

Role of Lactate-to-Albumin Ratio and SOFA Score in Predicting Clinical Outcomes in Sepsis and Septic Shock: A Narrative Review

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ABSTRACT

Infection-associated organ dysfunction or sepsis, was responsible for about 166 million sepsis cases and 21.4 million all-cause sepsis-related deaths globally, representing 31.5% of total global deaths in 2025. Heterogeneous pathophysiology (proinflammatory cytokine storm, microvascular dysfunction, metabolic derangements) makes prognosis difficult. This review assesses how well the LAR predicts clinical outcomes in sepsis and septic shock when compared to the Sequential Organ Failure Assessment (SOFA) score. With a focus on mortality prediction and comparative performance, studies evaluating lactate, albumin, LAR, and SOFA score in adult sepsis populations were considered. Tissue hypoperfusion is reflected in serum lactate, but systemic inflammation and low physiological reserve are indicated by hypoalbuminaemia. By combining these factors, LAR provides better predictive accuracy than any marker by itself. LAR is an independent predictor of 28-day death, with area under the curve values ranging from 0.70 to 0.90, according to many cohort studies. The suggested cut-offs range from 0.96 to 1.5, and higher levels are always linked to greater mortality. The SOFA score is still a reliable method for evaluating organ failure and forecasting results. According to new research, LAR may provide incremental or even better prognostic value in some situations and correlates with SOFA. LAR is an accessible and promising biomarker for early sepsis risk assessment. It may improve prognosis accuracy when combined with the SOFA score. However, prior to widespread clinical use, cut-off standardisation and validation via prospective multicentre trials are required.

Keywords: Organ dysfunction, Biomarkers, Prognosis

INTRODUCTION

According to the Sepsis-3 definition, sepsis is a potentially fatal organ failure brought on by a dysregulated host response to infection [1]. It resulted in around 166 million sepsis cases and 21.4 million all-cause sepsis-related deaths globally, representing 31.5% of total global deaths in 2025 [2]. The burden is greatest in low- and middle-income areas; extensive Intensive Care Unit (ICU) studies in India, show that in-hospital sepsis mortality is around 24% [3]. Early detection and risk assessment are essential since treatment delays of one hour may result in a 6-10% increase in mortality [2]. The LAR and the SOFA score are two new prognostic measures in sepsis that are examined in this study.

Although lactate, albumin and SOFA score are individually well-recognised prognostic tools in sepsis, their combined interpretation remains insufficiently synthesised in the current literature. Most available studies evaluate LAR as an isolated mortality marker, while fewer directly examine how it complements or compares with SOFA, the established organ dysfunction score used in Sepsis-3 [1,4-9]. This creates an important gap for clinicians, particularly in emergency and intensive care settings, where early risk stratification must be rapid, inexpensive and applicable even in resource-limited settings. This review was therefore undertaken to bridge this gap by integrating evidence on the biological rationale, prognostic performance and clinical utility of LAR in relation to SOFA score. The novelty of this review lies in its focused comparison of a simple biochemical ratio with a validated organ failure score, highlighting how the two may provide complementary prognostic information rather than functioning as competing tools.

Pathophysiology of Sepsis and Septic Shock

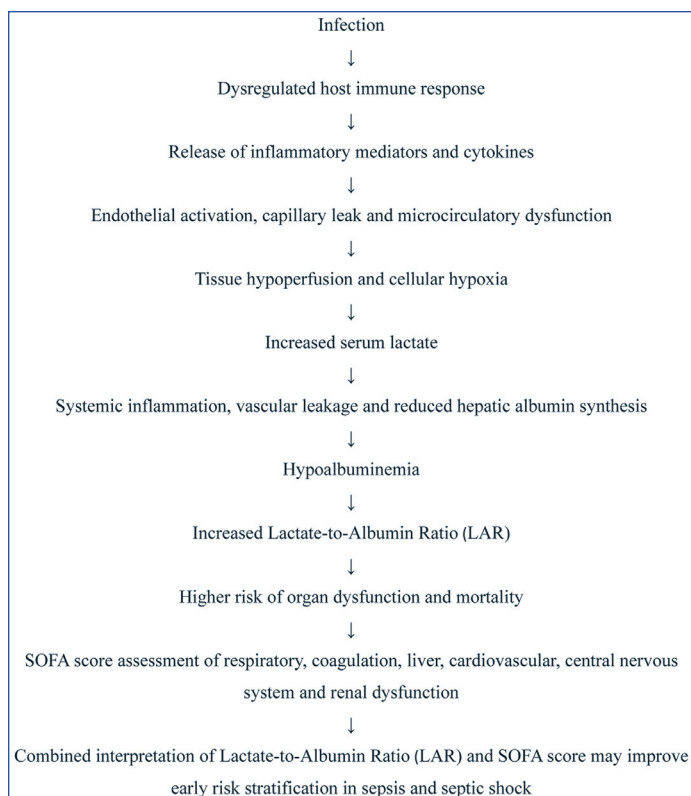
Sepsis arises from a dysregulated host response to infection, resulting in life-threatening organ dysfunction [10]. The initial recognition of pathogen-associated molecular patterns and

damage-associated molecular patterns activates innate immune pathways, leading to the release of inflammatory mediators such as tumour necrosis factor- α , interleukin-1 β and interleukin-6 [11]. This inflammatory response causes endothelial activation, increased vascular permeability, capillary leak, vasodilatation and impaired microcirculatory blood flow, which together contribute to tissue hypoperfusion and cellular hypoxia [11]. As oxygen delivery becomes inadequate for tissue demand, anaerobic metabolism increases and serum lactate levels rise [10].

In addition to inflammation and microcirculatory failure, activation of the coagulation cascade is a central component of sepsis pathophysiology [12]. Sepsis promotes tissue factor expression, thrombin generation, platelet activation and suppression of natural anticoagulant pathways, resulting in sepsis-associated coagulopathy and, in severe cases, disseminated intravascular coagulation [12]. Microvascular thrombosis further compromises tissue perfusion and contributes to multiorgan dysfunction. This is clinically relevant to the SOFA score because coagulation dysfunction is assessed through the platelet count domain, alongside respiratory, hepatic, cardiovascular, neurological and renal components [13]. Therefore, sepsis pathophysiology links infection-induced inflammation, endothelial dysfunction, coagulation activation, metabolic stress and progressive organ failure [Table/Fig-1] [10,12,13].

Biomarkers in Sepsis

Common laboratory markers assist sepsis diagnosis and prognosis. C-Reactive Protein (CRP) and Procalcitonin (PCT) rise in response to infection; they have moderate value in distinguishing sepsis from non-infectious inflammation (PCT generally more specific than CRP) [14]. However, neither CRP nor PCT reliably quantify severity or predict outcome in isolation. Serum lactate is a key indicator of tissue hypoperfusion [1], its levels reflect the balance between oxygen delivery and demand. Elevated lactate (typically $>2-4$ mmol/L) is



[Table/Fig-1]: Conceptual flow chart showing the pathophysiological basis for using Lactate-to-Albumin Ratio (LAR) and SOFA in sepsis prognosis.

associated with higher mortality in sepsis. Current guidelines include lactate in sepsis screening and resuscitation targets, since lactate clearance correlates with improved outcomes [15]. Serum albumin (main plasma protein) often falls in sepsis due to capillary leak and catabolism; hypoalbuminaemia (<35 g/L) is common and portends worse outcomes [1]. Low albumin may reflect both malnutrition and severity of inflammation. Although lactate may change more rapidly in response to tissue hypoperfusion and resuscitation, albumin levels can also decline early during sepsis as part of the acute-phase response and vascular leak; therefore, albumin reflects both acute inflammatory burden and underlying physiological reserve [1]. Thus, while lactate and albumin each carry prognostic information, they have limitations when used alone.

Role of Serum Lactate in Sepsis

Lactate accumulates as a result of anaerobic glycolysis during hypoperfusion and shock. Numerous studies show that higher lactate predicts mortality. For example, in one cohort of Emergency Department (ED)-admitted sepsis patients, initial lactate ≥ 4.0 mmol/L (vs <2.0) quintupled the odds of 28-day death [15]. In the Surviving Sepsis Campaign data, lactate >4.0 mmol/L in hypotension was linked to increase in-hospital mortality. Serial lactate measurements (clearance over 6–24 h) also inform prognosis: poor lactate clearance predicts higher mortality [15]. Lactate's predictive ability is generally comparable to the SOFA score: one study found lactate AUC ~0.66 vs SOFA AUC ~0.69 for 30-day mortality [15]. Lactate may rise for non-sepsis reasons (e.g. seizures, drugs), and normalisation can lag if clearance mechanisms (e.g. liver function) are impaired [16].

Role of Serum Albumin in Sepsis

Albumin's drop in acute illness (negative acute-phase protein) has prognostic implications. Observational data show hypoalbuminaemia on admission correlates with higher mortality. In one review, septic non-survivors had significantly lower albumin than survivors [4]. Mechanisms include decreased oncotic pressure (exacerbating oedema), impaired antioxidant/anti-inflammatory effects, and malnutrition. Unlike lactate, albumin level changes more slowly and reflects chronic and acute factors. Its prognostic value is inferior to dynamic markers, but it provides information on host reserves [17].

Lactate-to-Albumin Ratio (LAR): Mechanism and Clinical Significance

The two indicators are combined to create the LAR, which is calculated by dividing serum lactate (mmol/L) by albumin (g/dL). In theory, it combines baseline nutritional/inflammatory state (low albumin) and acute metabolic stress (high lactate) into a single indicator [1]. A higher LAR indicates both severe hypoperfusion and limited physiological reserves. As a composite, LAR may normalise inter-patient variability: for example, two patients with lactate 4.0 mmol/L might differ in risk depending on albumin (e.g., 3.0 vs 4.0 g/dL) [5].

Several studies have tested LAR's predictive value. Most find LAR outperforms lactate or albumin alone. In a Korean ICU cohort, Shin JW et al., found LAR independently predicted 28-day mortality (AUC~0.81; cut-off 1.2 mmol/(g/dL)), superior to lactate alone (AUC 0.73) [18]. In this review of multiple cohorts, non-survivors consistently had higher LAR (e.g., mean ~1.9 vs 1.2 in survivors). These and other series report LAR AUCs for mortality ~0.70-0.90, reflecting good to excellent prognostic power [1,7].

The optimal LAR cut-off varies by cohort. Reported thresholds include ~0.96 [7], 1.2, and 1.5 [4]. For instance, Kabra R et al., reported that LAR ≥ 0.96 yielded ~100% sensitivity and 88% specificity for ICU mortality in sepsis [19]. Hoang QV et al., (Vietnam) used 1.2 as cut-off (sensitivity 91%, specificity 57%). Differences reflect patient mix and albumin distribution [1].

Mechanistically, high LAR has face validity: severe sepsis often shows combined tissue hypoxia and low albumin due to capillary leak. LAR may be especially helpful in settings where baseline albumin varies (e.g., malnourished populations) [20]. Li F et al., confirmed this in a larger sepsis sample, with LAR significantly associated with 28-day death (adjusted OR ~2.1) and better discrimination than lactate or albumin [21].

SOFA Score and its Prognostic Utility

The SOFA score quantifies dysfunction in six organ systems (respiratory, coagulation, liver, cardiovascular, Central Nervous System (CNS), renal), each 0-4, summing to 0-24. In Sepsis-3, an acute increase in SOFA ≥ 2 defines organ dysfunction in sepsis [10]. SOFA was originally developed to track ICU organ failure but also predicts mortality: each 1-point increase in SOFA associates with ~10–20% higher death risk. Large datasets show rising SOFA parallels mortality. For example, in one ICU cohort, patients with SOFA <5 had survival ~80%, whereas those with SOFA >10 had <50% survival [22].

In practice, SOFA is easy to compute but requires labs and parameters. It is routinely used in research and increasingly at bedside for risk stratification. Studies find SOFA on admission correlates with ICU mortality: e.g., median SOFA ~6 (IQR 4-9) with in-hospital mortality ~24% in a large Indian sepsis cohort [3]. SOFA's predictive accuracy (AUC) for mortality is generally moderate (~0.70-0.80), similar to that of lactate [15]. Notably, SOFA can miss early risk: as notes, both SOFA and quick-SOFA have suboptimal sensitivity in some settings.

Comparison of LAR with SOFA Score

Both LAR and SOFA reflect illness severity, but via different domains: LAR through metabolic and nutritional markers, SOFA through organ function metrics. Studies directly comparing them are scarce. However, indirect evidence suggests LAR often provides incremental value to SOFA. For instance, Acharya CP et al., found that LAR had an edge over SOFA for mortality prediction, although lactate alone was similar [8]. In acute respiratory failure (an ICU group analogous to sepsis), higher SOFA and higher LAR both indicated worse prognosis, but LAR was a better stand-alone predictor than SOFA [8].

In multivariable models, combining LAR with SOFA tends to improve discrimination. LAR and SOFA were incorporated in Hoang QV et

al., sepsis model (Vietnam, n=170); the addition of SOFA to a LAR + age model increased AUC from 0.855 to 0.864 [1]. According to a different study [1,4,8,23], LAR outperformed scores alone with an AUC of around 0.91 when paired with APACHE II and other scores. All things considered, LAR seems to supplement SOFA, while there is some indication that LAR may outperform SOFA in particular cohorts. Importantly, SOFA represents cumulative organ deficiencies, whereas LAR is dynamic (lactate may vary quickly); the two might be utilised in combination.

Review of Recent Literature

Several key observational and meta-analytic studies (2001-2025) underpin these conclusions [Table/Fig-2] [1,9,18,19,21,22,24]. These studies cover a variety of contexts (high-income vs low-middle income countries, ICU vs ED) [1,9,18,19,22-24]. LAR is a reliable prognostic biomarker that often enhances traditional scores' predictive power, according to their consistent message. They further highlight the lack of a single, global cut-off since LAR may be calibrated differently in each ICU or area.

In contrast, there is a wealth of research on the SOFA score itself. A 2026 review revealed that a 2-point rise in SOFA increases mortality risk [25], and several studies confirm the connection between SOFA and death [4,15,16,18,20,23,25]. Since SOFA-guided treatment is mostly used as a research/assessment tool, we were unable to locate a recent RCT using it.

Clinical Consequences

Risk stratification: Determine LAR (lactate in mmol/L divided by albumin in g/dL) for sepsis patients, particularly those in the intensive care unit. Patients should be regarded as high-risk if their LAR is noticeably higher (e.g., >1.0-1.5). This may lead to more intensive treatment or surveillance. Along with lactate and vital signs, a LAR threshold might be included into sepsis bundles or computerised warnings in the ED/ICU.

Combined scoring: Use LAR for metabolic stress and SOFA (or quick-SOFA) for organ failure evaluation. The prognosis for a patient with high SOFA and high LAR is likely to be bad; situations in the middle may be prioritised differently. Some writers provide models that include the place of respiratory illness or composite scores such as "LAR age."

Treatment recommendations: LAR may guide resuscitation, albeit this is questionable. For instance, occult hypoperfusion or the requirement for fluids or inotropes may be indicated if LAR does not improve with first treatment. In contrast to lactate alone,

albumin-related lactate clearance problems are partly explained by LAR.

Allocating resources: Only bedside labs are used in a calculated LAR in areas with minimal resources, like as India. LAR-guided strategies may improve antibiotic priority or ICU bed use if they are verified.

As of now, LAR is not an official guideline metric. It is important to apply cut-offs from the literature with caution. Local validation is required for LAR's additive value above SOFA, if any. LAR may be impacted by chronic liver disease, albumin infusions, and other conditions. Additionally, SOFA is still essential for monitoring organ failures (such CNS or coagulation) that are not represented in LAR and for identifying sepsis ($\Delta\text{SOFA} \geq 2$).

Current Evidence's Limitations

The majority of the evidence to far comes from observational studies, case series, and retrospective analyses, all of which have inherent biases. Numerous LAR studies have modest sample sizes and are single-centre. The possibility of publication bias exists (negative research may be underreported). Applicability to non-ICU sepsis is unknown since the majority of data are from ICU populations. There is no agreement, and the variation in reported LAR cut-offs (0.96 to >2) reflects various demographics and measuring techniques. Few studies specifically examine whether LAR adds independent predictive value beyond SOFA or APACHE-II, and direct comparisons between LAR and conventional severity ratings are uncommon. LAR is not included in any recommendations or big multicentre studies.

Future directions

These deficiencies should be filled by future studies. To evaluate LAR thresholds and their prognostic value against known scores, prospective multicentre studies are required, preferably in a variety of contexts (such as LMICs and paediatric care). It is worthwhile to investigate the incorporation of LAR into prediction models. For instance, dynamic LAR trends (serial measurements) may provide more prognostic information than a single value. The effectiveness of LAR-driven treatment algorithms (e.g., more vigorous resuscitation for high LAR) might be tested in interventional studies. Interpretation might be improved by mechanistic research into how albumin alters lactate prognostic value (e.g., evaluating the impact of capillary leak, hepatic clearance). Lastly, guideline committees may take LAR into account when revising sepsis care packages, particularly if further proof of its beneficial effects becomes available.

Author, year	Study design	Study period	Study population	Key findings	Prognostic performance
Shin JW et al., 2018 [18]	Retrospective observational study	-	Critically-ill patients with sepsis	Lactate-to-Albumin Ratio (LAR) independently predicted 28-day mortality	AUC approximately 0.81; cut-off approximately 1.2.
Li F et al., 2023 [21]	Retrospective cohort study	January 2018 to December 2022	Patients with sepsis	Lactate-to-Albumin Ratio (LAR) showed better mortality prediction than lactate or albumin alone	AUC approximately 0.65; adjusted odds ratio approximately 2.1.
Bou Chebl R et al., 2021 [22]	Prospective observational study	September 2018 till February 2021	Septic patients presenting to the emergency department	Non-survivors had significantly higher LAR than survivors	Mean LAR: 1.93 in non-survivors versus 1.20 in survivors.
Kabra R et al., 2023 [19]	Observational study	December 2020 to December 2022	Patients with sepsis	LAR was a strong predictor of mortality	Cut-off ≥ 0.96 ; sensitivity 100%.
Chen S et al., 2023 [9]	Retrospective cohort study	2001 to 2012	Critically-ill patients with septic myocardial injury	Higher LAR was associated with increased all-cause mortality	AUC approximately 0.89.
Hu J et al., 2024 [24]	Retrospective study	January 2021 and December 2022	Patients with septic shock	Higher LAR was associated with worse short-term outcomes	AUC approximately 0.80.
Hoang QV et al., 2025 [1]	Prognostic model study using Bayesian model averaging	January 2023 to December 2024	ICU patients with sepsis	LAR contributed to 28-day mortality prediction models	LAR-based model showed good discrimination; addition of SOFA marginally improved AUC.

[Table/Fig-2]: Summary of recent studies evaluating Lactate-To-Albumin Ratio (LAR) in sepsis and related critical illness [1,9,18,19,21,22,24].

CONCLUSION(S)

A new predictive biomarker for sepsis that measures the patient's baseline reserves as well as acute metabolic stress is the LAR. In observational studies, it often outperforms either lactate or albumin alone in predicting death. The SOFA score is still a reliable indicator of risk and organ dysfunction. Early detection of high-risk sepsis may be improved by combining LAR and SOFA. Clinicians should be aware of the possibility of LAR. For instance, a LAR ≥ 1.0 (with albumin in g/dL and lactate in mmol/L) may indicate that a patient has a very high chance of dying. Although promising, LAR has to be used carefully, preferably in combination with organ failure ratings and a thorough clinical evaluation. How best to include LAR into sepsis care and prognosis will be determined by further excellent research.

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AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? NA
- For any images presented appropriate consent has been obtained from the subjects. NA

PLAGIARISM CHECKING METHODS:

- Plagiarism X-checker: May 14, 2026
- Manual Googling: Jul 09, 2026
- iThenticate Software: Jul 11, 2026 (3%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

Date of Submission: **Apr 28, 2026**

Date of Peer Review: **Jun 19, 2026**

Date of Acceptance: **Jul 13, 2026**

Date of Publishing: **Aug 01, 2026**