



RESEARCH ARTICLE

Lymphocyte-To-C-Reactive Protein Ratio As An Early Prognostic Marker In Emergency Department Patients With Sepsis: A Retrospective Study

Palak Raj¹, Riya Shah², Sanjeev Narang³, Romi Srivastava⁴, V K Jain⁵

ABSTRACT

Background: Sepsis is a life-threatening condition associated with systemic inflammation, immune dysfunction and organ failure. Early identification of patients at increased risk of mortality and acute kidney injury (AKI) remains challenging. The lymphocyte-to-C-reactive protein ratio (LCR) combines information on lymphocyte depletion and systemic inflammation and may have prognostic value.

Methods: A retrospective observational study was conducted among 400 adult patients with sepsis presenting to the emergency department. Baseline demographic, clinical and laboratory data were collected. LCR was calculated from the initial absolute lymphocyte count and CRP concentration. Patients were categorized into four LCR quartiles. The primary outcome was 30-day all-cause mortality, while AKI was evaluated as a secondary outcome. ROC analysis, Kaplan-Meier survival analysis and multivariable regression were performed.

Results: Overall, 110 patients (27.5%) died within 30 days and 182 (45.5%) developed AKI. Mortality decreased progressively across LCR quartiles, from 36.6% in Q1 to 16.2% in Q4 ($p=0.003$). AKI similarly decreased from 59.4% in Q1 to 31.3% in Q4 ($p<0.001$). Lower LCR was associated with higher disease severity, inflammatory burden and organ dysfunction. LCR demonstrated an AUC of 0.671 for 30-day mortality and 0.648 for AKI. Kaplan-Meier analysis showed significantly poorer survival among patients with lower LCR (log-rank $p<0.001$). Higher LCR remained associated with lower mortality and AKI after multivariable adjustment.

Conclusion: Lower LCR was associated with increased 30-day mortality and AKI in emergency department patients with sepsis. LCR may serve as a simple adjunctive biomarker for early risk stratification.

Keywords: Sepsis; Lymphocyte-to-C-reactive protein ratio; Mortality; Acute kidney injury; Prognosis.

Indian J. Pharm. Biol. Res. (2026); <https://doi.org/10.30750/ijpbr.14.4.31>

INTRODUCTION

Sepsis is a life-threatening clinical syndrome resulting from a dysregulated host response to infection and is characterized by potentially fatal organ dysfunction [1,2]. Despite advances in early recognition and management, sepsis continues to impose a substantial global burden, with considerable mortality among affected patients [3]. Early identification of patients at high risk of adverse outcomes is therefore essential, particularly in the emergency department (ED), where timely risk stratification can facilitate appropriate monitoring and treatment.

The pathophysiology of sepsis involves a complex interplay between excessive inflammation and subsequent immune dysfunction [4]. During the early phase, activation of immune cells and release of inflammatory mediators contribute to systemic inflammation and tissue injury [5]. At the same time, profound alterations in lymphocyte homeostasis occur. Lymphopenia is frequently observed in sepsis and has been associated with disease severity and increased mortality [6]. Persistent depletion of circulating lymphocytes may reflect impaired immune competence and

has been recognized as an important component of sepsis-associated immune dysfunction [7].

C-reactive protein (CRP) is a readily available acute-phase inflammatory marker that has been widely used in the assessment of patients with infection and sepsis [8]. Elevated CRP levels have been associated with disease severity and prognosis in critically ill and septic patients [9,10]. However, CRP predominantly reflects systemic inflammation, whereas lymphocyte count provides information regarding the immune response. Assessment of either parameter alone may therefore provide only a partial representation of the complex inflammatory and immunological alterations occurring during sepsis.

The lymphocyte-to-C-reactive protein ratio (LCR) combines these two complementary biomarkers into a single index and may provide a more integrated reflection of the balance between inflammation and immune status. LCR has demonstrated potential prognostic utility in several inflammatory and malignant conditions [11,12]. Emerging evidence has also suggested its potential value in infectious diseases, including sepsis, where lower LCR values may

¹Junior Resident-3, Department of Pathology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India

²Assistant Professor, Department of Pathology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India

³Professor and Head of Department, Department of Pathology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India

⁴Professor, Department of Pathology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India

⁵Professor, Department of Pathology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India

Corresponding Author: Dr. Palak Raj, PG Resident, Department of Pathology, Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh, India Email id: Raj.palak94@gmail.com

How to cite this article: Raj P, Shah R, Narang S, Srivastava R, Jain VK. Lymphocyte-To-C-Reactive Protein Ratio As An Early Prognostic Marker In Emergency Department Patients With Sepsis: A Retrospective Study. Indian J. Pharm. Biol. Res. 2026;14(4):212-226.

Source of support: Nil

Conflict of interest: None.

Received: 02/08/2026 **Revised:** 18/09/2026 **Accepted:** 20/09/2026

be associated with adverse outcomes [13,14]. Nevertheless, evidence regarding the prognostic significance of LCR in adult patients presenting with sepsis to the ED remains limited.

Therefore, the present retrospective study was undertaken to evaluate the lymphocyte-to-C-reactive protein ratio as an early prognostic marker in emergency department patients with sepsis, and to determine its association with 30-day mortality and the development of acute kidney injury (AKI). Identification of a simple and routinely available biomarker such as LCR may improve early risk stratification and assist in recognizing patients at greater risk of poor outcomes.

MATERIALS AND METHODS

This retrospective observational study was conducted among adult patients presenting to the Emergency Department (ED) with sepsis. The study evaluated the prognostic significance of the lymphocyte-to-C-reactive protein ratio (LCR) at the time of initial ED evaluation and its association with short-term clinical outcomes. A total of 400 patients with sepsis who fulfilled the predefined eligibility criteria were included in the study.

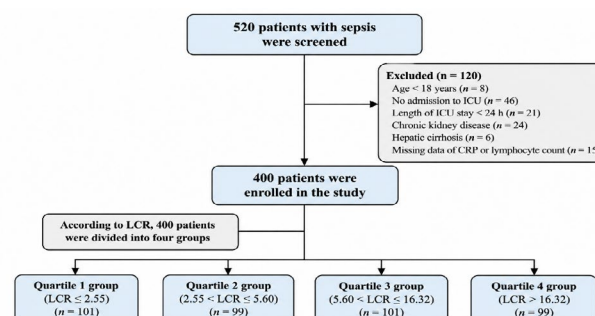


Figure 1. Flow of patient selection and grouping in the study. Abbreviations: LCR, lymphocyte-to-C-reactive protein ratio; ICU, intensive care unit.

Figure 1. Flow of patient selection and grouping in the study.

The study population comprised patients aged ≥ 18 years who presented to the ED with a clinical diagnosis of sepsis during the specified study period. Sepsis was defined according to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3) as life-threatening organ dysfunction caused by a dysregulated host response to infection.

Patients with a documented diagnosis of sepsis and available measurements of absolute lymphocyte count and serum CRP obtained during the initial ED evaluation were considered eligible for inclusion.

Inclusion Criteria

Patients aged ≥ 18 years.

Patients presenting to the Emergency Department with sepsis.

Patients fulfilling the Sepsis-3 definition of sepsis.

Availability of absolute lymphocyte count and serum CRP measurement at initial ED evaluation.

Availability of clinical outcome data for assessment of 30-day mortality and AKI.

EXCLUSION CRITERIA

Patients were excluded if they were < 18 years of age, had pre-existing chronic kidney disease where assessment of new-onset AKI was not reliably possible, had incomplete data required for calculation of LCR, or had insufficient follow-up information to determine the primary study outcome.

Data Collection

Data were retrospectively obtained from hospital medical records and electronic databases. Demographic characteristics, clinical findings, relevant comorbidities, laboratory investigations, severity indicators, treatment-

Table 1: Baseline demographic and clinical characteristics according to LCR

Variable	Overall (n=400)	Q1 (n=101)	Q2 (n=99)	Q3 (n=101)	Q4 (n=99)	P value
Age, years	74 (65–83)	76 (66–85)	75 (65–83)	73 (64–82)	72 (63–81)	0.214
Male, n (%)	249 (62.3)	62 (61.4)	63 (63.6)	63 (62.4)	61 (61.6)	0.982
Female, n (%)	151 (37.8)	39 (38.6)	36 (36.4)	38 (37.6)	38 (38.4)	0.982
BMI, kg/m ²	22.6 (20.4– 25.3)	22.4 (20.1– 24.8)	22.7 (20.5– 25.4)	22.6 (20.3– 25.5)	22.7 (20.6– 25.5)	0.781
Diabetes mellitus, n (%)	112 (28.0)	29 (28.7)	30 (30.3)	28 (27.7)	25 (25.3)	0.864
Hypertension, n (%)	208 (52.0)	50 (49.5)	52 (52.5)	55 (54.5)	51 (51.5)	0.846
Coronary artery disease, n (%)	43 (10.8)	11 (10.9)	12 (12.1)	11 (10.9)	9 (9.1)	0.914
COPD, n (%)	32 (8.0)	6 (5.9)	7 (7.1)	10 (9.9)	9 (9.1)	0.772
Cerebrovascular disease, n (%)	58 (14.5)	15 (14.9)	16 (16.2)	14 (13.9)	13 (13.1)	0.934
Vasoactive drugs, n (%)	261 (65.3)	80 (79.2)	69 (69.7)	62 (61.4)	50 (50.5)	<0.001
Invasive ventilation, n (%)	264 (66.0)	70 (69.3)	65 (65.7)	64 (63.4)	65 (65.7)	0.812
ICU admission, n (%)	348 (87.0)	95 (94.1)	89 (89.9)	87 (86.1)	77 (77.8)	0.003

Values are expressed as median (IQR) or n (%).

related variables, and clinical outcomes were recorded.

Laboratory parameters obtained during the initial ED assessment included complete blood count with absolute lymphocyte count, serum CRP, total leukocyte count, neutrophil count, hemoglobin, platelet count, serum creatinine, blood urea nitrogen, serum lactate, bilirubin, and other routinely available parameters relevant to assessment of sepsis severity and organ dysfunction.

Lymphocyte-to-C-Reactive Protein Ratio

The LCR was calculated from the absolute lymphocyte count and serum CRP concentration obtained during the initial ED evaluation using the following formula:

LCR = Absolute lymphocyte count / C-reactive protein concentration

LCR was initially analyzed as a continuous variable. For additional analysis, patients were categorized into groups according to LCR quartiles or an optimal cutoff value determined using receiver operating characteristic (ROC) curve analysis.

Study Outcomes

The primary outcome was 30-day all-cause mortality following ED presentation with sepsis.

The secondary outcome was the development of acute kidney injury (AKI) during hospitalization. AKI was

defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.

Additional clinical outcomes, where available, included ICU admission, length of hospital stay, ICU mortality, and hospital mortality.

Statistical Analysis

Data were entered into a computerized database and analyzed using appropriate statistical software. Continuous variables were expressed as mean $\pm$ standard deviation or median with interquartile range, depending on the distribution of the data. Categorical variables were presented as frequencies and percentages. Patients were compared according to 30-day survival status and according to the presence or absence of AKI. Appropriate parametric or non-parametric tests were used for comparison of continuous variables, while categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. The prognostic performance of LCR for predicting 30-day mortality and AKI was assessed using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC), 95% confidence interval, sensitivity, specificity, and optimal cutoff value were calculated.

For assessment of the association between LCR and 30-day mortality, Cox proportional hazards regression analysis was performed where appropriate. LCR was

Table 2: Laboratory parameters and severity scores according to LCR

Variable	Overall (n=400)	Q1 (n=101)	Q2 (n=99)	Q3 (n=101)	Q4 (n=99)	P value
WBC, ×10 ⁹ /L	11.5 (7.6–17.0)	10.6 (7.0–15.7)	12.3 (8.2–17.9)	11.7 (7.8–17.2)	11.2 (7.5–16.4)	0.018
Neutrophils, ×10 ⁹ /L	10.0 (6.4–15.2)	9.4 (6.2–14.8)	11.2 (7.1–16.0)	10.1 (6.5–15.1)	9.6 (6.1–14.5)	0.011
Lymphocytes, ×10 ⁹ /L	0.61 (0.34–0.91)	0.31 (0.21–0.42)	0.48 (0.34–0.67)	0.73 (0.53–1.01)	0.91 (0.65–1.31)	<0.001
Hemoglobin, g/dL	11.6 (9.8–13.1)	11.2 (9.5–12.6)	11.4 (9.7–12.9)	11.7 (9.9–13.1)	12.0 (10.5–13.6)	0.003
Platelets, ×10 ⁹ /L	151 (96–216)	119 (78–172)	137 (88–202)	158 (106–224)	184 (139–249)	<0.001
CRP, mg/L	108.6 (45.2–169.4)	176.4 (132.5–226.8)	137.2 (96.1–181.4)	91.5 (57.8–124.6)	82.7 (39.8–129.5)	<0.001
LCR	5.88 (2.61–17.14)	1.82 (1.39–2.21)	3.74 (3.01–4.82)	9.42 (7.06–12.34)	72.16 (31.24–188.42)	<0.001
Creatinine, mg/dL	1.04 (0.62–1.71)	1.34 (0.82–2.19)	1.21 (0.75–1.78)	1.01 (0.63–1.46)	0.84 (0.55–1.29)	<0.001
Lactate, mmol/L	2.2 (1.4–3.6)	2.5 (1.7–3.9)	2.3 (1.5–3.6)	2.1 (1.3–3.5)	1.9 (1.2–3.1)	0.006
SOFA score	11 (9–14)	13 (10–15)	12 (10–14)	11 (9–13)	10 (8–13)	<0.001
APACHE II score	24 (19–30)	26 (21–32)	25 (20–30)	24 (19–29)	23 (18–28)	0.017

analyzed both as a continuous variable and as a categorical variable according to quartiles or the optimal cutoff. Kaplan-Meier survival curves were constructed to compare 30-day survival between different LCR categories, and differences were assessed using the log-rank test. Binary logistic regression analysis was performed to evaluate the association between LCR and development of AKI. Univariable and multivariable models were constructed to determine whether LCR independently predicted the study outcomes after adjustment for clinically relevant confounding variables. Results were reported as hazard ratios (HRs) or odds ratios (ORs), as appropriate, with corresponding 95% confidence intervals (CIs). A two-sided P value <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki. Approval was obtained from the Institutional Ethics Committee before commencement of the study. As this was a retrospective study involving review of existing medical records, a waiver of informed consent was obtained where permitted by the Institutional Ethics Committee.

RESULTS

Study Population and Baseline Characteristics

A total of 400 patients with sepsis who fulfilled the predefined eligibility criteria were included in the study. The median age of the study population was 74 years (IQR, 65–83 years), and 249 (62.3%) patients were male. Hypertension was the most common comorbidity, present in 208 (52.0%) patients, followed by diabetes mellitus in 112 (28.0%) patients. According to LCR, patients were classified into four groups: Q1 (LCR ≤2.55; n=101), Q2 (LCR >2.55–5.60; n=99), Q3 (LCR >5.60–16.32; n=101), and Q4 (LCR >16.32; n=99). The median LCR in the overall study population was 5.88 (IQR, 2.61–17.14). There were no significant differences across the four LCR categories in age, sex, BMI, diabetes mellitus, hypertension, coronary artery disease, COPD or cerebrovascular disease. However, vasoactive drug requirement and ICU admission differed significantly between the groups. Vasoactive drugs were required most frequently among patients in Q1, while ICU admission was also more frequent among patients with lower LCR values. [Table 1]

Table 3: Thirty-day mortality according to LCR category

LCR category	Total	Survivor, n (%)	Non-survivor, n (%)
Q1 ≤ 2.55	101	64 (63.4)	37 (36.6)
Q2 $>2.55-5.60$	99	67 (67.7)	32 (32.3)
Q3 $>5.60-16.32$	101	76 (75.2)	25 (24.8)
Q4 >16.32	99	83 (83.8)	16 (16.2)
Total	400	290 (72.5)	110 (27.5)

Laboratory Characteristics According to LCR

A significant gradient was observed across LCR categories for several laboratory parameters. The median lymphocyte count increased progressively from $0.31 \times 10^9/L$ in Q1 to $0.91 \times 10^9/L$ in Q4 ($P < 0.001$). Conversely, CRP concentrations decreased with increasing LCR, from 176.4 mg/L in Q1 to 82.7 mg/L in Q4 ($P < 0.001$). Platelet counts also increased progressively across the LCR categories, whereas serum creatinine and lactate concentrations were higher among patients with lower LCR values. The median SOFA score decreased from 13 in Q1 to 10 in Q4 ($P < 0.001$), and the median APACHE II score decreased from 26 to 23 ($P = 0.017$) across the same groups. These findings indicated that lower LCR was associated with greater inflammatory activity and more severe physiological disturbance. [Table 2]

30-Day Mortality

During the 30-day follow-up period, 110 (27.5%) patients died, while 290 (72.5%) survived. A progressive decline in mortality was observed with increasing LCR. The mortality rate was 36.6% in Q1, 32.3% in Q2, 24.8% in Q3, and 16.2% in Q4. The difference in mortality across the four LCR categories was statistically significant ($P = 0.003$).

Table 4: Incidence of acute kidney injury according to LCR category

LCR category	Total	Non-AKI, n (%)	AKI, n (%)
Q1 ≤ 2.55	101	41 (40.6)	60 (59.4)
Q2 $>2.55-5.60$	99	50 (50.5)	49 (49.5)
Q3 $>5.60-16.32$	101	59 (58.4)	42 (41.6)
Q4 >16.32	99	68 (68.7)	31 (31.3)
Total	400	218 (54.5)	182 (45.5)

Thus, patients in the lowest LCR category had the highest observed mortality, whereas patients in the highest LCR category had the lowest mortality. [Table 3]

Acute Kidney Injury

AKI developed in 182 (45.5%) patients, while 218 (54.5%) patients did not develop AKI. The incidence of AKI decreased progressively with increasing LCR. AKI occurred in 59.4% of patients in Q1, compared with 49.5% in Q2, 41.6% in Q3, and 31.3% in Q4. The difference in AKI incidence across the LCR categories was statistically significant ($P < 0.001$). Therefore, the lowest LCR category was associated with the highest observed frequency of AKI. [Table 4]

Comparison According to 30-Day Survival and AKI Status

Comparison of patients according to 30-day survival demonstrated significant differences in several demographic and laboratory parameters. Non-survivors were significantly older than survivors, with median ages of 79 and 72 years, respectively ($P < 0.001$). The median lymphocyte count was significantly lower among non-survivors (0.39 vs. $0.67 \times 10^9/L$, $P < 0.001$), whereas CRP concentration was significantly higher (142.8 vs. 96.3 mg/L, $P < 0.001$).

Consequently, the median LCR was significantly lower among non-survivors than survivors (2.91 vs. 7.26, $P < 0.001$). Non-survivors also had lower hemoglobin and platelet counts and higher bilirubin, AST, creatinine and BUN levels. Similarly, patients who developed AKI had significantly lower lymphocyte counts and LCR values and higher CRP concentrations than patients without AKI. The median LCR was 3.71 among patients with AKI compared with 7.31 among those without AKI ($P < 0.001$). [Table 5]

Disease Severity, Treatment and Clinical Outcomes

Patients with adverse outcomes also demonstrated greater biochemical evidence of organ dysfunction. Non-survivors had significantly higher uric acid, D-dimer, potassium and lactate levels than survivors. Patients with AKI showed similar abnormalities, with higher values of these parameters compared with patients without AKI. Disease severity was significantly greater among non-survivors and patients with AKI. The median SOFA score was 14 in non-survivors compared with 10 in survivors, while the median APACHE II score was 28 versus 22, respectively (both $P < 0.001$).

Patients with AKI had median SOFA and APACHE II scores of 13 and 26, respectively, compared with 10 and 22 among patients without AKI. Treatment requirements

Table 5: Demographic, clinical and laboratory characteristics according to 30-day survival and AKI status

<i>Variables</i>	<i>Survivor (n=290)</i>	<i>Non-survivor (n=110)</i>	<i>P value</i>	<i>Non-AKI (n=218)</i>	<i>AKI (n=182)</i>	<i>P value</i>
Age, years	72 (64–81)	79 (70–87)	<0.001	72 (63–81)	77 (68–85)	<0.001
Male, n (%)	180 (62.1)	69 (62.7)	0.921	137 (62.8)	112 (61.5)	0.806
BMI, kg/m ²	22.7 (20.5–25.4)	22.1 (19.9–24.8)	0.041	22.8 (20.5–25.5)	22.3 (20.2–25.0)	0.067
Smoking, n (%)	56 (19.3)	27 (24.5)	0.274	43 (19.7)	40 (22.0)	0.590
Hypertension, n (%)	146 (50.3)	62 (56.4)	0.284	107 (49.1)	101 (55.5)	0.209
Diabetes, n (%)	75 (25.9)	37 (33.6)	0.128	52 (23.9)	60 (33.0)	0.052
Coronary artery disease, n (%)	26 (9.0)	17 (15.5)	0.078	18 (8.3)	25 (13.7)	0.087
COPD, n (%)	20 (6.9)	12 (10.9)	0.186	14 (6.4)	18 (9.9)	0.201
Cerebrovascular disease, n (%)	35 (12.1)	23 (20.9)	0.027	25 (11.5)	33 (18.1)	0.071
Gram-positive bacteria, n (%)	37 (12.8)	18 (16.4)	0.356	25 (11.5)	30 (16.5)	0.182
Gram-negative bacteria, n (%)	69 (23.8)	34 (30.9)	0.146	48 (22.0)	55 (30.2)	0.070
Fungus, n (%)	15 (5.2)	11 (10.0)	0.082	9 (4.1)	17 (9.3)	0.044
Virus, n (%)	14 (4.8)	10 (9.1)	0.102	15 (6.9)	9 (4.9)	0.421
WBC, ×10 ⁹ /L	11.2 (7.4–16.4)	12.3 (8.1–18.1)	0.084	10.5 (7.0–15.4)	12.9 (8.0–18.9)	<0.001
Neutrophils, ×10 ⁹ /L	9.7 (6.2–14.7)	11.5 (7.4–16.8)	0.005	9.2 (6.1–14.0)	11.8 (6.8–17.4)	<0.001
Lymphocytes, ×10 ⁹ /L	0.67 (0.40–0.96)	0.39 (0.23–0.62)	<0.001	0.68 (0.40–0.98)	0.49 (0.28–0.78)	<0.001
Hemoglobin, g/dL	11.8 (10.1–13.2)	10.9 (9.2–12.5)	<0.001	11.8 (10.1–13.2)	11.2 (9.6–12.8)	0.002
Platelets, ×10 ⁹ /L	158 (103–221)	129 (79–193)	0.002	167 (112–229)	128 (78–192)	<0.001
CRP, mg/L	96.3 (40.8–151.2)	142.8 (69.4–204.6)	<0.001	84.3 (31.5–149.8)	131.6 (61.8–167.7)	<0.001
LCR	7.26 (3.31–19.82)	2.91 (1.48–7.54)	<0.001	7.31 (3.40–20.16)	3.71 (1.68–10.21)	<0.001
Total bilirubin, μmol/L	17.2 (10.1–27.4)	22.8 (13.4–36.1)	0.006	15.9 (9.8–25.3)	22.7 (13.2–34.4)	<0.001
ALT, U/L	31 (20–50)	36 (22–60)	0.018	29 (19–46)	36 (23–59)	<0.001
AST, U/L	35 (22–64)	52 (28–125)	<0.001	33 (22–57)	52 (27–125)	<0.001
Albumin, g/L	29.1 (24.8–33.6)	27.0 (23.4–32.1)	0.002	29.5 (24.9–33.5)	27.2 (23.3–32.7)	<0.001

Glucose, mmol/L	8.1 (6.0–11.2)	8.9 (6.5–12.6)	0.093	7.9 (6.4–10.7)	8.8 (6.7–13.1)	0.008
Creatinine, mg/dL	0.96 (0.58–1.52)	1.29 (0.78–2.04)	<0.001	0.78 (0.52–1.10)	1.41 (0.96–2.16)	<0.001
BUN, mmol/L	7.6 (5.2–11.3)	11.4 (7.3–16.5)	<0.001	6.5 (4.7–8.9)	12.9 (8.1–17.6)	<0.001

were also greater in patients with adverse outcomes. Vasoactive drugs, invasive ventilation and CRRT were used significantly more frequently among non-survivors and patients with AKI.[Table 6]

Distribution of LCR According to Outcomes

LCR was significantly lower among non-survivors than survivors and among patients who developed AKI compared with those without AKI. This difference was statistically significant in both comparisons ($P < 0.001$). [Figure 2]

ROC Analysis for 30-Day Mortality

The predictive performance of LCR for 30-day mortality was evaluated using receiver operating characteristic curve analysis. LCR demonstrated moderate discriminatory ability, with an AUC of 0.671 (95% CI, 0.612–0.730; $P < 0.001$). The optimal cutoff value was 3.42, which provided a sensitivity of 78.2% and specificity of 52.4%. The corresponding positive and negative predictive values were 38.4% and 86.5%, respectively.[Table 7]

ROC Analysis for Acute Kidney Injury

LCR was also evaluated for its ability to predict AKI. The ROC analysis demonstrated an AUC of 0.648 (95% CI, 0.594–0.702; $P < 0.001$). The optimal cutoff was 4.18, with a sensitivity of 71.4% and specificity of 55.0%. The positive

and negative predictive values were 57.6% and 69.3%, respectively.[Table 8]

Kaplan-Meier Survival Analysis

Kaplan-Meier survival analysis demonstrated significant differences in 30-day survival among the four LCR categories (log-rank $P < 0.001$). Patients in Q1 had the lowest probability of survival during the follow-up period, whereas patients in Q4 demonstrated the highest survival probability. A progressive separation of the survival curves was observed with increasing LCR. [Figure 3]

Outcomes Across LCR Quartiles

There was a progressive decrease in 30-day mortality and AKI occurrence with increasing LCR. The mortality proportion declined from 36.6% in Q1 to 16.2% in Q4, while AKI occurrence declined from 59.4% to 31.3%. [Figure 4]

Cox Regression Analysis for 30-Day Mortality

In univariable Cox regression analysis, higher LCR was associated with a lower hazard of 30-day mortality. After adjustment for age, sex, diabetes mellitus, creatinine, lactate, SOFA score and APACHE II score, LCR remained independently associated with mortality. The adjusted hazard ratio for LCR was 0.86 (95% CI, 0.77–0.96;

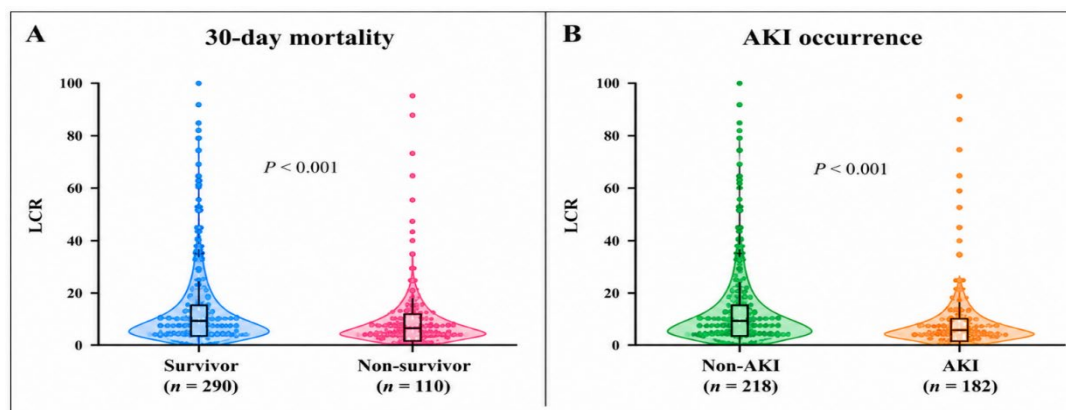


Figure 2: (A) Boxplots illustrating the distribution of LCR among survivors and non-survivors at 30 days. (B) Boxplots illustrating the distribution of LCR among patients with and without acute kidney injury (AKI).

Table 6: Biochemical parameters, severity scores, treatments and outcomes according to 30-day survival and AKI status

Variables	Survivor (n=290)	Non-survivor (n=110)	P value	Non-AKI (n=218)	AKI (n=182)	P value
Uric acid, $\mu\text{mol/L}$	286.4 (185.1–371.2)	341.8 (217.4–486.2)	<0.001	251.6 (181.2–319.4)	398.2 (263.5–526.7)	<0.001
D-dimer, mg/L	3.7 (2.0–7.1)	6.4 (2.8–10.9)	<0.001	3.4 (1.8–6.2)	6.0 (2.8–10.7)	<0.001
Potassium, mmol/L	3.7 (3.3–4.1)	3.9 (3.4–4.5)	0.003	3.6 (3.3–4.0)	3.9 (3.3–4.5)	<0.001
Lactate, mmol/L	1.9 (1.3–3.2)	2.8 (1.8–4.3)	<0.001	1.8 (1.2–3.7)	2.5 (1.6–4.0)	<0.001
APACHE II score	22 (18–28)	28 (23–33)	<0.001	22 (18–27)	26 (21–31)	<0.001
SOFA score	10 (8–13)	14 (11–16)	<0.001	10 (8–13)	13 (10–15)	<0.001
CRRT, n (%)	7 (2.4)	12 (10.9)	0.001	3 (1.4)	16 (8.8)	<0.001
Vasoactive drugs, n (%)	171 (59.0)	90 (81.8)	<0.001	123 (56.4)	138 (75.8)	<0.001
Invasive ventilation, n (%)	176 (60.7)	88 (80.0)	<0.001	132 (60.6)	132 (72.5)	0.013
30-day mortality, n (%)	—	—	—	42 (19.3)	68 (37.4)	<0.001
AKI, n (%)	114 (39.3)	68 (61.8)	<0.001	—	—	—
Length of ICU stay, days	5 (3–10)	8 (4–14)	0.003	5 (3–11)	7 (4–12)	<0.001
Length of hospital stay, days	18 (12–28)	13 (7–20)	<0.001	17 (11–26)	16 (9–25)	0.014
ICU mortality, n (%)	—	—	—	47 (21.6)	61 (33.5)	0.009
Hospital mortality, n (%)	—	—	—	52 (23.9)	65 (35.7)	0.009

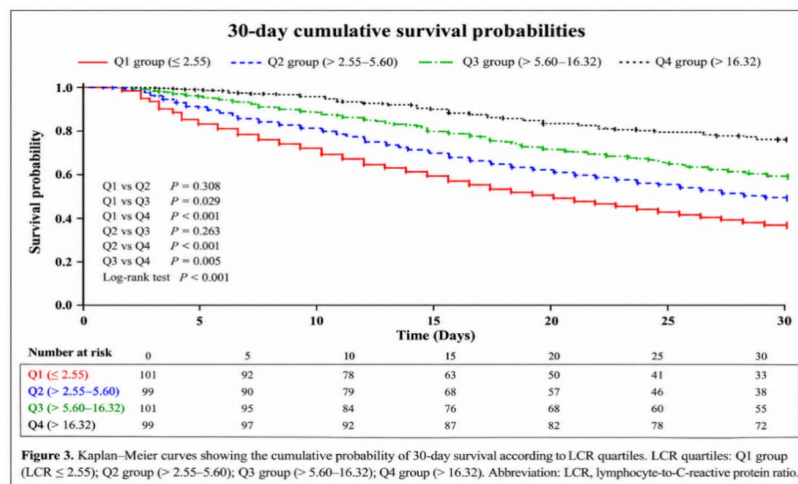


Figure 4: (A) Proportion of 30-day mortality across the different LCR quartile groups. (B) Proportion of AKI occurrence across the different LCR quartile groups. LCR quartiles: Q1 group (LCR ≤ 2.55); Q2 group ($2.55 < \text{LCR} \leq 5.60$); Q3 group ($5.60 < \text{LCR} \leq 16.32$); Q4 group ($\text{LCR} > 16.32$).

Table 7: ROC analysis of LCR for prediction of 30-day mortality

Parameter	Value
AUC	0.671
95% CI	0.612–0.730
P value	<0.001
Optimal cutoff	3.42
Sensitivity	78.2%
Specificity	52.4%
Positive predictive value	38.4%
Negative predictive value	86.5%

Table 8: ROC analysis of LCR for prediction of AKI

Parameter	Value
AUC	0.648
95% CI	0.594–0.702
P value	<0.001
Optimal cutoff	4.18
Sensitivity	71.4%
Specificity	55.0%
Positive predictive value	57.6%
Negative predictive value	69.3%

P=0.008), indicating an inverse association between LCR and the risk of 30-day mortality.[Table 9]

Logistic Regression Analysis for AKI

Univariable logistic regression demonstrated an inverse association between LCR and the development of AKI.

Table 9: Cox proportional hazards regression analysis for 30-day mortality

Variable	Unadjusted HR (95% CI)	P value	Adjusted HR (95% CI)	P value
Age	1.031 (1.015–1.048)	<0.001	1.024 (1.007–1.042)	0.006
Male sex	1.04 (0.71–1.52)	0.833	1.08 (0.72–1.62)	0.702
Diabetes mellitus	1.28 (0.87–1.89)	0.211	1.19 (0.79–1.79)	0.395
Creatinine	1.18 (1.08–1.29)	<0.001	1.11 (1.01–1.23)	0.031
Lactate	1.19 (1.11–1.28)	<0.001	1.13 (1.04–1.23)	0.004
SOFA score	1.18 (1.12–1.25)	<0.001	1.14 (1.07–1.21)	<0.001
APACHE II score	1.09 (1.06–1.13)	<0.001	1.06 (1.02–1.10)	0.002
LCR	0.82 (0.75–0.90)	<0.001	0.86 (0.77–0.96)	0.008

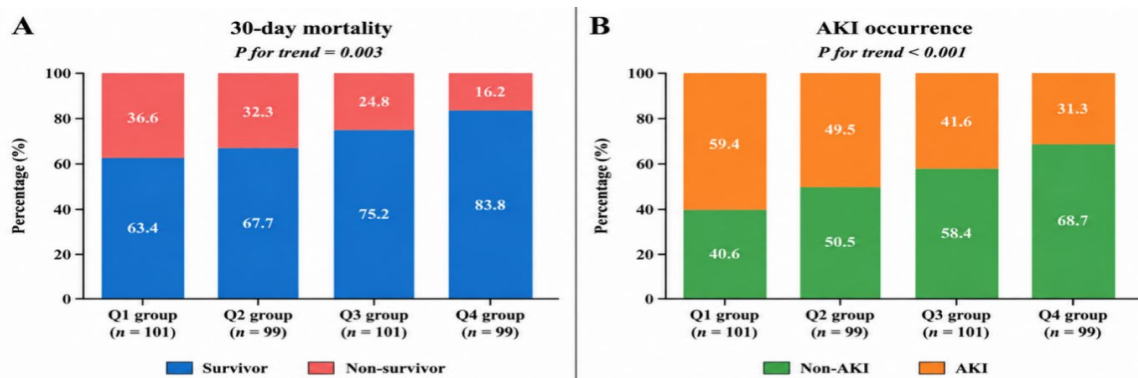


Figure 3: Kaplan–Meier curves showing the cumulative probability of 30-day survival according to LCR quartiles. LCR quartiles: Q1 group (LCR ≤ 2.55); Q2 group (2.55 < LCR ≤ 5.60); Q3 group (5.60 < LCR ≤ 16.32); Q4 group (LCR > 16.32).

Table 10: Logistic regression analysis for development of AKI

Variable	Unadjusted HR (95% CI)	P value	Adjusted HR (95% CI)	P value
Age	1.031 (1.015– 1.048)	<0.001	1.024 (1.007– 1.042)	0.006
Male sex	1.04 (0.71–1.52)	0.833	1.08 (0.72– 1.62)	0.702
Diabetes mellitus	1.28 (0.87–1.89)	0.211	1.19 (0.79– 1.79)	0.395
Creatinine	1.18 (1.08–1.29)	<0.001	1.11 (1.01– 1.23)	0.031
Lactate	1.19 (1.11–1.28)	<0.001	1.13 (1.04– 1.23)	0.004
SOFA score	1.18 (1.12–1.25)	<0.001	1.14 (1.07– 1.21)	<0.001
APACHE II score	1.09 (1.06–1.13)	<0.001	1.06 (1.02– 1.10)	0.002
LCR	0.82 (0.75–0.90)	<0.001	0.86 (0.77– 0.96)	0.008

This association remained statistically significant following adjustment for relevant demographic, clinical, laboratory and severity variables. The adjusted OR for LCR was 0.78 (95% CI, 0.66–0.92; $P=0.003$), indicating that higher LCR was independently associated with a lower likelihood of developing AKI. [Table 10]

Regression Analysis

Cox proportional hazards regression demonstrated an inverse association between LCR and 30-day mortality. In the unadjusted model, each unit increase in LCR was associated with a lower hazard of mortality. This association remained significant after adjustment for demographic, clinical, laboratory, and severity-related variables. In the fully adjusted model, continuous LCR was associated with a hazard ratio of 0.860 (95% CI 0.770–0.960; $P=0.008$).

When LCR was analyzed categorically, Q1 was considered the reference group. The adjusted hazard of mortality was progressively lower in Q3 and Q4, with the lowest hazard observed in Q4.

For AKI, logistic regression similarly demonstrated an inverse association between LCR and AKI occurrence. In the fully adjusted model, continuous LCR was associated with an OR of 0.780 (95% CI 0.660–0.920; $P=0.003$). Compared with Q1, patients in Q3 and Q4 had significantly lower odds of developing AKI. [Table 11, Figure 5]

Model 1

Unadjusted.

Model 2

Adjusted for age, gender, BMI, hypertension, diabetes, WBC count, neutrophil count, and invasive ventilation.

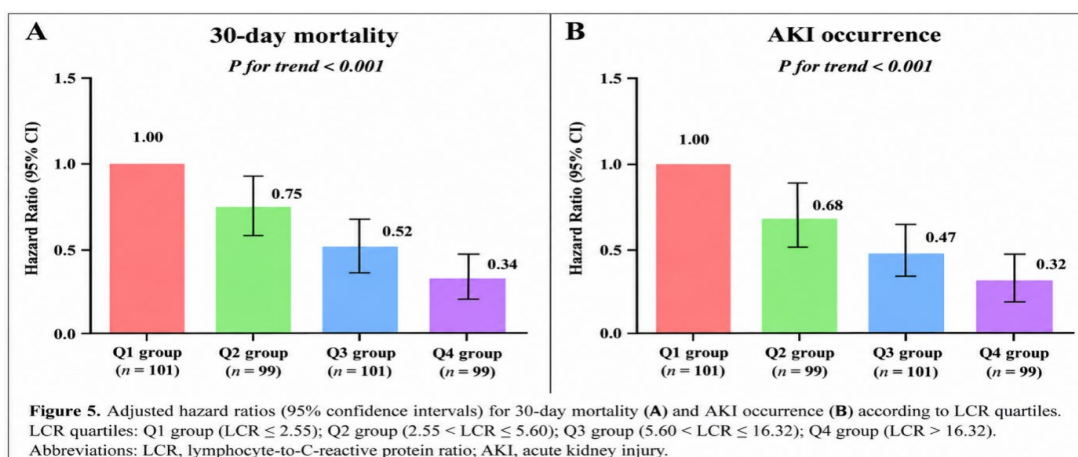


Figure 5: Adjusted Hazard Ratios for 30-Day Mortality and AKI According to LCR Quartiles

Table 7: Cox proportional hazards and logistic regression models for 30-day mortality and AKI occurrence

Outcome / Variable	Model 1 HR/OR (95% CI)	P	Model 2 HR/OR (95% CI)	P	Model 3 HR/OR (95% CI)	P
<i>30-day mortality</i>						
Continuous LCR	0.820 (0.750–0.900)	<0.001	0.850 (0.760–0.950)	0.005	0.860 (0.770–0.960)	0.008
Q1	Ref		Ref		Ref	
Q2	0.91 (0.63–1.32)	0.623	0.98 (0.67–1.43)	0.918	1.06 (0.72–1.55)	0.768
Q3	0.62 (0.41–0.94)	0.024	0.67 (0.44–1.01)	0.056	0.70 (0.51–0.95)	0.022
Q4	0.39 (0.25–0.61)	<0.001	0.45 (0.30–0.69)	<0.001	0.48 (0.33–0.69)	<0.001
P for trend		<0.001		<0.001		<0.001
<i>AKI occurrence</i>						
Continuous LCR	0.720 (0.620–0.830)	<0.001	0.760 (0.640–0.900)	0.001	0.780 (0.660–0.920)	0.003
Q1	Ref		Ref		Ref	
Q2	0.78 (0.54–1.12)	0.175	0.71 (0.49–1.02)	0.064	0.70 (0.49–1.01)	0.055
Q3	0.53 (0.36–0.77)	<0.001	0.49 (0.34–0.71)	<0.001	0.47 (0.33–0.69)	<0.001
Q4	0.36 (0.24–0.54)	<0.001	0.34 (0.23–0.50)	<0.001	0.33 (0.23–0.50)	<0.001
P for trend		<0.001		<0.001		<0.001

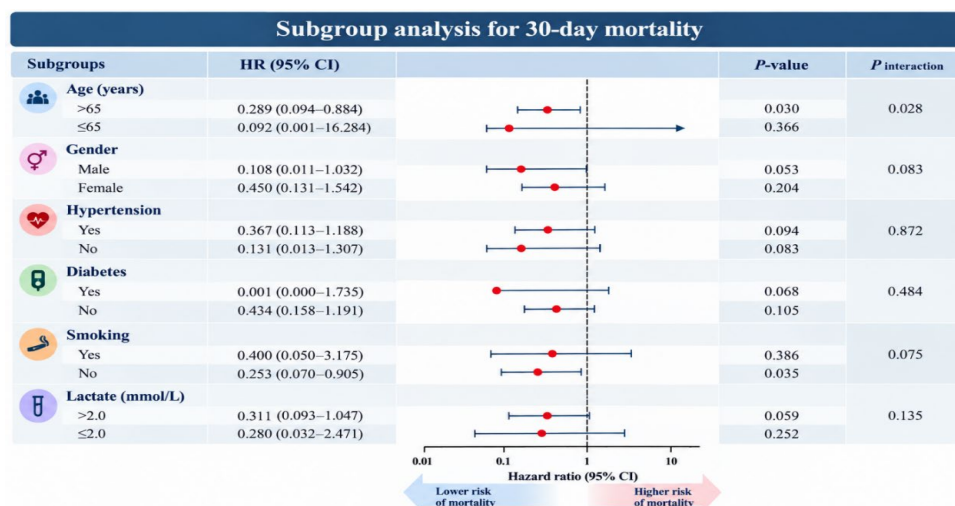


Figure 6. Subgroup analysis of the association between LCR and 30-day mortality.
Abbreviation: LCR, lymphocyte-to-C-reactive protein ratio.

Figure 6: Subgroup Analysis of the Association Between LCR and 30-Day Mortality

Model 3

Adjusted for age, gender, BMI, hypertension, diabetes, WBC count, neutrophil count, hemoglobin, lactate, APACHE II score, SOFA score, and invasive ventilation.

Subgroup Analysis

Subgroup analysis demonstrated that the association between LCR and 30-day mortality was generally consistent across the evaluated clinical subgroups. A significant interaction was observed for age, with a stronger association observed among patients aged >65 years. No significant interaction was observed for gender, hypertension, diabetes, smoking, or lactate category. [Figure 6]

DISCUSSION

The present study evaluated the prognostic significance of the lymphocyte-to-C-reactive protein ratio (LCR) in adult patients with sepsis presenting to the emergency department. Lower LCR was associated with poorer outcomes, particularly 30-day mortality and acute kidney injury (AKI). Among 400 patients, 30-day mortality was 27.5% and AKI occurred in 45.5%. Mortality decreased progressively across LCR quartiles, from 36.6% in Q1 to 16.2% in Q4, while AKI decreased from 59.4% to 31.3%. These findings indicate that a lower LCR at presentation was associated with a greater burden of adverse outcomes.

Sepsis involves a complex interaction between systemic inflammation, immune dysfunction and progressive organ injury. Chousterman et al. described sepsis as a dysregulated host response involving excessive cytokine production and amplification of inflammation. [4] In the present study, patients with lower LCR had higher SOFA and APACHE II scores, higher lactate and creatinine concentrations, and greater use of vasoactive drugs, suggesting an association between low LCR and greater physiological severity. Because LCR incorporates lymphocyte count and CRP, it may provide information regarding both immune-cell depletion and systemic inflammation.

Lymphocyte depletion is an important feature of severe sepsis. Hotchkiss et al. demonstrated extensive lymphocyte apoptosis, particularly involving B and CD4⁺ T cells, in patients with sepsis, supporting the role of lymphocyte loss in sepsis-associated immune dysfunction. [15] Lang and Matute-Bello further emphasized the contribution of lymphocyte apoptosis to immune dysfunction during sepsis. [16] In the present study, lymphocyte counts were significantly lower among non-survivors and among patients who developed AKI. de Pablo et al. described substantial alterations in circulating lymphocyte populations during

sepsis, while Venet et al. demonstrated early leukocyte alterations during septic shock. [7,17] These observations support the biological relevance of lymphocyte depletion as a marker of severe systemic illness.

Sheikh MotaharVahedi et al. reported an association between lymphopenia and adverse short-term outcomes in patients with sepsis evaluated in the emergency setting. [18] Adrie et al. found that persistent lymphopenia was associated with ICU-acquired infections and death in critically ill patients. [19] Their findings also suggest that changes in lymphocyte counts over time may provide additional prognostic information. Cilloniz et al. similarly reported an association between lymphopenia and poor outcomes in patients with community-acquired pneumonia and sepsis. [20] The present findings therefore support the role of lymphocyte depletion as an important component of early prognostic assessment.

CRP represents the inflammatory component of LCR. Black et al. described CRP as a major acute-phase protein that increases during inflammatory stimulation. [8] In the present study, CRP was significantly higher among non-survivors and patients with AKI. However, the prognostic value of CRP alone has varied between studies. Silvestre et al. demonstrated limitations in using CRP as an isolated prognostic marker in septic patients. [21] Pettilä et al. also emphasized the importance of interpreting CRP together with other clinical variables. [22] Koozi et al. reported an association between CRP and mortality in patients admitted to intensive care with sepsis, whereas Ryoo et al. found that the prognostic value of CRP was limited after adjustment for other clinical factors. [9,10] These findings suggest that CRP reflects inflammatory burden but does not fully capture the immune status of patients with sepsis.

The potential value of LCR therefore lies in combining lymphocyte depletion with systemic inflammation. In the present study, patients in Q1 had the lowest lymphocyte counts and highest CRP concentrations, whereas Q4 showed the opposite pattern. Song et al. reported that lower LCR was associated with increased 30-day mortality and AKI in patients with sepsis admitted to the ICU. [1] Their reported mortality of 28.1% and AKI rate of 45.6% were very similar to the 27.5% mortality and 45.5% AKI observed in the present study. This consistency supports the reproducibility of the association between LCR and adverse outcomes. Importantly, the present study assessed patients in the emergency department, suggesting that the prognostic relationship may be identifiable earlier in the clinical course.

Previous studies have also demonstrated prognostic

associations of LCR in other conditions. Yildirim et al. reported that lower LCR was associated with intestinal ischemia, while Yildirim and Koca demonstrated an association between LCR and postoperative complications following gastrointestinal oncological surgery. [23,11] Angin et al., Okugawa et al. and Takeuchi et al. reported prognostic associations of LCR in gastric, colorectal and esophageal cancers, respectively. [25-27] Utsumi et al. demonstrated that preoperative LCR was associated with hepatocellular carcinoma recurrence. [12] Although these populations differ substantially from patients with sepsis, the findings consistently suggest that LCR may reflect the balance between inflammation and immune status.

LCR has also been studied in infectious diseases. Lagunas-Rangel reported an association between reduced LCR and severe COVID-19, while Ullah et al. found lower LCR to be associated with adverse COVID-19 outcomes. [28,29] In contrast, AbensurVuillaume et al. found that although LCR was lower in severe COVID-19, its accuracy for predicting severity and mortality was inadequate. [30] This variation indicates that LCR performance may depend on the underlying disease and clinical setting. The present findings therefore support its potential application in sepsis rather than assuming universal prognostic utility.

The association between LCR and AKI was particularly noteworthy. Patients in Q1 had almost twice the frequency of AKI compared with Q4. Song et al. similarly reported an inverse association between LCR and AKI after adjustment. [1] In the present study, AKI was accompanied by higher creatinine, BUN, lactate, SOFA and APACHE II scores, together with lower lymphocyte counts and higher CRP concentrations. These findings suggest that low LCR may identify patients with a greater overall burden of systemic inflammation and organ dysfunction, although the observational design does not establish causality.

Lower LCR was also associated with greater disease severity. Jones et al. demonstrated the prognostic importance of SOFA among patients with severe sepsis and hypoperfusion at emergency department presentation. [31] The present findings suggest that LCR may provide complementary information regarding inflammatory and immune status but should not replace established severity scores.

LCR demonstrated moderate discriminatory ability, with an AUC of 0.671 for 30-day mortality and 0.648 for AKI. The corresponding cutoffs were 3.42 and 4.18, respectively. Song et al. reported an AUC of 0.620 for mortality. [1] The differences may reflect variations in patient selection and clinical setting. Kaplan-Meier analysis

further demonstrated significantly poorer 30-day survival among patients with lower LCR. The association remained significant after multivariable adjustment, suggesting that LCR may provide prognostic information beyond several conventional clinical and laboratory variables.

The stronger association observed among older patients is also noteworthy. Song et al. reported a stronger relationship between LCR and mortality in patients older than 65 years. [1] This finding may reflect differences in immune response, comorbidity burden and physiological reserve with increasing age, although subgroup findings should be interpreted cautiously.

LCR has practical advantages because both lymphocyte count and CRP are routinely available and its calculation requires no specialized testing. Nevertheless, it should be used as an adjunct rather than a standalone prognostic tool. The retrospective single-center design, potential residual confounding and assessment of only baseline LCR are important limitations. Adrie et al. demonstrated the prognostic relevance of persistent lymphopenia, supporting future evaluation of serial LCR measurements. [19] Prospective multicenter studies should therefore validate these findings and determine whether serial LCR assessment improves early prediction of mortality and AKI in sepsis.

CONCLUSION

Lower lymphocyte-to-C-reactive protein ratio (LCR) was associated with increased 30-day mortality and acute kidney injury among patients with sepsis presenting to the emergency department. Lower LCR was also associated with greater disease severity, higher inflammatory burden and adverse clinical outcomes. The ratio demonstrated moderate predictive ability for mortality and AKI and remained significantly associated with outcomes after adjustment for relevant clinical variables. LCR may therefore serve as a simple, readily available adjunctive biomarker for early risk stratification in sepsis. However, it should complement established clinical assessment and severity scores. Further prospective multicenter studies are needed to validate its prognostic value and determine clinically useful cutoff values.

REFERENCES

1. Song C, Hu Z, Zhang J. The value of lymphocyte-to-C-reactive protein ratio for predicting clinical outcomes in patients with sepsis in intensive care unit: a retrospective single-center study. *Front MolBiosci.* 2024;11:1429372.
2. Singer M, Deutschman CS, Seymour CW, et al. The third international Consensus Definitions for sepsis and septic

- shock (Sepsis-3). *JAMA*. 2016;315(8):801–810.
3. Fleischmann C, Scherag A, Adhikari NK, et al. Assessment of global incidence and mortality of hospital-treated sepsis. Current estimates and limitations. *Am J Respir Crit Care Med*. 2016;193(3):259–272.
4. Chousterman BG, Swirski FK, Weber GF. Cytokine storm and sepsis disease pathogenesis. *Semin Immunopathol*. 2017;39(5):517–528.
5. Hahn WO, Mikacenic C, Price BL, et al. Host derived biomarkers of inflammation, apoptosis, and endothelial activation are associated with clinical outcomes in patients with bacteremia and sepsis regardless of microbial etiology. *Virulence*. 2016;7(4):387–394.
6. Chung KP, Chang HT, Lo SC, et al. Severe lymphopenia is associated with elevated plasma interleukin-15 levels and increased mortality during severe sepsis. *Shock*. 2015;43(6):569–575.
7. De Pablo R, Monserrat J, Prieto A, Alvarez-Mon M. Role of circulating lymphocytes in patients with sepsis. *Biomed Res Int*. 2014;2014:671087.
8. Black S, Kushner I, Samols D. C-Reactive protein. *J Biol Chem*. 2004;279(47):48487–48490.
9. Koozi H, Lengquist M, Frigyesi A. C-reactive protein as a prognostic factor in intensive care admissions for sepsis: a Swedish multicenter study. *J Crit Care*. 2020;56:73–79.
10. Ryoo SM, Han KS, Ahn S, et al. The usefulness of C-reactive protein and procalcitonin to predict prognosis in septic shock patients: a multicenter prospective registry-based observational study. *Sci Rep*. 2019;9(1):6579.
11. Yildirim M, Koca B. Lymphocyte C-reactive protein ratio: a new biomarker to predict early complications after gastrointestinal oncologic surgery. *Cancer Biomark*. 2021;31(4):409–417.
12. Utsumi M, Inagaki M, Kitada K, et al. Preoperative lymphocyte-to-C-reactive protein ratio predicts hepatocellular carcinoma recurrence after surgery. *Ann Surg Treat Res*. 2022;103(2):72–80.
13. Li X, Wei Y, Xu Z, et al. Lymphocyte-to-C-Reactive protein ratio as an early sepsis biomarker for neonates with suspected sepsis. *MediatInflamm*. 2023;2023:9077787.
14. Liu X, Mu Y. Lymphocyte to C-reactive protein ratio as an early biomarker to distinguish sepsis from pneumonia in neonates. *J Inflamm Res*. 2023;16:3509–3517.
15. Hotchkiss RS, Tinsley KW, Swanson PE, Schmieg RE, Hui JJ, Chang KC, et al. Sepsis-induced apoptosis causes progressive profound depletion of B and CD4+ T lymphocytes in humans. *J Immunol*. 2001;166(11):6952–6963.
16. Lang JD, Matute-Bello G. Lymphocytes, apoptosis and sepsis: making the jump from mice to humans. *Crit Care*. 2009;13(1):109.
17. Venet F, Lukaszewicz AC, Payen D, Hotchkiss R, Monneret G. Monitoring the immune response in sepsis: a rational approach to administration of immunoadjuvant therapies. *Ther Clin Risk Manag*. 2010;6:119–127.
18. Sheikh MotaharVahedi HS, Bagheri A, Jahanshir A, Seyedhosseini J, Vahidi E. Association of lymphopenia with short term outcomes of sepsis patients: a brief report. *Arch AcadEmerg Med*. 2019;7(1):e14.
19. Adrie C, Lugosi M, Sonnevile R, Souweine B, Ruckly S, Cartier JC, et al. Persistent lymphopenia is a risk factor for ICU-acquired infections and for death in ICU patients with sustained hypotension at admission. *Ann Intensive Care*. 2017;7(1):30.
20. Cilloniz C, Peroni HJ, Gabarrús A, García-Vidal C, Pericàs JM, Bermejo-Martin J, et al. Lymphopenia is associated with poor outcomes of patients with community-acquired pneumonia and sepsis. *Open Forum Infect Dis*. 2021;8(6):ofab169. doi:10.1093/ofid/ofab169.
21. Silvestre J, Póvoa P, Coelho L, Almeida E, Moreira P, Fernandes A, et al. Is C-reactive protein a good prognostic marker in septic patients? *Intensive Care Med*. 2009;35(5):909–913.
22. Pettilä V, Pentti J, Pettilä M, Takkunen O, Jousela I. Predictive value of antithrombin III and serum C-reactive protein concentration in critically ill patients with suspected sepsis. *Crit Care Med*. 2002;30(2):271–275.
23. Yildirim M, Dasiran F, Angin YS, Okan I. Lymphocyte-C-reactive protein ratio: a putative predictive factor for intestinal ischemia in strangulated abdominal wall hernias. *Hernia*. 2021;25(3):649–656.
24. Liu Y, et al. Preoperative lymphocyte to C-reactive protein ratio: a new prognostic indicator of post-primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. *IntImmunopharmacol*. 2023;114:109594.
25. Angin YS, Yildirim M, Dasiran F, Okan I. Could lymphocyte to C-reactive protein ratio predict the prognosis in patients with gastric cancer? *ANZ J Surg*. 2021;91:1521–1527.
26. Okugawa Y, Toiyama Y, Yamamoto A, Shigemori T, Ide S, Kitajima T, et al. Lymphocyte-C-reactive protein ratio as promising new marker for predicting surgical and oncological outcomes in colorectal cancer. *Ann Surg*. 2020;272(2):342–351.
27. Takeuchi M, Kawakubo H, Hoshino S, Matsuda S, Mayanagi S, Irino T, et al. Lymphocyte-to-C-reactive protein ratio as a novel marker for predicting oncological outcomes in patients with esophageal cancer. *World J Surg*. 2021;45(11):3370–3377.
28. Lagunas-Rangel FA. Neutrophil-to-lymphocyte ratio and lymphocyte-to-C-reactive protein ratio in patients with severe coronavirus disease 2019 (COVID-19): a meta-analysis. *J Med Virol*. 2020;92(10):1733–1734.
29. Ullah W, Basyal B, Tariq S, Almas T, Saeed R, Roomi S, et al. Lymphocyte-to-C-reactive protein ratio: a novel

- predictor of adverse outcomes in COVID-19. *J Clin Med Res.* 2020;12(7):415-422.
30. AbensurVuillaume L, Lefebvre F, Benhamed A, Schnee A, Hoffmann M, Godoy Falcao F, et al. Lymphocyte-to-C-reactive protein (LCR) ratio is not accurate to predict severity and mortality in patients with COVID-19 admitted to the ED. *Int J Mol Sci.* 2023;24(6):5996.
31. Jones AE, Trzeciak S, Kline JA. The Sequential Organ Failure Assessment score for predicting outcome in patients with severe sepsis and evidence of hypoperfusion at the time of emergency department presentation. *Crit Care Med.* 2009;37(5):1649-1654.