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Original Article

The Role of Lactate Albumin Ratio as a Prognostic Factor in Sepsis

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ABSTRACT

Background: Sepsis as well as septic shock remain major causes of the intensive care unit (ICU) mortality. The lactate-to-albumin ratio (LAR), a simple biochemical index, has been proposed as a novel prognostic biomarker. We aimed in this research to evaluate the prognostic performance of LAR for ICU mortality in adult patients who had sepsis and septic shock and compare it with established known scoring systems: Sequential Organ Failure Assessment (SOFA), and Acute Physiology and Chronic Health Evaluation II(APACHE II).

Methods: This prospective cohort research was performed on 74 patients in the medical ICU. Demographics, comorbidities, laboratory values, and severity scores were collected within 24 hours of admission. LAR was evaluated using lactate (mmol/L) divided by albumin (g/dL). Outcomes included ICU mortality, organ failure, and need for organ support.

Results: Non-survivors had higher lactate (3.0 vs 2.35 mmol/L, p=0.001), LAR (1.3 vs 0.9, p=0.001), bilirubin (0.70 vs 0.42 mg/dL, p=0.027), SOFA (7 vs 4, p=0.001), and APACHE II scores (26.0 vs 16.8, p=0.001). They required more vasopressors (65.8% vs 13.9%, p=0.001) and ventilation (21.1% vs 0%, p=0.004). LAR demonstrated fair predictive ability (AUC 0.74, cutoff 1.11, sensitivity 71%, specificity 75%), comparable to lactate (AUC 0.78), but inferior to SOFA (AUC 0.88) and APACHE II (AUC 0.86). In multivariate analysis, SOFA remained the only independent predictor of mortality (OR 2.59, 95% CI 1.52–4.39, p=0.0004). Elevated LAR correlated positively with bilirubin, vasopressor and ventilator use, cardiovascular failure, and septic shock (all $p<0.05$). Patients with LAR >1.11 had significantly worse neurological status (median GCS 11 vs 14, p=0.001) and higher rates of multiorgan failure.

Conclusion: LAR is a simple, inexpensive, and readily available biomarker that provides moderate predictive value for mortality in septic ICU patients, reflecting both metabolic stress and systemic inflammation. While it does not outperform SOFA or APACHE II, its ease of calculation makes it a valuable complementary tool, especially in resource-limited settings.

Keywords: Lactate Albumin Ratio, Prognostic Factor, Sepsis.

INTRODUCTION

Sepsis is a life-threatening syndrome of infection-induced organ dysfunction and remains a major global health problem, causing high morbidity and mortality worldwide [1,2]. Despite advances in critical care, early and reliable prediction of patient outcomes continues to be challenging [3].

Several approaches have been developed to improve prognostication. Widely used scoring systems such as the Sequential Organ Failure Assessment (SOFA) and the Acute Physiology and Chronic Health

Evaluation II (APACHE II) provide valuable risk estimates [4,5]. However, these scores require numerous clinical and laboratory variables and may not always be immediately available during the first hours of sepsis management [6]. Individual biomarkers—including serum lactate and serum albumin—have also been investigated, but each reflects only a single aspect of the complex pathophysiology [7,8]. To integrate information on both tissue hypoperfusion (lactate) and nutritional or inflammatory status (albumin), the lactate-to-albumin ratio (LAR) has been proposed as a simple, readily obtainable marker [9,10]. We hypothesized that admission LAR could provide early prognostic information and complement established severity scores in septic patients [11]. Accordingly, this study evaluates the prognostic value of LAR for predicting 28-day intensive care unit (ICU) mortality and explores its relationship with established clinical scores and organ dysfunction [12].

Although individual biomarkers such as lactate and albumin are widely used, their predictive accuracy is limited when considered separately. The lactate-to-albumin ratio has emerged as a potentially valuable prognostic marker in diverse clinical scenarios; however, its application in directing therapeutic decisions among septic patients in the intensive care unit remains insufficiently investigated. Additional research is warranted to confirm its consistency as a predictor of mortality across varying cohorts and clinical environments; therefore, this research aimed to evaluate whether the LAR could serve as a reliable indicator of mortality in patients admitted to the ICU with sepsis or septic shock.

METHODS

This prospective cohort study was performed in the Medical Intensive Care Unit (MICU) of the Internal Medicine Department at Zagazig University Hospitals. The study period lasted six months, from April 2024 to October 2024. A total of 74 adult patients admitted to the MICU having a diagnosis of sepsis or septic shock were enrolled. Patient selection followed specific inclusion and exclusion criteria to ensure a homogeneous study population.

After approval from the Zagazig University Institutional Review Board (ZU-IRB#149/19-March-2024), written informed consent was obtained from each participant or their legal representative before enrollment at admission. All procedures adhered to the ethical principles outlined in the Declaration of Helsinki.

A target sample size of 74 patients was calculated using a single-proportion formula for an anticipated mortality of 50% among septic ICU patients [2]. With a confidence level of 95% and a margin of error of 12%, the minimum sample required was 68; we enrolled 74 patients to account for potential attrition.

The primary study question was: Does the admission lactate-to-albumin ratio (LAR) predict 28-day ICU mortality in adult sepsis or septic shock patients? Primary objective: To determine whether LAR on admission is an independent predictor of 28-day ICU mortality. Secondary objectives: (1) To compare the prognostic accuracy of LAR with serum lactate, SOFA score, and APACHE II score; (2) To explore associations between LAR and organ-support requirements and organ-failure patterns.

Eligible participants included adults aged 18 years or above who fulfilled the Sepsis-3 definition of sepsis, characterized by a suspected or confirmed infectious process and an acute elevation of two or more points in the Sequential Organ Failure Assessment (SOFA) score [9].

Exclusion criteria included patients younger than 18 years [10], those with liver cirrhosis

identified through clinical history, laboratory investigations, or imaging [11], and individuals with nephrotic syndrome, confirmed by the presence of significant proteinuria and hypoalbuminemia [12]. Patients with chronic kidney disease, defined as a persistent reduction in estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m² for at least three months, were also excluded [13]. Additionally, cases of non-septic shock—such as hypovolemic, cardiogenic, or anaphylactic shock—verified by clinical and diagnostic evaluation, were not eligible [14]. Finally, patients lacking valid informed consent were excluded from enrollment [15].

Sepsis was diagnosed when infection was accompanied by life-threatening organ dysfunction, indicated by an increase of ≥2 points in the SOFA score [9]. Septic shock was defined as persistent hypotension unresponsive to fluids, requiring vasopressors to maintain MAP ≥65 mmHg and a lactate level >2 mmol/L [9].

Data Collection

Upon admission, detailed demographic information and comorbidities (like ischemic heart disease, diabetes, hypertension, and COPD) were recorded. Clinical assessments included vital signs, neurological status [Glasgow Coma Scale (GCS)], and a standard 12-lead ECG. Imaging investigations included chest CT for suspected pulmonary infections and abdominal ultrasound for intra-abdominal or urinary sources.

Laboratory Investigations

Venous blood samples (10 mL) were obtained under sterile conditions within the first 24 hours of ICU admission. Biochemical parameters, including AST, ALT, albumin, total protein, urea, creatinine, and fasting glucose, were measured using a MICROLAB 300 automated analyzer. Complete blood counts were performed with a MINDRAY PC 2800 hematology analyzer. Serum lactate was assessed in fluoride-containing tubes to prevent glycolysis, employing the lactate oxidase enzymatic method. Additional investigations comprised C-reactive protein (CRP), coagulation profile, Prothrombin time (PT), partial thromboplastin time (PTT), International Normalized Ratio (INR), serum electrolytes (Na, K), arterial blood gases, and urinalysis, while microbiological cultures were collected as clinically indicated. LAR was obtained as the quotient of serum lactate (mmol/L) over serum albumin (g/dL).

The primary exposure variable was the LAR. The primary outcome was ICU mortality within 28 days. Secondary outcomes included the development of multiorgan dysfunction, defined as an increase in SOFA score by ≥2 points, and the length of ICU stay.

Severity Assessment

Organ dysfunction was evaluated through the Sequential Organ Failure Assessment (SOFA) score [9]. Disease severity and the probability of mortality were further quantified using the APACHE II scoring system, determined within the first 24 hours following ICU admission [14]. To ensure consistency, both scores were computed with pre-validated Excel-based calculators.

Outcome Measures

The primary outcome was 28-day ICU mortality. Secondary outcomes included: need for vasopressors, invasive mechanical ventilation, renal replacement therapy, individual organ failures (respiratory, cardiovascular, renal, hepatic, hematologic, neurologic), length of ICU stay, and correlations between LAR and these clinical parameters.

Statistical analysis

Data analysis was performed with IBM SPSS Statistics v26. Normality was tested with the Shapiro–Wilk test. Normally distributed data were expressed as mean ±

SD and compared using Student's t-test, while skewed variables were reported as median (IQR) and analyzed by Mann–Whitney U. Categorical outcomes were given as frequencies (%) and compared with Chi-square or Fisher's exact tests. Correlation analyses employed Pearson's r, Spearman's rho, or point-biserial correlation depending on data type. Logistic regression (univariate and multivariate) identified independent predictors of mortality. ROC curves were used to evaluate the prognostic accuracy of SOFA, APACHE II, and LAR. Subgroup analyses assessed outcomes in septic shock cases and across LAR groups using a cutoff of 1.11.

RESULTS

Male patients had significantly higher mortality (47.4% vs. 22.2%, p=0.043), and stroke was significantly associated with higher mortality (55.3% vs. 16.7%, p=0.001). Other comorbidities showed no significant differences. Regarding age, non-survivors were slightly older (median 66 vs. 62.5 years, p=0.109) (Table 1).

Non-survivors had significantly higher serum lactate (3.0 vs. 2.35 mmol/L, p = 0.001) and lactate–albumin ratio (1.3 vs. 0.9, p = 0.001), along with lower platelet counts (p = 0.034) and higher bilirubin (p = 0.027). They also showed higher WBC counts (p = 0.037), SOFA (7.0 vs. 4.0, p = 0.001), and APACHE II scores (26.0 vs. 16.8, p = 0.001). Organ dysfunction and support were more frequent, including vasopressor use (65.8% vs. 13.9%, p = 0.001), ventilator requirement (p = 0.004), respiratory failure (p = 0.0011), cardiovascular failure (p = 0.001), and CNS failure (p = 0.0003). ICU stay was also longer among non-survivors (11.5 vs. 7.0 days, p = 0.001) (Table 2).

Serum lactate (AUC 0.78, cutoff ≥2.7 mmol/L), lactate-to-albumin ratio (AUC 0.74, cutoff ≥1.11), Total SOFA Score (AUC 0.88, cutoff ≥ 7) and APACHE II Score (AUC 0.86, cutoff ≥ 19) demonstrated fair ability for predicting mortality in sepsis, all of them offering a balanced sensitivity (66 %–95%) and specificity (64%–97%). In contrast, serum albumin alone (AUC 0.42) lacked predictive value. The Total SOFA score emerged as the most accurate predictor with an excellent AUC of 0.88 (Figure 1)

Univariate analysis showed significant predictors of mortality, including lower temperature, reduced PaO_2/FiO_2 ratio, higher lactate–albumin ratio, elevated SOFA score, male sex, and stroke history. In multivariate analysis, only the SOFA score remained an independent predictor (OR 2.59, 95% CI: 1.52–4.39, p = 0.0004). The SOFA score is the strongest independent risk factor, with each point increasing mortality odds by 2.6 times (Table 3).

Lactate–albumin ratio showed significant positive correlations with serum bilirubin (ρ = 0.271, p = 0.0194), ventilator use (ρ = 0.389, p = 0.0006), vasopressor use (ρ = 0.347, p = 0.0025), coagulopathy (ρ = 0.271, p = 0.0197), cardiovascular failure (ρ = 0.310, p = 0.0073), septic shock (ρ = 0.347, p = 0.0025), SOFA score (ρ = 0.421, p = 0.0002), and APACHE II score (ρ = 0.395, p = 0.0005), the borderline association with ICU length of stay (ρ=0.208, p=0.0753) further supports its potential prognostic value (Table 4).

Among septic patients, non-survivors had a significantly higher prevalence of prior stroke (p = 0.045) and pneumonia as the infection site in septic shock cases (p = 0.023). Laboratory parameters showed higher serum bilirubin in non-survivors (0.79 vs. 0.30 mg/dL, p = 0.011). Disease severity markers were notably worse in non-survivors, with higher SOFA scores (8.0 vs. 5.0, p = 0.005) and longer ICU stays (10 vs. 7 days, p = 0.046). Non-significant variations were observed in most comorbidities, cultures, or other laboratory values (Table 5).

Patients in the high LAR group (above 1.11) required significantly more organ support, with higher rates of ventilator use (22.9% vs. 0%, p = 0.0053) and vasopressor use (60.0% vs. 23.1%, p = 0.0028) compared to those below the cutoff. They also had worse neurological status, reflected by lower GCS scores (median 11.0 vs. 14.0, p = 0.001). Multiorgan failure was more frequent in the high LAR group, particularly coagulopathy (51.4% vs. 25.6%, p = 0.041), cardiovascular failure (82.9% vs. 56.4%, p = 0.0276), and CNS failure (91.4% vs. 69.2%, p = 0.0374) (Table 6).

Table (1): Demographics and comorbidities among survivors and non survivors (n=74)

Variable	Overall (n=74) Frequency(percentage)	Survivors (n=36) frequency (percentage)	Non-survivors (n=38) frequency (percentage)	p-value
Sex (Male)	26 (35.1%)	8 (22.2%)	18 (47.4%)	0.0433*
Sex (Female)	48 (64.9%)	28 (77.8%)	20 (52.6%)	0.0433*
DM	48 (64.9%)	24 (66.7%)	24 (63.2%)	0.752*
HTN	49 (66.2%)	22 (61.1%)	27 (71.1%)	0.366*
IHD	7 (9.5%)	1 (2.8%)	6 (15.8%)	0.056*
stroke	27 (36.5%)	6 (16.7%)	21 (55.3%)	0.001*
COPD	1 (1.4%)	0 (0%)	1 (2.6%)	0.327*
Malignancy	4 (5.4%)	1 (2.8%)	3 (7.9%)	0.331*
Other comorbidities	8 (10.8%)	3 (8.3%)	5 (13.2%)	0.504*
Age (years)	Median (IQR) 65.0 (55.5-72.0)	Median (IQR) 62.5 (50.5-69.75)	Median (IQR) 66.0 (63.0-73.5)	0.109**

*:Chi-square test; **:Mann-Whitney U test. DM: diabetes mellitus; HTN: hypertension; IHD: ischemic heart disease; COPD: chronic obstructive pulmonary disease.

Table (2): laboratory and clinical parameters among survivors and non survivors (n=74)

Variable	Overall Median (IQR)	Survivors Median (IQR)	Non-survivors Median (IQR)	p-value
CRP	165.0 (90.0-209.0)	185.0 (108.5-210.25)	144.0 (73.5-208.0)	0.166*
Serum Lactate (mmol/L)	2.7 (2.1-3.38)	2.35 (1.8-2.75)	3.0 (2.7-4.0)	0.001*
Serum Albumin (g/dL)	2.48 (2.24-2.84)	2.62 (2.3-2.88)	2.42 (2.16-2.83)	0.215*
Hemoglobin (g/dL)	9.34 (8.56-10.23)	9.5 (8.56-10.26)	9.22 (8.59-10.2)	0.408*
Platelets (x 10^9/L)	172.0 (109.25-253.75)	203.0 (147.5-270.5)	149.5 (87.0-223.0)	0.034*
Serum sodium (mmol/L)	139.0 (132.0-144.75)	141.0 (132.0-146.0)	137.5 (131.25-143.0)	0.341*
Serum potassium (mmol/L)	3.83 (3.26-4.34)	3.89 (3.34-4.34)	3.72 (3.23-4.22)	0.729*
AST (U/L)	32.0 (26.25-38.0)	30.5 (24.75-37.25)	32.0 (27.25-38.0)	0.381*
ALT (U/L)	25.0 (20.0-30.75)	24.5 (19.0-30.0)	25.0 (21.25-31.0)	0.579*
Serum urea (mg/dL)	154.0 (77.25-223.75)	180.0 (107.75-224.25)	137.5 (70.0-222.75)	0.21*

Variable	Overall Median (IQR)	Survivors Median (IQR)	Non-survivors Median (IQR)	p-value
Serum creatinine (mg/dL)	1.19 (0.66-3.05)	1.15 (0.68-2.53)	1.5 (0.62-3.15)	0.837*
Serum Bilirubin (mg/dL)	0.56 (0.36-0.94)	0.42 (0.3-0.8)	0.7 (0.44-1.09)	0.027*
INR	1.1 (1.0-1.4)	1.05 (0.98-1.4)	1.2 (1.0-1.4)	0.207*
Lactate Albumin Ratio	1.11 (0.8-1.45)	0.9 (0.65-1.12)	1.3 (1.01-1.67)	0.001*
WBCS (x 10^9/L)	Mean ± SD 19.98 ± 5.02	Mean ± SD 18.74 ± 4.61	Mean ± SD 21.16 ± 5.16	0.037**
Total SOFA Score	Median (IQR) 5.00 (4.00-7.00)	Median (IQR): 4.00 (3.00-5.00)	Median (IQR): 7.00 (5.00-9.00)	0.001*
APACHE II Score	Mean ± SD 21.54 ± 7.71	Mean ± SD: 16.83 ± 5.44	Mean ± SD: 26.00 ± 6.88	0.001**
Ventilator Use	Frequency (percentage) 8 (10.8%)	frequency (percentage) 0 (0%)	frequency (percentage) 8 (21.1%)	0.004***
Hemodialysis Sessions	9 (12.2%)	5 (13.9%)	4 (10.5%)	0.658***
Vasopressor Use	30 (40.5%)	5 (13.9%)	25 (65.8%)	0.001***
Respiratory failure	36 (48.6%)	10 (27.8%)	26 (68.4%)	0.0011***
Coagulopathy	28 (37.8%)	9 (25.0%)	19 (50.0%)	0.048***
Liver failure	13 (17.6%)	5 (13.9%)	8 (21.1%)	0.614***
CVS failure	51 (68.9%)	18 (50.0%)	33 (86.8%)	0.001***
CNS failure	59 (79.7%)	22 (61.1%)	37 (97.4%)	0.0003***
Renal failure	43 (58.1%)	20 (55.6%)	23 (60.5%)	0.843***
Length of ICU Stay (days)	Median (IQR) 9.5 (6.25-12.0)	Median (IQR) 7.0 (5.0-8.0)	Median (IQR) 11.5 (10.0-14.0)	0.001*

*:Mann-Whitney U test; **: student's T-test ***:Chi-square test

(CRP: C-reactive protein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; INR: international normalized ratio; WBCs: white blood cells; CVS: cardiovascular system; CNS: central nervous system; SOFA: sequential organ failure assessment; APACHE: acute physiology and chronic health evaluation; IQR: inter quartile range)

Table (3): Multivariate Logistic Regression for mortality predictor

	Univariate Logistic Regression			Multivariate logistic regression analysis		
Variable	OR	95% CI	p-value	OR	95% CI	p-value
Age (years)	1.022	0.99-1.054	0.175			
CRP (mg/dl)	0.995	0.987-1.002	0.160			
Temperature	0.479	0.259-0.885	0.0189			
PaO2_FiO2	0.979	0.969-0.988	0.001			
AST (mg/dl)	1.021	0.962-1.085	0.495			

	Univariate Logistic Regression			Multivariate logistic regression analysis		
ALT (mg/dl)	**1.001**	**0.936-1.07**	**0.984**			
INR	**2.238**	**0.523-9.581**	**0.278**			
Lactate-Albumin Ratio	**3.915**	**1.455-10.54**	**0.007**	**2.342**	**0.645-8.5**	**0.196**
Total SOFA Score	**2.795**	**1.744-4.478**	**0.001**	**2.587**	**1.523-4.396**	**0.0004**
Sex	**0.318**	**0.116-0.873**	**0.026**	**0.244**	**0.055-1.069**	**0.061**
Stroke	**6.177**	**2.087-18.279**	**0.001**	**4.333**	**0.971-19.338**	**0.055**

($PaO_{2:}$ partial pressure of oxyge; FiO_2: fraction of inspired oxygen;CRP: C-reactive protein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; INR: international normalized ratio; SOFA: sequential organ failure assessment; 95% CI: confidence interval; OR: odds ratio)

Table (4): Correlation Between Lactate–Albumin Ratio (LAR) and Laboratory/Clinical Variables

Variable	Correlation Coefficient (ρ)	p-value
Laboratory Variables		
Hemoglobin (g/dL)	**-0.172**	**0.1435***
WBCs (×10⁹/L)	**-0.174**	**0.2242****
Platelets (×10⁹/L)	**-0.168**	**0.1530***
Serum sodium (mmol/L)	**-0.158**	**0.1789***
Serum potassium (mmol/L)	**-0.099**	**0.3999***
AST (U/L)	**0.163**	**0.1657***
ALT (U/L)	**0.115**	**0.3307***
Serum urea (mg/dL)	**-0.163**	**0.1648***
Serum creatinine (mg/dL)	**-0.002**	**0.9872***
Serum bilirubin (mg/dL)	**0.271**	**0.0194***
Clinical Outcomes		
Ventilator use	**0.389**	**0.0006*****
Hemodialysis sessions	**-0.142**	**0.2266*****
Vasopressor use	**0.347**	**0.0025*****
Length of ICU stay (days)	**0.208**	**0.0753***
Total SOFA score	**0.421**	**0.0002***
APACHE II score	**0.395**	**0.0005****
Respiratory failure	**0.099**	**0.3996*****
Coagulopathy	**0.271**	**0.0197*****
Liver failure	**0.118**	**0.3165*****
Cardiovascular failure	**0.310**	**0.0073*****
CNS failure	**0.209**	**0.0735*****
Renal failure	**-0.061**	**0.6062*****
Septic shock	**0.347**	**0.0025*****

WBCs: White blood cells; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; SOFA: Sequential Organ Failure Assessment; APACHE II: Acute Physiology and Chronic Health Evaluation II; CNS: Central nervous system; LAR: Lactate–albumin ratio. Statistical tests used: *Spearman's rho; **Pearson's rho; ***Point biserial correlation.

Table (5):Comparison of Infection Sites, Culture Results, Comorbidities, and Laboratory/Clinical Parameters Between Survivors and Non-Survivors With Sepsis/Septic Shock (n=74)

Variable	Survivors (n, %) / Median (IQR) / Mean ± SD	Non-Survivors (n, %) / Median (IQR) / Mean ± SD	Overall (n, %) / Median (IQR) / Mean ± SD	p-value
Infection Sites				
Pneumonia	29 (80.6%)	36 (94.7%)	65 (87.8%)	0.0818*
UTI	11 (30.6%)	6 (15.8%)	17 (23.0%)	0.1705*
Skin infection	4 (11.1%)	11 (28.9%)	15 (20.3%)	0.0826*
Other infections	1 (2.8%)	3 (7.9%)	4 (5.4%)	0.6151*
Culture Results				
Blood culture	36 (100%)	38 (100%)	74 (100%)	1*
Sputum culture	29 (80.6%)	36 (94.7%)	65 (87.8%)	0.0818*
Urine culture	11 (30.6%)	6 (15.8%)	17 (23.0%)	0.1705*
Skin swab	4 (11.1%)	11 (28.9%)	15 (20.3%)	0.0826*
Comorbidities & Infection Patterns in Septic Shock				
Sex (male)	1 (20.0%)	11 (44.0%)	12 (40.0%)	1.000*
DM	3 (60.0%)	16 (64.0%)	19 (63.3%)	1.000*
HTN	4 (80.0%)	19 (76.0%)	23 (76.7%)	1.000*
IHD	0 (0.0%)	5 (20.0%)	5 (16.7%)	0.556*
Stroke	0 (0.0%)	14 (56.0%)	14 (46.7%)	0.045*
COPD	0 (0.0%)	1 (4.0%)	1 (3.3%)	1.000*
Malignancy	0 (0.0%)	2 (8.0%)	2 (6.7%)	1.000*
Pneumonia	3 (60.0%)	25 (100.0%)	28 (93.3%)	0.023*
UTI	0 (0.0%)	3 (12.0%)	3 (10.0%)	1.000*
Skin infection	2 (40.0%)	11 (44.0%)	13 (43.3%)	1.000*
Other infections	0 (0.0%)	2 (8.0%)	2 (6.7%)	1.000*
Ventilator use	0 (0.0%)	6 (24.0%)	6 (20.0%)	0.553*
Hemodialysis sessions	0 (0.0%)	4 (16.0%)	4 (13.3%)	1.000*
Vasopressor use	5 (100.0%)	25 (100.0%)	30 (100.0%)	1.000*
Laboratory & Clinical Parameters in Septic Shock				
Age (years)	62.00 (57.00–76.00)	67.00 (65.0–75.0)	66.50 (63.50–75.75)	0.468**
Serum Lactate (mmol/L)	2.90 (1.80–3.00)	3.40 (2.70–5.30)	3.10 (2.62–5.03)	0.254**
Serum Albumin (g/dL)	2.72 (2.30–2.73)	2.35 (2.16–2.97)	2.39 (2.16–2.83)	0.781**
Hemoglobin (g/dL)	9.87 (8.34–10.12)	9.12 (8.45–10.12)	9.16 (8.45–10.12)	0.780**
Platelets (×10⁹/L)	272.0 (170.0–277.0)	149.00 (100.0–256.0)	154.00 (100.50–275.75)	0.231**
Serum sodium (mmol/L)	142.0 (131.0–148.0)	138.00 (129.0–143.0)	138.00 (129.25–143.75)	0.596**
Serum potassium (mmol/L)	3.89 (3.01–4.45)	3.78 (3.23–4.34)	3.78 (3.23–4.42)	0.738**
AST (U/L)	30.00 (24.0–35.0)	33.00 (27.0–38.0)	32.50 (27.00–38.00)	0.387**

Variable	Survivors (n, %) / Median (IQR) / Mean ± SD	Non-Survivors (n, %) / Median (IQR) / Mean ± SD	Overall (n, %) / Median (IQR) / Mean ± SD	p-value
ALT (U/L)	24.00 (19.0–28.0)	26.00 (21.0–31.0)	25.50 (21.00–30.75)	0.328**
CRP (mg/dL)	186.00 (176.0–204.0)	189.00 (115.0–209.0)	187.50 (116.00–208.75)	0.824**
Serum urea (mg/dL)	178.00 (167.0–209.0)	189.00 (87.0–226.0)	183.50 (104.75–225.25)	0.829**
Serum creatinine (mg/dL)	1.37 (1.20–1.40)	1.82 (0.87–3.90)	1.55 (0.88–3.05)	0.373**
Serum Bilirubin (mg/dL)	0.30 (0.24–0.40)	0.79 (0.45–1.12)	0.50 (0.40–1.00)	0.011**
INR	1.10 (0.98–1.60)	1.20 (1.00–1.40)	1.19 (1.00–1.48)	0.889**
Lactate–Albumin Ratio	1.30 (0.66–1.41)	1.44 (1.11–2.26)	1.38 (0.91–2.11)	0.355**
Total SOFA Score	5.00 (5.00–5.00)	8.00 (7.00–9.00)	7.00 (5.25–8.75)	0.005**
Length of ICU Stay (days)	7.00 (5.00–8.00)	10.00 (9.00–14.00)	10.00 (8.00–13.00)	0.046**
APACHE II Score (Mean ± SD)	22.40 ± 3.05	28.12 ± 6.11	27.17 ± 6.07	0.074**
WBCs (×10⁹/L) (Mean ± SD)	18.94 ± 6.83	21.37 ± 5.52	20.96 ± 5.70	0.373**

UTI: Urinary tract infection; DM: Diabetes mellitus; HTN: Hypertension; IHD: Ischemic heart disease; COPD: Chronic obstructive pulmonary disease; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; CRP: C-reactive protein; INR: International normalized ratio; WBCs: White blood cells; SOFA: Sequential Organ Failure Assessment; APACHE II: Acute Physiology and Chronic Health Evaluation II; ICU: Intensive care unit; LAR: Lactate–albumin ratio. Statistical tests used: *Fisher's exact test; **Mann–Whitney U test.

Table (6): Comparison of Organ Support, Mortality, Neurological Status, and Multiorgan Failure Between High and Low Lactate–Albumin Ratio (LAR) Groups

Variable	High LAR Group	Low LAR Group	p-value
Organ Support			
Ventilator use	8 (22.9%)	0 (0.0%)	0.0053*
Hemodialysis sessions	3 (8.6%)	6 (15.4%)	0.5898*
Vasopressor use	21 (60.0%)	9 (23.1%)	0.0028*
Age-based Mortality			
60–80 years	17 (73.9%)	10 (47.6%)	0.1391*
>80 years	3 (75.0%)	1 (25.0%)	0.4795*
<40 years	4 (80.0%)	1 (16.7%)	0.1356*
40–60 years	2 (66.7%)	0 (0.0%)	0.0938*
Neurological Status (GCS)			
GCS (Median, IQR)	11.0 (8.0–13.0)	14.0 (12.5–15.0)	0.001
Multiorgan Failure			
Respiratory failure	20 (57.1%)	16 (41.0%)	0.2493*
Coagulopathy	18 (51.4%)	10 (25.6%)	0.0410*
Liver failure	7 (20.0%)	6 (15.4%)	0.8298*
Cardiovascular failure	29 (82.9%)	22 (56.4%)	0.0276*
CNS failure	32 (91.4%)	27 (69.2%)	0.0374*
Renal failure	19 (54.3%)	24 (61.5%)	0.6926*

LAR: Lactate–albumin ratio; GCS: Glasgow Coma Scale; CNS: Central nervous system; IQR: Interquartile range. Statistical tests used: *Chi-square test; **Mann–Whitney U test.

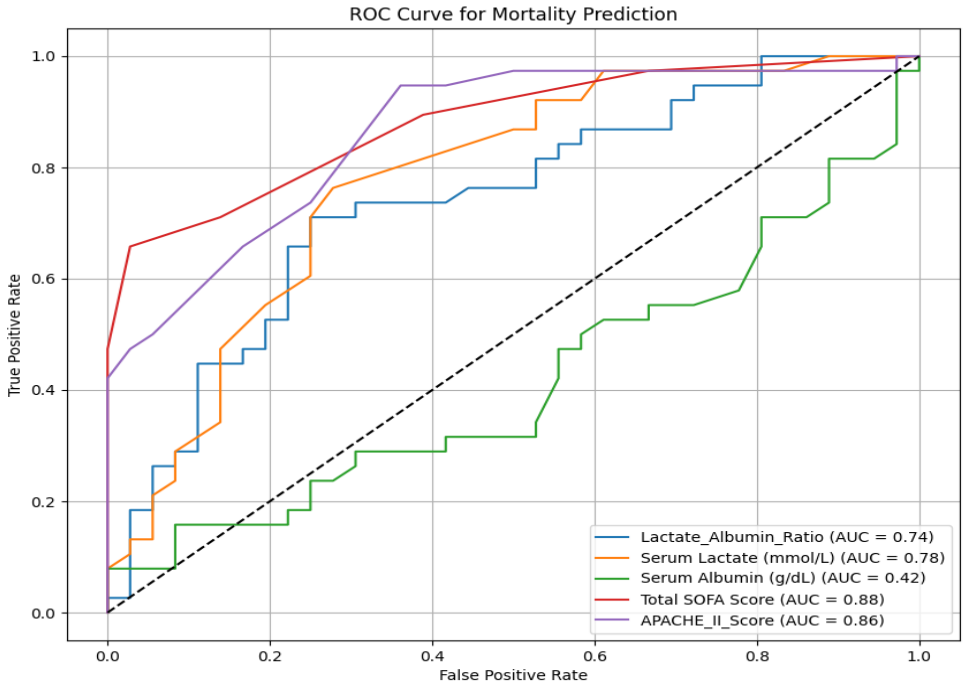

Figure 1: ROC curve analysis of lactate/albumin ratio for predicting mortality

DISCUSSION

In this study, the LAR exhibited a significant association with ICU mortality in patients with sepsis and septic shock. Receiver operating characteristic analysis yielded an area under the curve (AUC) of 0.74, reflecting a moderate level of discriminative accuracy. The cutoff value of 1.11 provided 71% sensitivity and 75% specificity. Notably, LAR surpassed serum albumin (AUC 0.42) and was nearly comparable to lactate alone (AUC 0.78), underscoring its clinical utility as a composite marker that merges tissue hypoperfusion and systemic inflammation.

The present study findings align with those of Cakir and Turan [15], who analyzed over 1,100 sepsis cases and reported an AUC of 0.869 at a cutoff of 0.71, confirming the strong prognostic power of LAR. Similarly, Yoo et al. [16] evaluated more than 3,000 patients and found that LAR achieved an AUC of 0.715 for predicting 28-day mortality, even outperforming the SOFA score (AUC 0.669). In their cohort, patients with LAR >1.52 had nearly four times higher odds of death (OR 3.75, 95% CI: 3.16–4.45).

The prognostic performance of LAR, however, appears to vary across settings. Bou Chebl et al. [17] assessed septic patients in the emergency department and reported a lower AUC of 0.67, while Gharipour et al. [18] documented an AUC of 0.69 for ICU mortality prediction. Possible explanation: these cohorts differed in clinical setting and illness severity, and LAR was often measured earlier in the disease course, which may reduce predictive strength compared with our ICU population. These findings suggest that disease severity, timing of measurement, and patient populations may influence LAR's predictive accuracy.

Meta-analytic evidence reinforces its role. Zhao et al. [19] pooled data from nine studies including more than 3,000 patients and demonstrated that LAR predicted mortality with a pooled AUC of 0.75, along

with significant associations with both death (OR 2.16, 95% CI: 1.58–2.95) and multi-organ dysfunction (OR 3.41, 95% CI: 1.78–6.50). In agreement, Wang et al. [20] observed an AUC of 0.74, nearly identical to that in the present study, and confirmed that higher LAR independently increased in-hospital mortality risk (OR 1.44, 95% CI: 1.31–1.59). Notably, they also showed that LAR outperformed lactate alone (AUC 0.74 vs 0.70), which echoes the present finding that the ratio is superior to albumin considered independently.

Further supporting evidence comes from Li et al. [21], who analyzed 274 septic patients and reported an AUC of 0.807 at a lower cutoff value of 0.16, again showing LAR's superiority over lactate or albumin alone. Possible explanation: their much lower cutoff may reflect different laboratory calibration or population characteristics compared with our ICU study. Yoon et al. [22], in a systematic review of eight studies comprising 4,723 patients, confirmed pooled sensitivity and specificity of 0.71 and 0.68, with an overall AUC of 0.74. Taken together, these findings place the present study in concordance with the majority of published evidence, highlighting LAR as a practical biomarker that can complement existing scoring systems for early mortality prediction in sepsis.

As regards the present study findings with SOFA and APACHE II, both scores demonstrated strong prognostic performance. SOFA had the highest accuracy (AUC 0.88 at cutoff ≥7), followed by APACHE II (AUC 0.86 at cutoff ≥19). When adjusted for SOFA, LAR lost statistical significance as an independent predictor (OR 2.34, 95% CI: 0.65–8.50, p = 0.20). These findings confirm the established role of traditional scoring systems in mortality prediction. Consistent with this, Chung et al. [23] showed that SOFA had the best discriminatory power among multiple biomarkers (AUC 0.931), surpassing LAR (AUC 0.830). Similarly, Zhang et al. [24] studied patients with community-acquired pneumonia and reported AUCs of 0.741 for SOFA and 0.774 for APACHE II in predicting 28-day mortality, which—though slightly lower—support the robustness of these scores.

Concerning the current study, LAR correlations, elevated ratios above 1.11 were associated with higher requirements for mechanical ventilation (22.9% vs 0%, p = 0.005) and vasopressors (60% vs 23%, p = 0.003). They were also linked with cardiovascular failure (82.9% vs 56%, p = 0.03) and altered consciousness (91% vs 69%, p = 0.04). These findings agree with Kabra et al. [25], who observed higher LAR levels in patients needing vasopressors, and with Acharya et al. [26], who reported that LAR strongly predicted mechanical ventilation (AUC 0.881) and inotropic support (AUC 0.819). Kasapoglu et al. [27] further confirmed its value in acute hypercapnic respiratory failure, where an LAR cutoff of 0.605 predicted non-invasive ventilation failure (AUC 0.718), with each unit increase raising intubation risk fivefold (OR 5.58).

The present study did not demonstrate significant correlations between LAR and hepatic, renal, or respiratory dysfunction (p = 0.2493, 0.8298, and 0.6926, respectively). Possible explanation: our sample size was smaller and focused on ICU admissions with early sepsis, which may limit the detection of associations with later organ-specific complications. This diverges from Gao et al. [26], who showed that LAR predicted both ARDS onset (AUC 0.878) and hepatic injury (AUC 0.905), and was linked to prolonged invasive ventilation. Such discrepancies may reflect differences in patient populations, disease severity, and sample sizes across studies.

As regards demographic and comorbidity factors, the present study showed higher mortality in males compared with females (47.4% vs 22.2%, p = 0.04), and prior cerebrovascular disease was significantly more common among non-survivors (55.3% vs 16.7%, p = 0.001). Although older age trended toward poorer outcomes (median 66 vs 62.5 years), the association was not statistically significant (p = 0.11). Mortality in septic shock was accompanied by higher bilirubin levels (0.79 vs 0.30 mg/dL, p = 0.01), higher SOFA scores (8 vs 5, p = 0.005), and longer ICU stays (10 vs 7 days, p = 0.05). Pneumonia as the source of infection was strongly associated with fatal outcomes (100% vs 60%, p = 0.02). These results contrast with Pietropaoli et al. [29], who reported that female patients with severe sepsis or septic shock had a higher adjusted risk of mortality than males (OR 1.11, 95% CI: 1.04–1.19). Such differences may be linked to immunomodulatory effects of estrogen or detrimental influences of male sex hormones on cell-mediated immunity, as highlighted by Suarez De La Rica et al. [30].

As regards the present study infection characteristics, pneumonia and skin infections were more frequent among non-survivors (94.7% vs 80.6% and 28.9% vs 11.1%, respectively), though these differences did not reach statistical significance (p = 0.08 for both). In contrast, urinary tract infections were more often seen among survivors (30.6% vs 15.8%). Our results are in line with those reported by He et al. [31], who analyzed 483 cases of sepsis and demonstrated that pulmonary sources, compared with abdominal infections, were linked to older patient age, higher APACHE II scores, greater ICU mortality (31.7% vs 12.6%), increased one-year mortality (45.4% vs 24.4%), and poorer long-term quality of life. These findings collectively reinforce the notion that pneumonia as the underlying cause of sepsis is associated with unfavorable outcomes.

Concerning the present study septic shock subgroup, non-survivors were more likely to have a prior history of stroke (56.0% vs 0.0%, p = 0.045) and pneumonia as the infection source (100% vs 60.0%, p = 0.023). In addition, non-survivors had significantly higher SOFA scores (median 8 vs 5, p = 0.005), longer ICU stays (median 10 vs 7 days, p = 0.046), and elevated bilirubin levels (0.79 vs 0.30 mg/dL, p = 0.011). These results are consistent with the concept that multi-organ failure remains the key driver of sepsis mortality, as emphasized in the international guidelines by Dellinger et al. [32].

As regards the clinical relevance of LAR, its strength lies in reflecting two interconnected disturbances central to the pathogenesis of sepsis: metabolic stress and systemic inflammation. Lactate, once considered a mere byproduct of anaerobic glycolysis, is now recognized as a signaling mediator that reflects mitochondrial dysfunction, altered energy metabolism, and cellular stress responses [33]. These processes are commonly activated in sepsis, contributing to multiorgan dysfunction.

In the current study, a modest but significant correlation between LAR and serum bilirubin (r = 0.271, p = 0.019) suggests a hepatic component. This is reinforced by the finding that non-survivors exhibited higher bilirubin levels, implicating impaired liver function in reduced lactate clearance. This interpretation aligns with the observations of Hernandez et al. [34], who emphasized the pitfalls of lactate clearance in sepsis when hepatic dysfunction is present.

The second element of the ratio, serum albumin, has diverse physiological roles beyond oncotic pressure maintenance. It carries hormones, fatty acids, and metal ions; modulates pharmacokinetics; and exerts antioxidant, anti-inflammatory, and

detoxification effects. Albumin also contributes to acid-base buffering. In critical illness, hypoalbuminemia is consistently associated with adverse outcomes, including in sepsis [35]. Egbert et al. [36] further demonstrated that hypoalbuminemia, particularly in the context of obesity and trauma, predicts complications and poor recovery.

Albumin's prognostic relevance has also been incorporated into several scoring systems, including APACHE II, the Critical Illness Severity Score System (CISSS), and the Glasgow Prognostic Score, which combines albumin with CRP to improve predictive performance [36]. These associations explain why albumin is often viewed as a systemic illness severity surrogate marker.

The dependency of LAR on SOFA in multivariate analysis can also be understood from this pathophysiological overlap. SOFA incorporates cardiovascular, hepatic, and coagulation parameters—all systems influenced by lactate and albumin metabolism. Consequently, part of LAR's predictive capacity is inherently captured within SOFA. This was particularly evident in the septic shock subgroup of the present study, where both LAR (median 1.38 vs 0.9, $p = 0.001$) and SOFA (median 8 vs 5, $p = 0.005$) were significantly higher among non-survivors. Nevertheless, LAR offers the advantage of objectivity and early detectability. Bou Chebl et al. [17] emphasized this in emergency department settings, where LAR rose earlier than complete SOFA scoring, supporting its role in rapid risk stratification.

As regards the strengths of the present study, one of the most practical advantages of the lactate-to-albumin ratio (LAR) is its reliance on two routinely measured parameters—serum lactate and albumin—that are universally available in ICU settings. This makes LAR an inexpensive, rapid, and easily reproducible biomarker, even in hospitals with limited resources. Unlike complex scoring systems requiring multiple clinical and laboratory inputs, LAR can be calculated immediately at the bedside, supporting early triage and guiding timely intervention. Its accessibility is particularly valuable in overcrowded or resource-constrained settings, where rapid prognostic tools may assist in prioritizing care and optimizing outcomes.

The present study had several limitations. LAR lost statistical significance when adjusted for SOFA, indicating partial overlap with established scoring tools. Reported cutoff values vary widely across studies, limiting standardization. The single-center design and restriction to medical ICU patients reduce generalizability. Measurements were limited to a single time point, although evidence suggests serial monitoring may enhance accuracy. Finally, surgical and obstetric sepsis cases were not included, narrowing applicability to broader populations.

CONCLUSION

In the present study, SOFA and APACHE II remained the most accurate predictors of ICU mortality in sepsis and septic shock. The lactate-to-albumin ratio (LAR) showed moderate predictive value and correlated with the need for vasopressors and mechanical ventilation, highlighting its utility as a rapid, inexpensive bedside marker. While its prognostic strength overlaps with SOFA, LAR's simplicity and availability make it particularly useful for early risk stratification, especially in resource-limited settings. Broader multicenter studies and serial measurements are needed to refine cutoffs and better define its role in sepsis management.

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