete separation. The intubation-LAR comparison was restricted to survivors ($n = 6,557$) to eliminate confounding by mortality (all non-survivors were intubated); effect sizes were expressed as rank-biserial r . LAR's discriminatory performance was compared with PRISM-3 and PELOD-2 via DeLong tests, and its incremental value assessed by adding it to PRISM-3/PELOD-2 models and evaluating the change in fit with the likelihood ratio test. LAR-mortality associations were also examined within each of the 10 diagnosis subgroups, with a Bonferroni-corrected threshold ($\alpha = 0.005$) applied given the 10 comparisons. A p -value <0.05 was considered significant for all other tests.

RESULTS

The study included 7,317 patients with lactate and albumin measurements available at admission (mean age 6.71 $\pm$ 5.86 years, median 5.00 years [IQR 1.00–12.00]; 55.43% male, n=4,056). Overall, 6,557 patients (89.61%) survived, and 760 (10.39%) died, with no significant difference in age or gender between groups ($p>0.05$). The most frequent admission diagnoses were respiratory tract disease (n=1,535, 21.0%), neurological conditions (n=1,316, 18.0%), and trauma/surgery (n=963, 13.2%); mortality was highest in the hematological (26.5%) and cardiovascular (18.3%) groups ($p<0.001$) (Table 1). Invasive mechanical ventilation was required in 28.17% (n=2,061) of patients. Median length of stay was 3 days (IQR 1–7) overall, and was significantly longer among non-survivors than survivors (median 6 [2–16] vs. 3 [1–6] days, $p<0.001$) (Table 1).

Admission lactate, albumin, and LAR all differed significantly between groups (Table 1). Lactate was higher in non-survivors than survivors (median 2.75 [1.69–5.43] vs. 1.74 [1.27–2.54] mmol/L, $p<0.001$), while albumin was lower (median 3.10 [2.60–3.62] vs. 3.82 [3.33–4.22] g/dL, $p<0.001$). LAR was correspondingly higher among non-survivors (median 0.90 [0.52–1.98] vs. 0.47 [0.34–0.70], $p<0.001$), consistent with the combined effect of rising lactate and falling albumin in non-survivors.

LAR predicted PICU mortality with an AUC of 0.743 (95% CI 0.723–0.763), significantly superior to lactate alone (AUC 0.691, 95% CI 0.669–0.712; $p<0.001$). Albumin alone achieved an AUC of 0.748 (95% CI 0.727–0.769), not significantly different from LAR (DeLong $Z=-0.44$, $p=0.662$). The optimal cutoff for LAR was 0.70 (sensitivity 63.3%, specificity 74.9%); for lactate, 2.70 mmol/L (sensitivity 51.3%, specificity 78.1%); and for albumin, 3.47 g/dL (sensitivity 69.2%, specificity 68.8%) (Figure 2 a).

In univariate logistic regression analysis, each 1-unit increase in LAR was associated with a 2.86-fold increase in mortality risk (OR 2.86, 95% CI 2.60–3.14, $p<0.001$) (Table 2). This remained significant after adjusting for age, sex, and PRISM-3 (adjusted OR 2.07, 95% CI 1.86–2.29) or PELOD-2 (adjusted OR 2.12, 95% CI 1.91–2.35); mechanical ventilation was excluded as a covariate because it was a near-deterministic correlate of death, producing quasi-complete separation. LAR was also a significant univariate predictor of mortality within all 10 admission-diagnosis subgroups, with odds ratios ranging from 1.96 (oncological) to 5.67 (trauma/surgical). Applying a Bonferroni-corrected threshold for these 10 comparisons ($\alpha = 0.005$), nine of ten subgroups remained significant; only the endocrinological group (8 deaths) fell short ($p = 0.007$), despite a similarly elevated point estimate (Table 4). Confidence intervals were widest in the smallest subgroups (endocrinological, 8 deaths; musculoskeletal, 13 deaths), and these estimates warrant cautious interpretation.

Because all non-survivors were intubated, this comparison was restricted to survivors ($n = 6,557$) to avoid confounding by mortality. Intubated survivors ($n = 1,301$) had significantly higher admission LAR than non-intubated survivors ($n = 5,256$) (median 0.531 vs. 0.455, $p < 0.001$; rank-biserial $r = 0.145$) (Table 3), indicating a modest association that is weaker than LAR's relationship with mortality.

PRISM-3 and PELOD-2, recorded for all 7,317 patients, were significantly higher among non-survivors (PRISM-3: median 13.0 vs. 4.0; PELOD-2: median 7.0 vs. 2.0; both $p < 0.001$) (Table 1). Both scores markedly outperformed LAR in predicting mortality: PRISM-3 AUC 0.897 (cutoff 9; sensitivity 79.9%, specificity 82.9%) and PELOD-2 AUC 0.889 (cutoff 4; sensitivity 87.6%, specificity 74.0%), versus 0.743 for LAR (Figure 2b). DeLong tests confirmed LAR's discriminatory power was significantly inferior to both PRISM-3 and PELOD-2 ($Z = -13.83$ and -13.17 , respectively; both $p < 0.001$).

LAR correlated only weakly with PRISM-3 and PELOD-2 (Spearman $r = 0.19$ for both), suggesting it captures largely independent information. When added to logistic models already containing PRISM-3 or PELOD-2, LAR remained a significant independent predictor (adjusted OR 2.07, 95% CI 1.86–2.29 with PRISM-3; OR 2.13, 95% CI 1.91–2.36 with PELOD-2; both $p < 0.001$), significantly improving model fit (likelihood ratio test, both $p < 0.001$) and modestly increasing AUC (PRISM-3 model: 0.897→0.910; PELOD-2 model: 0.889→0.906). Thus, while LAR alone discriminates mortality considerably less well than PRISM-3 or PELOD-2, it adds statistically significant complementary information and, given its simplicity and rapid availability, may serve as a practical adjunct to formal severity scoring.

DISCUSSION

In our large-scale retrospective cohort of 7,317 PICU patients, admission LAR was a strong, independent predictor of mortality. LAR values were roughly two-fold higher in non-survivors than survivors (median 0.90 vs. 0.47, $p<0.001$), and LAR predicted mortality with an AUC of 0.743 (cutoff 0.70; 63.3% sensitivity, 74.9% specificity), significantly outperforming lactate alone. Each 1-unit increase in LAR was associated with a 2.86-fold increase in mortality risk, and LAR remained independently predictive after adjusting for age, sex, and PRISM-3 or PELOD-2. However, LAR did not significantly outperform albumin alone (AUC 0.748; DeLong $p=0.662$), indicating that albumin is the dominant contributor to LAR's discriminatory power. These findings support LAR as a clinically usable, rapidly available tool for mortality risk assessment in the PICU.

Serum lactate is a well-established marker of tissue hypoxia and impaired perfusion, generated by anaerobic metabolism, and elevated admission lactate is consistently associated with mortality in critically ill children [3,4,8]. In our cohort, lactate levels in non-survivors were roughly twice those of survivors, and each 1 mmol/L increase in lactate raised mortality risk by 36% (OR 1.36, 95% CI 1.32–1.41) (Table 2). However, both the AUC (0.691) and optimal cutoff for lactate were lower than reported in sepsis-restricted cohorts, such as Srigiri et al. (AUC 0.86, cutoff 3.37 mmol/L, $n=100$) [9] and Choudhary et al. (cutoff ≥ 4 mmol/L in septic shock) [10]; this likely reflects that our cohort was not restricted to sepsis or septic shock and instead spanned a heterogeneous range of admission diagnoses (respiratory 21.0%, neurological 18.0%, trauma/surgery 13.2%). These findings confirm lactate's predictive value while highlighting the limitations of using it in isolation, since it can also be elevated by non-hypoxic causes such as drug effects or underlying metabolic disease [5].

Serum albumin reflects hepatic synthetic capacity, capillary integrity, and systemic inflammatory burden, and hypoalbuminemia is a recognized marker of poor prognosis in critical illness [6,7]. Admission albumin was significantly lower among non-survivors in our cohort, and our AUC (0.748) and cutoff (3.47 g/dL) closely matched those reported by Ari et al. in a multicenter PICU cohort (AUC 0.731, cutoff 3.785 g/dL) [11]; each 1 g/dL increase in albumin was associated with 73% lower odds of mortality (OR 0.27, 95% CI 0.24–0.31) (Table 2), comparable in direction to the 4.1-fold risk increase for hypoalbuminemia reported by Gowa et al. [12] and the U-shaped albumin-mortality relationship described by Zhang et al. in a large cohort of PICU patients [13]. Notably, albumin alone achieved the highest AUC of the three parameters examined. In univariate analysis, each 1 g/dL increase in albumin was associated with 73% lower odds of mortality.

The lactate/albumin ratio combines two distinct pathophysiological signals — tissue hypoperfusion and metabolic stress (lactate) and systemic inflammation and capillary leakage (albumin) — into a single marker [7]. In principle, this should give LAR greater discriminatory power than either component alone; in our cohort, this held true relative to lactate (DeLong $p < 0.001$) but not relative to albumin (DeLong $p = 0.662$), indicating that albumin is the stronger contributor within the ratio. Our AUC for LAR is broadly consistent with prior pediatric cohorts, though reported thresholds vary widely — from 0.5 in Aygün et al.'s cohort of 379 critically ill patients [15] to 1.84 in Le et al.'s septic shock cohort (AUC 0.91) [14] and >1.016 in Choi et al.'s cohort with chronic disease (AUC 0.867) [6] — reflecting differences in population, illness type, and measurement timing. Wang et al. similarly reported an AUC of 0.74 for LAR in 8,832 critically ill children, with each unit increase raising mortality risk 1.44-fold [7]; our corresponding univariate OR of 2.86 (95% CI 2.60–3.14) likewise underscores LAR's clinical predictive strength.

A biomarker's clinical value depends on whether it retains independent predictive value after adjusting for confounders. LAR remained an independent risk factor for mortality after adjusting for age, sex, and PRISM-3 (adjusted OR 2.07, 95% CI 1.86–2.29) or PELOD-2 (adjusted OR 2.12, 95% CI 1.91–2.35); mechanical ventilation status was excluded from these models because it was a near-deterministic correlate of death, producing quasi-complete separation. This indicates that LAR's prognostic value is not simply explained by age, sex, or overall illness severity as captured by these composite scores. Similarly, Wang et al. and Pollack et al. reported LAR as an independent predictor of mortality and multi-organ dysfunction, respectively, in their multivariate models [7,16].

Subgroup analysis showed that LAR was associated with mortality across all 10 admission-diagnosis categories before correction, and the association remained statistically significant in nine of ten categories after Bonferroni correction (Table 4). The association was strongest in the trauma/surgical group, consistent with the combined effects of blood loss, tissue injury, and hypovolemic shock on lactate and albumin, and in the neurological group, where the sympathetic stress response and systemic inflammation associated with severe neurological illness may similarly elevate lactate and lower albumin. A comparably strong association was also observed in the gastrointestinal/hepatic group, where hepatic synthetic dysfunction and hypoperfusion likely act together. These findings suggest LAR may be particularly useful for early risk stratification in trauma/surgical, neurological, and gastrointestinal/hepatic admissions, while remaining informative, if to a lesser degree, across other diagnostic groups.

Intubation status reflects disease severity and organ dysfunction, and prior adult studies have linked LAR to mechanical ventilation requirements and mortality [17,18]; pediatric data on this relationship remain limited. To avoid confounding by mortality (all non-survivors required intubation), we restricted this comparison to survivors (n=6,557): intubated survivors had significantly higher admission LAR than non-intubated survivors, though the small effect size (rank-biserial $r=0.145$) indicates this association is considerably weaker than LAR's relationship with mortality. This likely reflects that intubated patients often present with severe respiratory failure, shock, sepsis, or neurological compromise — conditions that independently elevate lactate and lower albumin — suggesting LAR may offer modest additional support for early respiratory-support decisions.

Several limitations should be noted. This was a single-center, retrospective study, limiting establishment of causal relationships and generalizability to other settings. Only a single admission measurement of lactate and albumin was used; serial measurements and clearance dynamics (e.g., LAR values at 6 and 24 hours) were not evaluated, and could further clarify the prognostic trajectory of LAR over time. Exclusion of patients with metabolic disease or nephrotic syndrome limits generalizability of our findings to these groups. While we were able to compare LAR against PRISM-3 and PELOD-2, LAR alone showed considerably lower discriminatory power than these composite scores, and its incremental contribution, though statistically significant, was modest in absolute terms; PIM data were not available in our dataset, so comparison with this score remains a subject for future work. The main strengths of this study are its large sample size (7,317 patients) — among the largest pediatric LAR cohorts reported to date — and its heterogeneous diagnostic case-mix, which allowed us to test LAR's performance as a broadly applicable marker rather than one restricted to sepsis or septic shock, including its relationship with intubation need, its consistency across ten diagnostic subgroups, and its discriminatory performance.

In conclusion, this study demonstrates that the LAR value at admission to the PICU, based on a large cohort of 7,317 patients, is a valid, independent, and clinically meaningful biomarker for predicting mortality. LAR (AUC 0.743, cutoff value 0.70) significantly outperforms lactate alone (DeLong $p<0.001$) while maintaining a statistically comparable discriminatory accuracy to albumin alone (DeLong $p=0.662$). Its association with intubation and its significant association with mortality in nine of ten diagnostic subgroups after Bonferroni correction suggest that LAR retains prognostic value in a heterogeneous PICU population. Its extremely simple calculation and availability from routine laboratory tests make LAR a practical clinical tool, especially in resource-constrained settings. Confirming our results through multicenter prospective studies and investigating LAR's contribution to prognostic value alongside current disease severity scores will contribute to the literature.

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Human rights statement: The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

408 **Table 1.** Comparison of clinical and laboratory characteristics between survivors and non-survivors

Parameter	Total	Survivors (n=6.557)	Non-survivors (n=760)	p*
Age (year). Mean $\pm$ SD	6.71 $\pm$ 5.86	6.70 $\pm$ 5.84	6.75 $\pm$ 6.00	0.684 ^a
Gender				0.387 ^b
Male. n (%)	4056 (55.4)	3.623 (55.3)	433 (57.0)	
Female. n (%)	3261 (44.6)	2.934 (44.7)	327 (43.0)	
Diagnoses				
Neurological. n (%)	1.316	1.224	92	<0.001 ^b
Hematological. n (%)	603	443	160	
Infectious Diseases - Sepsis. n (%)	266	224	42	
Cardiovascular. n (%)	744	608	136	
Endocrinological . n (%)	419	411	8	
Trauma-Postoperative Surgery. n (%)	963	902	61	
Musculoskeletal Disorders. n (%)	535	522	13	
Oncological. n (%)	602	496	106	
Respiratory System. n (%)	1.535	1.412	123	
Gastrointestinal. n (%)	334	315	19	
Intubation. n (%)	2.061	1.301(19.8%)	760(100%)	<0.001 ^b
PICU length of stay (day). Median (Q1-Q3)	3 (1-7)	3 (1-6)	6 (2-16)	<0.001 ^a
Lactate Median (Q1-Q3)	1.8 (1.30-2.69)	1.74(1.27-2.54)	2.75(1.69-5.43)	<0.001 ^a
Albumin Median (Q1-Q3)	3.76 (3.23-4.20)	3.82(3.33-4.22)	3.10(2.60-3.62)	<0.001 ^a
Lactate/Albumin Ratio (LAR) Median (Q1-Q3)	0.49 (0.34-0.76)	0.47(0.34-0.70)	0.90(0.52-1.98)	<0.001 ^a
PRISM-3 Median (Q1-Q3)	5.0 (2.0-8.0)	4.0(2.0-7.0)	13.0(9.0-20.0)	<0.001 ^a
PELOD-2 Median (Q1-Q3)	3.0 (0.0-4.0)	2.0(0.0-4.0)	7.0(4.0-10.0)	<0.001 ^a

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410 * Data are presented as n (%), mean $\pm$ standard deviation, or median (interquartile range), as
411 appropriate. ^a Mann-Whitney U test; ^b Pearson's chi-square test. The p value reported for admission
412 diagnosis represents the overall comparison of the distribution of the 10 diagnostic categories between
413 survivors and non-survivors.

414 **SD:** standard deviation; **IQR:** interquartile range; **LAR:** lactate-to-albumin ratio; **PRISM-3:** Pediatric
415 Risk of Mortality-3; **PELOD-2:** Pediatric Logistic Organ Dysfunction-2, **PICU:** Pediatric intensive care
416 unit

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419 **Table 2.** Univariate Logistic Regression for Lactate, Albumin, and LAR

Parameter	Odds Ratio (OR)	Confidence Interval %95 (CI)	p-value
Lactate (for each 1 mmol/L increase)	1.36	1.32 - 1.41	<0.001
Albumin (for each 1 g/dL increase)	0.27	0.24 - 0.31	<0.001
LAR (for each 1-unit increase)	2.86	2.60 - 3.14	<0.001

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Table 3: Comparison of Lactate/Albumin Ratio by Intubation Status (Survivors Only)

Parameter	Non-intubated (n=5,256)	Intubated (n=1,301)	p-value
L/A Ratio (Mean ± SD)	0.59 ± 0.93	0.75 ± 0.69	<0.001
L/A Ratio (Median, Q1-Q3)	0.455 (0.331–0.672)	0.531 (0.358–0.869)	<0.001

Table 4. Univariate odds ratios for LAR predicting mortality within each of the 10 admission-diagnosis subgroups.

Diagnostic subgroup	n	Deaths	Odds Ratio	95% CI	p-value
Trauma-Postoperative Surgery	963	61	5.67	4.02–8.01	<0.001
Neurological	1316	92	3.98	2.99–5.28	<0.001
Gastrointestinal	334	19	3.80	2.18–6.64	<0.001
Musculoskeletal	535	13	3.01	1.68–5.39	<0.001
Endocrinological	419	8	2.82	1.33–6.00	0.00703 *
Cardiovascular	744	136	2.42	1.95–3.01	<0.001
Respiratory	1535	123	2.36	1.80–3.09	<0.001
Hematological	603	160	2.20	1.77–2.73	<0.001
Infectious-Sepsis	266	42	2.10	1.51–2.92	<0.001
Oncological	602	106	1.96	1.48–2.60	<0.001

* Did not reach the Bonferroni-corrected significance threshold ($\alpha = 0.005$)

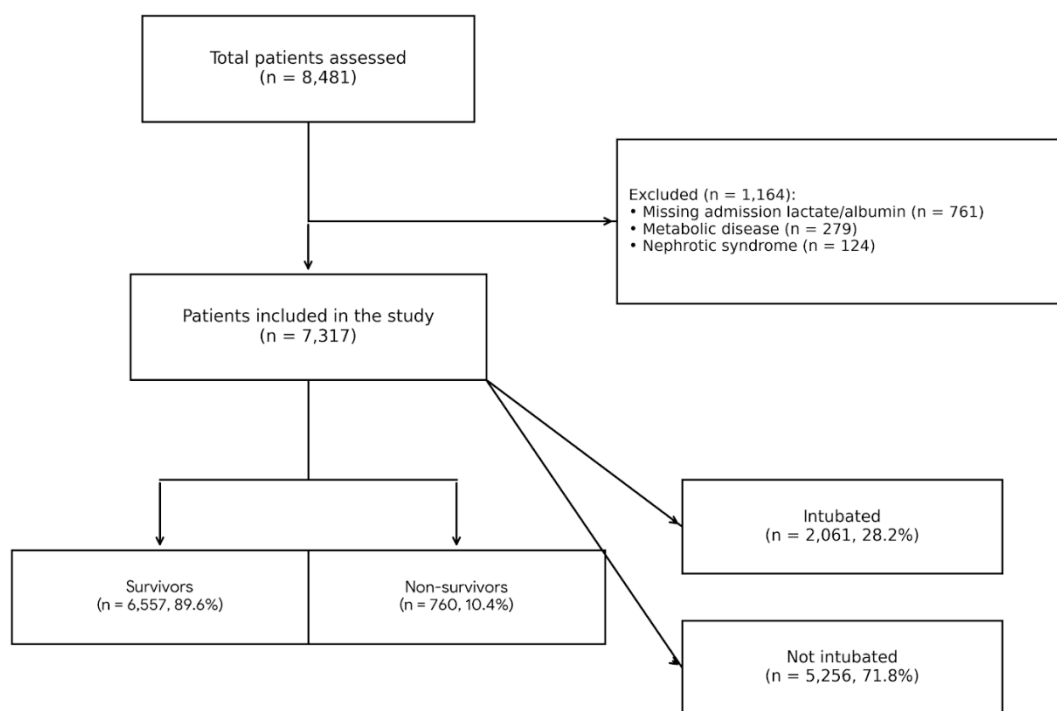


Figure 1. Flow chart of recruitment of the study population

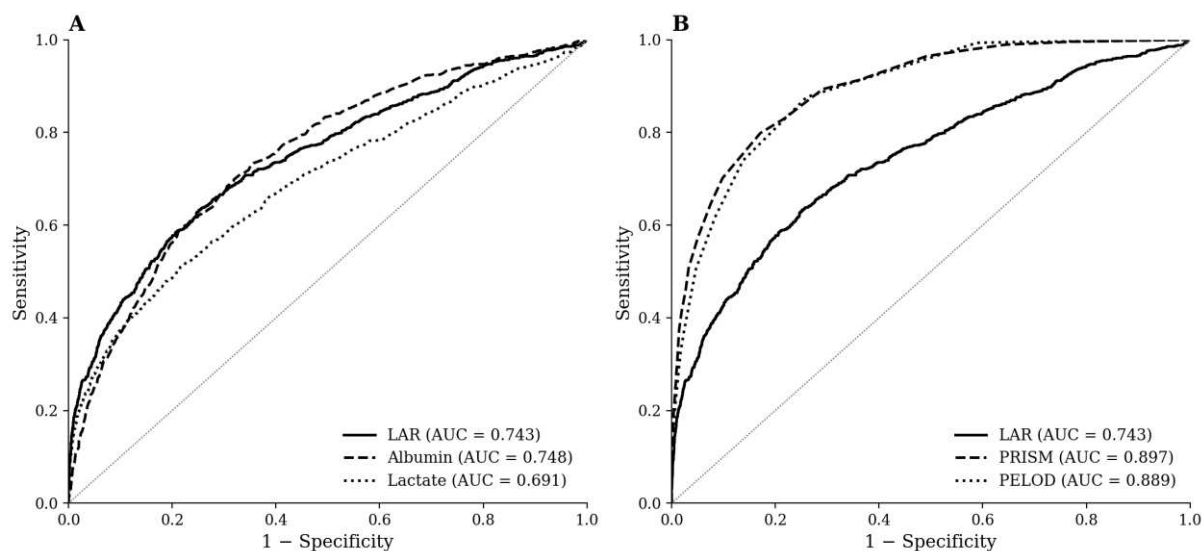


Figure 2. Receiver operating characteristic (ROC) curves for predicting PICU mortality. (A) LAR versus its individual components, lactate and albumin. (B) LAR versus the PRISM and PELOD severity scores.