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Predictive Value of The Lactate-to-Albumin Ratio for 28-Day and 90-Day Mortality in Patients with Sepsis-Induced Coagulopathy

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Abstract: Background/Objectives: To investigate the utility of the lactate-to-albumin ratio (LAR) as a novel clinical risk stratification tool for predicting 28-day and 90-day mortality in patients with sepsis-induced coagulopathy (SIC). Methods: We identified patients meeting Sepsis-3 and International Society on Thrombosis and Haemostasis (ISTH) SIC criteria from the MIMIC-IV database. The cohort was stratified into high-LAR (n=1,343) and low-LAR (n=1,344) groups based on the median value (M=0.805). Baseline characteristics were analyzed, and survival patterns were assessed using Kaplan–Meier analysis and multivariable Cox regression. Restricted cubic splines (RCS) were employed to detect nonlinear relationships, while threshold effect analysis identified specific inflection points. Predictive performance was quantified using the area under the receiver operating characteristic curve (AUC). Results: Baseline and Kaplan–Meier analyses revealed significantly elevated mortality risks in the high-LAR group compared to the low-LAR group (28-day: 50.89% vs 32.39%, $P < 0.001$; 90-day: 64.14% vs 46.91%, $P < 0.001$; log-rank $P < 0.0001$). Multivariable Cox regression confirmed LAR as an independent risk factor for SIC outcomes (28-day: HR=1.11, 95% CI: 1.06–1.16, $P < 0.001$; 90-day: HR=1.12, 95% CI: 1.08–1.17, $P < 0.001$). The AUCs for 28-day and 90-day mortality were 0.64 (95% CI: 0.62–0.66) and 0.63 (95% CI: 0.61–0.65), respectively. RCS and threshold analyses indicated a nonlinear association; below an inflection point of approximately 1.5, mortality risk increased progressively with rising LAR (28-day: LAR < 1.56, HR=1.42, P=0.002; 90-day: LAR < 1.50, HR=1.42, $P < 0.001$). Conclusions: LAR is an independent risk factor for both 28-day and 90-day mortality in patients with SIC and serves as a practical tool for early clinical risk stratification.

Keywords: Sepsis-induced Coagulopathy; Lactate-to-albumin Ratio; Mortality; Prognosis; Retrospective Study.

1. Introduction

Sepsis is characterized by a dysregulated host response to infection, leading to life-threatening organ dysfunction. It remains a leading cause of mortality in intensive care units (ICUs) worldwide. Epidemiological estimates suggest sepsis accounts for approximately 48.9 million cases and 11 million deaths annually, representing a mortality rate of roughly 22.5% [1].

A substantial proportion of sepsis cases, ranging from 50% to 70%, are complicated by sepsis-induced coagulopathy (SIC) [2]. In 2019, the Scientific and Standardization Committee of the International Society on Thrombosis and Haemostasis (ISTH) developed a scoring system for SIC, which integrates the SOFA score, prothrombin time, and platelet count into its diagnostic criteria [3]. The pathophysiology of SIC involves excessive activation of coagulation, impaired anticoagulant pathways, and dysfunctional fibrinolysis, ultimately leading to the consumption of clotting factors [4]. Without timely intervention, mortality rates can reach 23.1% [5] Consequently, early risk stratification is crucial for identifying high-risk patients and informing clinical monitoring and resource allocation. While previous studies have correlated SIC severity with age, SOFA score, fibrinogen, PT-INR, and lactate levels [6][7], assessing these parameters individually can delay intervention. Therefore, there is an urgent need for a simple, cost-effective, and readily available marker to complement existing severity scores for prognostic assessment in SIC.

Elevated lactate, a byproduct of anaerobic glycolysis, is a hallmark of tissue hypoperfusion and cellular hypoxia. It is a well-established predictor of organ dysfunction and death in critically ill patients [8][9]Nevertheless, the reliability of lactate as a standalone predictor is limited because levels can be influenced by non-hypoxic conditions, such as hepatic impairment and certain medications [10][11].

Albumin is a multifunctional plasma protein synthesized by the liver, playing roles in antioxidant defense, pH buffering, and transport [12][13]. However, serum albumin levels may be suppressed by factors independent of chronic pathology, such as malnutrition and persistent inflammatory states [14], thereby reducing their specificity for prognostic assessment.

The Lactate-to-Albumin Ratio (LAR) integrates these two parameters. Previous studies have confirmed the predictive significance of LAR in conditions such as sepsis with acute kidney injury, sepsis with cirrhosis, and severe pancreatitis [15][16][17]. However, SIC represents a distinct sepsis phenotype characterized by dysregulated coagulation, endothelial injury, and microcirculatory dysfunction. These features may influence lactate production/clearance and albumin kinetics, potentially modifying the prognostic performance of perfusion- and inflammation-related biomarkers when SIC is present. However, the prognostic relevance of LAR specifically within the SIC population remains unclear. This study aims to investigate the association between admission LAR and short-term all-cause mortality among patients with SIC.

2. Materials and Methods

2.1. Data Source

Data were derived from the MIMIC-IV version 3.0 database. This publicly available repository contains de-identified electronic health records from over 450,000 hospital admissions at the Beth Israel Deaconess Medical Center (BIDMC) between 2008 and 2019. As an anonymized database, it was exempted from ethical approval per the review board protocol. One author (Dong Chufan; Record ID: 58063775) completed the required CITI Program courses to access the database.

2.2. Study Population

We identified adult patients (≥18 years) with a primary diagnosis of sepsis based on Sepsis-3 criteria (documented infection and SOFA score ≥2) using ICD-9 and ICD-10 codes. From an initial cohort of 28,932 sepsis patients, we applied the ISTH SIC criteria (SIC score ≥4 calculated from initial ICU data). Exclusion criteria were: (1) ICU stay <24 hours; (2) pregnancy; (3) congenital coagulopathy; (4) active malignancy; and (5) missing baseline data for lactate, albumin, PT-INR, or platelets. The final analysis included 2,687 patients (0).

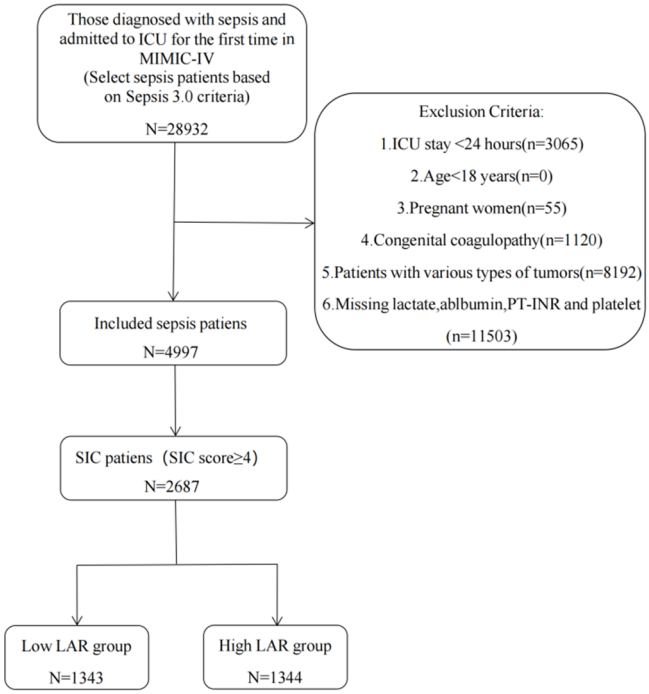


Figure 1. Flowchart of participant selection from the MIMIC-IV database.

2.3. Data Extraction

LAR was calculated by dividing the first serum lactate measurement (mmol/L) by the first serum albumin measurement (g/L) obtained upon ICU admission. Extracted variables included: demographics (age, race, sex); vital signs (heart rate, oxygen saturation, respiratory rate); severity scores (SOFA, APS III, GCS); clinical interventions (invasive mechanical ventilation, vasopressor use, corticosteroid use, thrombolytics, antiplatelet agents); comorbidities (congestive heart failure, stroke, acute renal failure, diabetes mellitus, chronic renal insufficiency, hepatitis); and laboratory assessments (complete blood counts, electrolytes, liver and kidney function tests, coagulation profiles). Survival data included survival time and 28-day/90-day mortality status. Data retrieval utilized Structured Query Language (SQL) within PostgreSQL (v13.7.1) and Navicat Premium (v15.0).

2.4. Grouping and Outcomes

Patients were stratified into low-LAR and high-LAR groups based on the median LAR value of the cohort. The primary endpoints were 28-day and 90-day all-cause mortality.

2.5. Handling of Missing Data and Outliers

A tiered strategy was adopted for managing missing data: exclusion for >15% missingness, multiple imputation ('mice' package) for 5-15%, and mean imputation for <5%. This workflow was implemented in R (v4.3.0).

2.6. Statistical Analysis

Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Normally distributed data are presented as mean ± SD and compared using t-tests or ANOVA. Non-normally distributed variables are presented as median (IQR) and compared using Mann-Whitney U or Kruskal-Wallis tests. Categorical variables are summarized as percentages and compared using Chi-square or Fisher's exact tests.

We incorporated all variables reaching the significance threshold ($P<0.05$) in univariate analysis into univariate Cox proportional hazards models. We selected variables with $P < 0.05$ from univariate screening for inclusion in multivariable Cox models, applying progressive adjustments in three sequential stages. Model 1 was unadjusted. Model 2 was adjusted for demographic characteristics and severity scores. Model 3 was further adjusted for vital signs, laboratory parameters, comorbidities, and clinical interventions.

To evaluate predictive accuracy, we calculated the concordance index (C-index) and the area under the receiver operating characteristic curve (AUC). Restricted cubic splines (RCS) were used to explore non-linear associations, followed by threshold effect analysis. Subgroup analyses were performed to assess interaction effects. All analyses were conducted using R (v4.3.0) and Zstats (www.zstats.net), with a two-tailed P-value < 0.05 considered statistically significant.

3. Results

3.1. Baseline Characteristics

Analysis of 2,687 patients with SIC stratified by median LAR (0.805) confirmed significant differences between the high (n=1,343) and low (n=1,344) LAR groups (Table 1). The cohort comprised 1,079 females (40.16%) and 1,608 males (59.84%), with a mean age of 65 years. Overall 28-day and 90-day mortality rates were 41.64% (n=1,119) and 55.53% (n=1,492), respectively. The high-LAR group exhibited significantly worse survival outcomes compared to the low-LAR group (28-day: 50.89% vs. 32.39%, $P<0.001$; 90-day: 64.14% vs. 46.91%, $P<0.001$). Although the high-LAR group was slightly younger ($P<0.001$), they presented with greater disease severity, indicated by higher heart rate, respiratory rate, SOFA score, and APS III score (all $P<0.01$). Laboratory findings showed pronounced metabolic disturbances in the high-LAR group, including elevated lactate and anion gap, alongside lower albumin levels (all $P<0.001$). Significant differences were also observed in white blood cell count, renal and liver function markers, and coagulation profiles.

The high-LAR group had a higher prevalence of acute kidney injury and hepatitis (P<0.001) and required more frequent use of vasopressors, anticoagulants, corticosteroids, and thrombolytics (P<0.001). See 0 for details.

Table 1. Baseline characteristics

Variables	Total (n = 2687)	Lower group (n = 1343)	Higher group (n = 1344)	P
LAR	0.80 (0.52, 1.43)	0.52 (0.40, 0.65)	1.43 (1.03, 2.21)	<.001
Demographic variables				
Age, years	65.07 ± 16.27	66.36 ± 15.72	63.79 ± 16.71	<.001
Gender, n(%)				0.795
Female	1079 (40.16)	536 (39.91)	543 (40.40)	
Male	1608 (59.84)	807 (60.09)	801 (59.60)	
Race, n(%)				<.001
White	1649 (61.37)	870 (64.78)	779 (57.96)	
Black	213 (7.93)	114 (8.49)	99 (7.37)	
Others	825 (30.70)	359 (26.73)	466 (34.67)	
Vital signs				
Heart rate, beats/min	92.00 (79.00, 108.00)	88.00 (75.00, 104.00)	96.00 (82.00, 113.00)	<.001
Respiratory rate, times/min	20.00(16.00, 24.00)	19.00(16.00, 23.00)	20 .00(16.00, 25.00)	0.005
SPO2, %	98.00 (94.00, 100.00)	98.00 (95.00, 100.00)	98.00 (94.00, 100.00)	0.780
Scores				
SOFA	9 (6.00, 11.50)	7.00 (5.00, 10.00)	10.00 (7.00, 13.00)	<.001
APACHE III	62.00 (48.00, 79.00)	55.00 (43.00, 69.00)	71.00 (55.00, 89.00)	<.001
GCS	15.00 (13.00, 15.00)	15.00 (13.00, 15.00)	15.00 (13.00, 15.00)	0.799
Laboratory parameters				
White blood cell, 10^9/L	12.00 (8.10, 17.60)	10.90 (7.50, 15.70)	13.30 (8.70, 19.22)	<.001
Red blood cells, 10^9/L	3.22 (2.71, 3.82)	3.27 (2.75, 3.83)	3.18 (2.65, 3.81)	0.052
Platelet count, 10^9/L	129.00 (83.00, 195.00)	133.00 (88.00, 203.50)	124.00 (79.00, 186.00)	<.001
Creatinine, mg/dL	1.50 (0.90, 2.50)	1.40 (0.90, 2.40)	1.60 (1.00, 2.60)	0.016
Blood urea nitrogen, mg/dL	30.00 (18.00, 51.00)	31.00 (18.00, 53.00)	30.00 (18.00, 49.25)	0.457
Total bilirubin, mg/dL	1.30 (0.60, 3.70)	1.00 (0.50, 2.80)	1.70 (0.70, 4.50)	<.001
Aspartate aminotransferase, U/L	65.00 (33.00, 174.00)	48.00 (28.00, 105.00)	91.00 (41.00, 309.50)	<.001
Alanine aminotransferase, U/L	34.00 (19.00, 93.00)	29.00 (17.00, 62.00)	44.00 (22.00, 163.25)	<.001
Lactate, mmol/L	2.30 (1.50, 3.75)	1.50 (1.20, 1.90)	3.70 (2.80, 5.90)	<.001
Albumin, g/dL	2.90 (2.40, 3.30)	3.00 (2.60, 3.40)	2.60 (2.20, 3.10)	<.001
Prothrombin time, s	19.10 (16.40, 25.40)	18.30 (15.90, 24.40)	20.00 (16.90, 26.30)	<.001
Activated partial thromboplastin time, s	37.10 (31.30, 48.95)	35.40 (30.30, 45.25)	39.55 (32.60, 53.02)	<.001
Potassium, mmol/L	4.20 (3.70, 4.70)	4.10 (3.70, 4.60)	4.30 (3.70, 4.90)	0.001
Sodium, mmol/L	138.00 (134.00, 141.00)	138.00 (135.00, 141.00)	138.00 (134.00, 142.00)	0.502
Chloride, mmol/L	103.00 (98.00, 109.00)	103.00 (98.00, 108.00)	104.00 (98.00, 109.00)	0.421
Calciumtotal, mmol/L	8.10 (7.50, 8.70)	8.20 (7.70, 8.80)	8.00 (7.30, 8.60)	<.001
Glucose, mg/dL	130.00 (104.00, 174.50)	126.00 (104.00, 160.50)	137.00 (104.00, 189.25)	<.001
Aniongap, mmol/L	16.00 (13.00, 19.00)	15.00 (12.00, 18.00)	17.00 (14.00, 21.00)	<.001
Comorbidities				
Heart Failure, n(%)				<.001
No	1687 (62.78)	766 (57.04)	921 (68.53)	
Yes	1000 (37.22)	577 (42.96)	423 (31.47)	
Acute Renal Failure, n(%)				<.001
No	883 (32.86)	491 (36.56)	392 (29.17)	
Yes	1804 (67.14)	852 (63.44)	952 (70.83)	
Hepatitis, n(%)				<.001
No	2292 (85.30)	1185 (88.24)	1107 (82.37)	
Yes	395 (14.70)	158 (11.76)	237 (17.63)	
Chronic Kidney Disease, n(%)				<.001
No	2021 (75.21)	962 (71.63)	1059 (78.79)	

Variables	Total (n = 2687)	Lower group (n = 1343)	Higher group (n = 1344)	P
Yes	666 (24.79)	381 (28.37)	285 (21.21)	
Stroke, n(%)				0.015
No	2473 (92.04)	1219 (90.77)	1254 (93.30)	
Yes	214 (7.96)	124 (9.23)	90 (6.70)	
Diabetes Mellitus, n(%)				0.069
No	1799 (66.95)	877 (65.30)	922 (68.60)	
Yes	888 (33.05)	466 (34.70)	422 (31.40)	
Treatments				
Ventilation, n(%)				0.805
No	278 (10.35)	137 (10.20)	141 (10.49)	
Yes	2409 (89.65)	1206 (89.80)	1203 (89.51)	
Vasoactiveagents, n(%)				<.001
No	1024 (38.11)	605 (45.05)	419 (31.18)	
Yes	1663 (61.89)	738 (54.95)	925 (68.82)	
Rxglucocorticoids, n(%)				<.001
No	1999 (74.40)	1040 (77.44)	959 (71.35)	
Yes	688 (25.60)	303 (22.56)	385 (28.65)	
Anticoagulants, n(%)				<.001
No	1255 (46.71)	563 (41.92)	692 (51.49)	
Yes	1432 (53.29)	780 (58.08)	652 (48.51)	
Antiplatelets, n(%)				<.001
No	1778 (66.17)	836 (62.25)	942 (70.09)	
Yes	909 (33.83)	507 (37.75)	402 (29.91)	
Clinical Outcomes				
Survival day	46.58 (11.09, 288.00)	99.88 (17.98, 398.28)	25.23 (6.97, 185.22)	<.001
Death within hosp 28days, n(%)				<.001
No	1568 (58.36)	908 (67.61)	660 (49.11)	
Yes	1119 (41.64)	435 (32.39)	684 (50.89)	
Death within hosp 90days, n(%)				<.001
No	1195 (44.47)	713 (53.09)	482 (35.86)	
Yes	1492 (55.53)	630 (46.91)	862 (64.14)	

LAR: lactate to albumin ratio; SOFA: sequential organ failure assessment; APACHE III: acute physiology and chronic health evaluation (APACHE) III; GCS: Glasgow coma scale

3.2. LAR as an Independent Risk Factor for short-term Mortality

Univariate Cox regression showed that each unit increase in LAR was associated with a significantly increased risk of death at 28 days (HR=1.28, 95% CI: 1.24–1.32) and 90 days (HR=1.27, 95% CI: 1.24–1.31). In the fully adjusted model (Model 3), LAR remained an independent predictor of mortality (28-day: HR=1.11, 95% CI: 1.06–1.16; 90-day: HR=1.12, 95% CI: 1.08–1.17; both P < 0.001). Detailed results are shown in 0, Supplementary Table S1 and Supplementary Table S2.

Table 2. Cox proportional hazard ratios (HR) for all-cause mortality

Variables	Model1	P	Model2	P	Model3	P
	HR (95%CI)		HR (95%CI)		HR (95%CI)	
28-day all-cause mortality						
LAR	1.28 (1.24 ~ 1.32)	<.001	1.16 (1.12 ~ 1.20)	<.001	1.11 (1.06 ~ 1.16)	<.001
90-day all-cause mortality						
LAR	1.27 (1.24 ~ 1.31)	<.001	1.15 (1.12 ~ 1.20)	<.001	1.12 (1.08 ~ 1.17)	<.001

Model 1: Unadjusted;

Model 2: Adjusted for age, gender, race, SOFA, APACHE III.

Model 3: Adjusted for age, gender, race, SOFA, APACHE III, heart rate, respiratory rate, white blood cell, creatinine, aspartate aminotransferase, alanine aminotransferase, prothrombin time, activated partial thromboplastin time, potassium, glucose, and aniongap, acute renal failure, hepatitis, chronic kidney disease, vasoactiveagents, rxglucocorticoids, anticoagulants, antiplatelets.

3.3. Kaplan-Meier Survival Analysis

Kaplan-Meier curves (0) demonstrated a clear survival disadvantage for the high-LAR group compared to the low-

LAR group for both 28-day and 90-day outcomes (log-rank test, P < 0.0001).

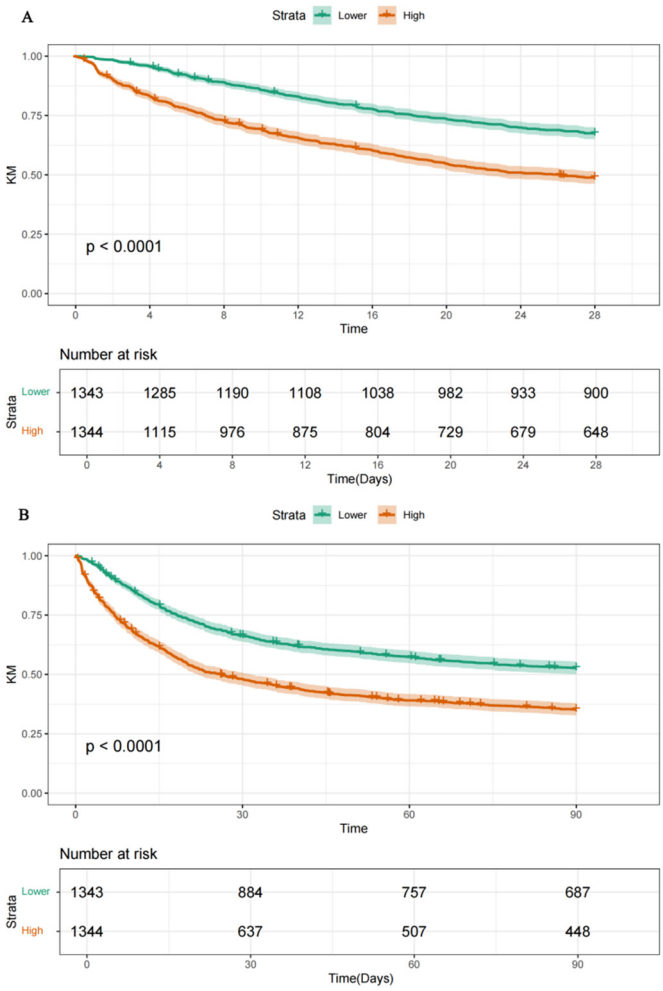


Figure 2. Kaplan–Meier survival curves stratified by LAR groups. Curves show the cumulative incidence of (A) 28-day and (B) 90-day all-cause mortality. Patients in the lower LAR group showed significantly reduced mortality rates compared to the higher LAR group (log-rank tests, P < 0.0001). LAR, lactate-to-albumin ratio.

3.4. Model Evaluation

ROC analysis was applied to quantify predictive ability (0, Supplementary Table S3). For predicting 28-day mortality, the AUC values were: LAR 0.64 (95% CI: 0.62-0.66), lactate 0.63 (95% CI: 0.61-0.66), albumin 0.54 (95% CI: 0.52-0.56), and SOFA 0.66 (95% CI: 0.64-0.68). The predictive performance of LAR for 90-day mortality was consistent (AUC=0.63, 95% CI: 0.61-0.65). The models exhibited acceptable discrimination, with C-indices of 0.70 (28-day, 95% CI: 0.68-0.72) and 0.68 (90-day, 95% CI: 0.67-0.70), as shown in Supplementary Figure S1.

3.5. Dose-Response Relationship and Threshold Effect Analysis

Restricted cubic spline analyses suggested a nonlinear association between LAR and mortality at 28 days (P for nonlinearity = 0.006) and 90 days (P for nonlinearity = 0.007), with an apparent change in slope (0). Threshold effect analyses identified inflection points at LAR = 1.56 for 28-day mortality and LAR = 1.50 for 90-day mortality (likelihood ratio test P = 0.019 and 0.027, respectively). In piecewise models(0), below the inflection point (LAR < 1.5), each 1-unit increase in LAR was associated with a 42% higher hazard of mortality (28-day: HR 1.42, 95% CI 1.13–1.78; 90-day: HR 1.42, 95% CI 1.16–1.72). Beyond the inflection point (LAR ≥ 1.50), the association with mortality appeared to reach a plateau (28-day: HR = 1.02, P = 0.613; 90-day: HR = 1.03, P = 0.383).

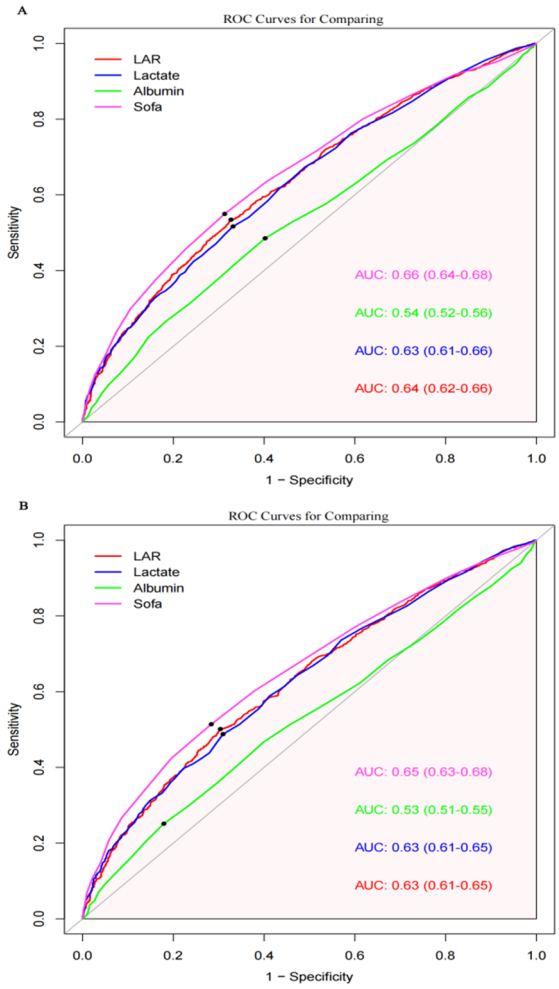


Figure 3. ROC curves of LAR predicting short-term mortality. Receiver operating characteristic curves assessing the predictive capability of LAR for (A) 28-day mortality (AUC = 0.64; 95% CI: 0.62–0.66) and (B) 90-day mortality (AUC = 0.63; 95% CI: 0.61–0.65). AUC, area under the curve; CI, confidence interval.

The data have been adjusted for all of the factors included in Model 3 in Table 2. HR hazard ratio, CI confidence interval, LAR lactate to albumin ratio.

3.6. Subgroup Analysis

Subgroup analyses were performed across predefined strata (age, race, acute renal failure, hepatitis, stroke, and diabetes; 0). No statistically significant interaction was observed across these subgroups (all P for interaction > 0.05), supporting a generally consistent association between higher LAR and mortality.

4. Discussion

Our findings indicate that the admission lactate-to-albumin ratio (LAR) is independently associated with short-term mortality in patients with SIC. After multivariable adjustment, each 1-unit increase in LAR was associated with an 11% higher hazard of 28-day mortality and a 12% higher hazard of 90-day mortality. We also observed a nonlinear association in spline analyses, with an apparent change in the risk slope

around LAR ≈ 1.5.

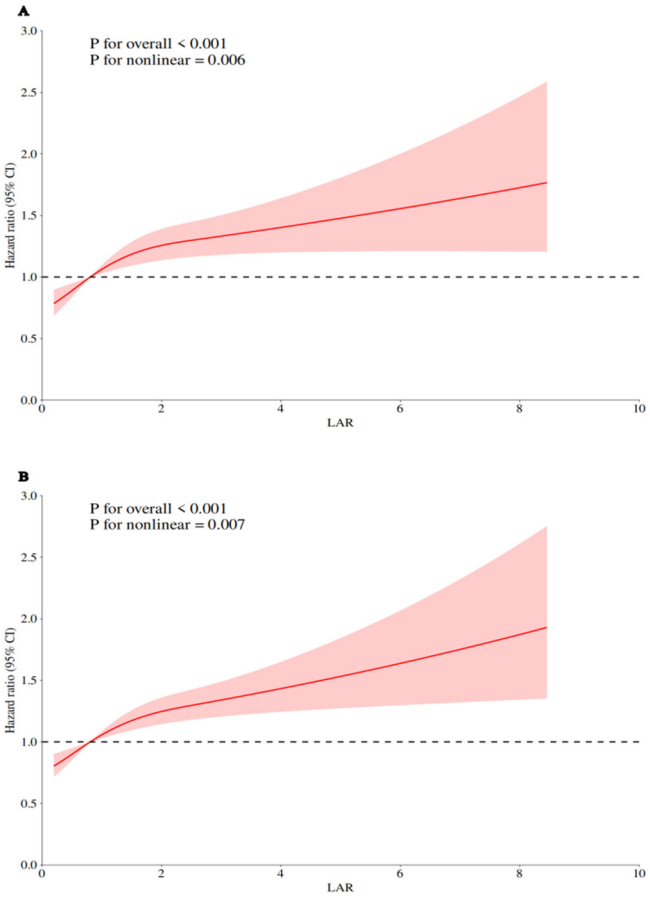


Figure 4. Restricted cubic spline analysis of LAR and mortality. The plots depict the non-linear relationship between continuous LAR and (A) 28-day all-cause mortality (P for nonlinearity = 0.006) and (B) 90-day all-cause mortality (P for nonlinearity = 0.007). The solid line represents the hazard ratio, and the shaded area indicates the 95% confidence interval.

Table 3. Threshold effect analysis of LAR on 28-day and 90-day mortality in SIC patients

Threshold of LAR	28-day all-cause mortality			90-day all-cause mortality	
	HR (95% CI)	P value		HR (95% CI)	P value
Model 1 Fitting model by standard linear regression	1.09 (1.05 - 1.15)	<.001		1.10 (1.05 ~ 1.15)	<.001
Model 2 Fitting model by two-piecewise linear regression					
Inflection point	1.56			1.50	
<1.56	1.42 (1.13 - 1.78)	0.002	<1.50	1.42 (1.16 - 1.72)	<.001
≥1.56	1.02 (0.95 - 1.09)	0.613	≥1.50	1.03 (0.96 - 1.10)	0.383
P for likelihood test		0.019			0.027

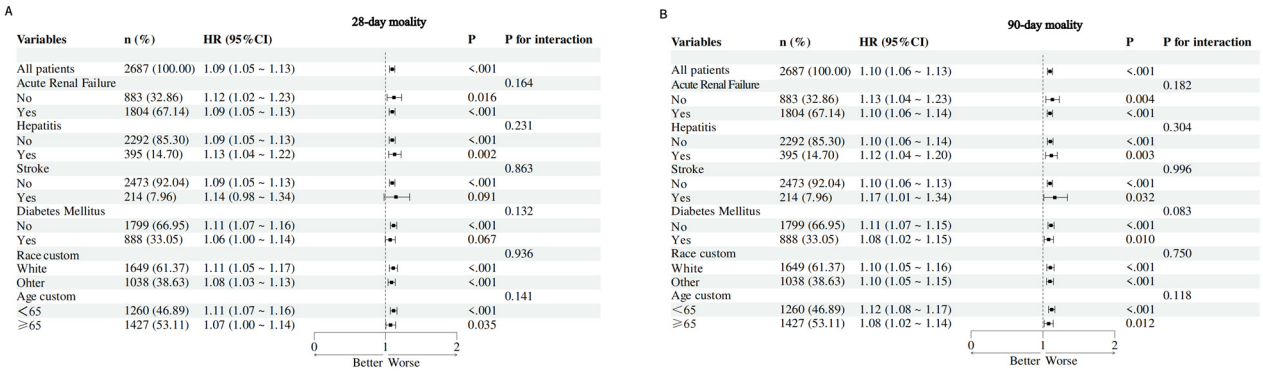


Figure 5. Subgroup analyses of LAR for predicting mortality. Forest plots display the stratified analyses for (A) 28-day and (B) 90-day all-cause mortality. No significant interactions were observed across subgroups (all P for interaction > 0.05), indicating a consistent correlation between LAR and mortality.

Current evidence has identified various prognostic factors for SIC, ranging from pharmacologic agents like heparin [18], dexmedetomidine [19], and statins [20] to clinical signs such as body temperature [21]. However, the role of composite biomarkers remains to be fully elucidated. While LAR is a well-established predictor of outcomes in general sepsis [22], liver injury [23], and ARDS [24], its specific prognostic value for SIC patients has not yet been determined. This study fills this gap, offering a novel contribution to the existing literature.

In line with the 2021 Surviving Sepsis Campaign International Guidelines, which highlight serum lactate as a key biochemical indicator of hypoxia, tissue perfusion, and dysfunction [25], our findings are consistent with research by Liu et al. [26] and He et al. [27]. Notably, lactate concentrations can be confounded by liver dysfunction or medication effects [28], limiting its independent predictive value. Simultaneously, serum albumin is influenced by nutritional status and persistent inflammation [29], with synthesis decreasing during liver disease [30] and clearance increasing during sepsis [31]. Consequently, relying on either marker in isolation is often insufficient. This underscores the importance of the LAR, which integrates perfusion (lactate) and inflammation/nutrition (albumin) to mitigate the limitations of single-parameter assessment.

Recent studies further demonstrate the prognostic relevance of LAR in various acute conditions. Kokkoris et al. found LAR to be a more dependable COVID-19 severity predictor than lactate alone [32], while investigations in sepsis [33] and post-cardiac arrest cohorts [34] also reported enhanced predictive accuracy with the composite index.

Building upon this evidence, we confirmed the independent prognostic value of LAR in a large cohort of 2,687 SIC patients. We identified an inflection point around LAR ≈ 1.5, below which mortality risk increases sharply with the ratio, while the risk plateaus at higher values. However, several limitations warrant consideration. As a single-center retrospective analysis, these findings require validation through multicenter prospective trials. Furthermore, relying on admission measurements precludes the assessment of dynamic LAR trajectories, and unmeasured confounders—such as pre-ICU fluid resuscitation or exogenous albumin administration—may have influenced baseline levels.

While LAR demonstrated slightly lower discrimination than the SOFA score, its simplicity and cost-effectiveness make it a valuable complementary tool for rapid risk stratification. These findings provide a robust foundation for future research to evaluate whether longitudinal LAR trends or their underlying physiological processes are more closely linked to SIC outcomes.

5. Conclusion

This study establishes the admission lactate-to-albumin ratio (LAR) as an independent predictor of 28-day and 90-day mortality in patients with sepsis-induced coagulopathy (SIC). The predictive performance of LAR is slightly superior to that of lactate or albumin alone. Given its cost-effectiveness, simplicity, and efficiency, LAR holds promise as an early risk-stratification tool for this high-risk population.

Supplementary Materials

Table S1: Univariate analysis of 28-day motality; Table S2: Univariate analysis of 90-day motality; Table S3: Diagnostic performance of LAR and other indicators for predicting short-term mortality; Figure S1: Forest plots of C-indices for adjusted multivariate models. Detials in https://rpubs. com/yang-yonggui/ALR_Supplementary Materials.

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