

Online: <https://jurnal.fk.uisu.ac.id/index.php/ibnusina>Ibnu Sina: Jurnal Kedokteran dan Kesehatan-Fakultas Kedokteran Universitas
Islam Sumatera Utara

ISSN 1411-9986 (Print) | ISSN 2614-2996 (Online)



Artikel Penelitian

**HUBUNGAN ANTARA *SHOCK INDEX* DAN LUARAN KLINIS AWAL PADA PASIEN SEPSIS DI
INSTALASI GAWAT DARURAT*****THE RELATIONSHIP BETWEEN THE SHOCK INDEX AND EARLY CLINICAL OUTCOMES IN
PATIENTS WITH SEPSIS IN THE EMERGENCY DEPARTMENT*****Melvin Andrean^{a*}, Jennifer Rose^b, Rifky Budi Triyanto^c**^a*Emergency Department, Cengkareng General Hospital, West Jakarta, Indonesia*^b*Emergency Department, Tugu Koja General Hospital, North Jakarta, Indonesia*^c*Department of Internal Medicine, Cengkareng General Hospital, West Jakarta, Indonesia***Histori Artikel**Diterima:
18 Maret 2026Revisi:
1 Juli 2026Terbit:
1 Januari 2027**Kata Kunci**Sepsis, shock index,
instalasi gawat
darurat, luaran klinis**Keywords***Sepsis, shock index,
emergency
department, clinical
outcomes****Korespondensi**Email:
melvinandrea
n@gmail.com**A B S T R A K**

Sepsis masih merupakan penyebab utama morbiditas dan mortalitas di seluruh dunia, dengan perburukan klinis dini yang sering terjadi dalam beberapa jam pertama setelah presentasi ke instalasi gawat darurat (IGD). Penelitian kohort retrospektif ini mengevaluasi hubungan antara shock index saat masuk dengan luaran klinis awal pada 200 pasien dewasa dengan sepsis di IGD, yang didefinisikan secara operasional sebagai qSOFA ≥ 2 dengan dugaan infeksi, yang datang antara Januari dan Desember 2025. *Shock index* saat masuk dihitung dari tanda vital triase dan dianalisis sebagai variabel kontinu dengan skala peningkatan 0,1 unit. Luaran klinis awal dalam 24 jam meliputi mortalitas, kebutuhan vasopresor, dan perawatan ICU/HCU. Analisis regresi logistik bivariat dan multivariat serta analisis kurva ROC dilakukan. Rerata *shock index* adalah $0,92 \pm 0,27$. *Shock index* yang lebih tinggi secara independen berhubungan dengan peningkatan risiko mortalitas (AOR 1,41; 95% CI 1,24–1,61; $p=0,001$) dan kebutuhan vasopresor (AOR 1,38; 95% CI 1,21–1,58; $p=0,001$). *Shock index* menunjukkan kemampuan diskriminasi yang dapat diterima untuk mortalitas (AUC=0,721) dan kebutuhan vasopresor (AUC=0,718), dengan nilai cut-off optimal SI $\geq 0,95$. Tidak ditemukan hubungan bermakna dengan perawatan ICU/HCU ($p=0,222$). *Shock index* saat masuk dapat menjadi indikator bedside sederhana untuk stratifikasi risiko dini pada pasien sepsis di IGD.

A B S T R A C T

Sepsis remains a major cause of morbidity and mortality worldwide, with early clinical deterioration frequently occurring within the first hours of emergency department (ED) presentation. This retrospective cohort study evaluated the association between admission shock index and early clinical outcomes in 200 adult ED patients with sepsis, operationally defined as qSOFA ≥ 2 with suspected infection, presenting between January and December 2025. Admission shock index was calculated from triage vital signs and analyzed as a continuous variable scaled per 0.1-unit increase. Early outcomes within 24 hours included mortality, vasopressor requirement, and ICU/HCU admission. Bivariate and multivariable logistic regression and ROC curve analyses were performed. The mean admission shock index was 0.92 ± 0.27 . Higher shock index was independently associated with increased mortality (AOR 1.41, 95% CI 1.24–1.61; $p=0.001$) and vasopressor requirement (AOR 1.38, 95% CI 1.21–1.58; $p=0.001$). The shock index demonstrated acceptable discriminatory ability for mortality (AUC=0.721) and vasopressor requirement (AUC=0.718), with an optimal cut-off of SI ≥ 0.95 . No significant association was found with ICU/HCU admission ($p=0.222$). Admission shock index may serve as a simple bedside indicator for early risk stratification in ED sepsis patients.

DOI: <http://doi.org/10.30743/ibnusina.v26i1.1230>This is an open-access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Sepsis remains a major cause of morbidity and mortality worldwide and represents a significant burden in emergency departments (EDs). Recent global estimates suggest that sepsis affects approximately 49 million individuals annually and accounts for nearly 11 million deaths, representing about one in five deaths worldwide.¹ Among ED patients with sepsis, early clinical deterioration — manifesting as death, the need for vasopressor support, or escalation to intensive or high-dependency care — frequently occurs within the first 24 hours of presentation.² Because these early outcomes largely determine a patient's trajectory, identifying which patients are most likely to deteriorate during this critical window is essential for guiding monitoring intensity, early resuscitation, and timely escalation of care.

Several scoring systems have been developed to predict outcomes in sepsis, including the Sequential Organ Failure Assessment (SOFA) score, quick Sequential Organ Failure Assessment (qSOFA), and serum lactate levels.³ However, many of these tools require laboratory measurements or complex calculations, which may limit their utility during the initial triage phase in the ED where rapid clinical decision-making is required.⁴

The Shock Index (SI), defined as the ratio of heart rate to systolic blood pressure (HR/SBP), is a simple hemodynamic parameter that can be calculated rapidly at the bedside using routinely measured vital signs.⁵ Physiologically, an elevated shock index reflects early circulatory compromise and impaired hemodynamic compensation, which may

precede overt hypotension. Previous studies have suggested that elevated SI is associated with adverse outcomes, including mortality and the need for vasopressor support, in patients with sepsis and septic shock.^{6–9}

Although prior studies have established an association between elevated shock index and adverse outcomes across various critically ill populations, findings specific to sepsis remain mixed and incompletely characterized. A recent comparative study evaluating multiple early warning scores and shock indices for sepsis prediction in the ED found that the shock index demonstrated only modest discriminatory performance relative to other composite scores such as NEWS2, though it remained simpler to calculate at the point of triage.¹⁰ Most existing evidence on the shock index in sepsis has been derived from tertiary academic centers, and comparatively little is known about its prognostic performance in secondary referral hospital settings, where case mix, resource availability, and patient acuity at presentation may differ substantially. This gap is particularly relevant given that secondary referral hospitals often manage a disproportionate share of high-acuity sepsis presentations.

Despite its simplicity and physiologic relevance, the shock index remains underutilized in routine ED risk stratification for patients with sepsis, particularly outside tertiary academic settings. Therefore, this study aimed to evaluate the association between admission shock index and early clinical outcomes — mortality, vasopressor requirement, and ICU/HCU admission — in emergency department patients

with sepsis presenting to a secondary referral hospital.

METHODS

Study Design

This study employed an analytic observational approach with a retrospective cohort design to examine the association between admission shock index and early clinical outcomes among patients with sepsis presenting to the emergency department. Although data were extracted retrospectively from electronic medical records, this study retained the defining feature of a cohort design: exposure preceded outcome in time. The admission shock index (exposure) was recorded at ED triage, and each patient's clinical course was then followed forward through the medical record over the subsequent 24 hours to determine whether the outcomes of interest occurred.

Population and Sample

The inclusion criteria in this study are adult patients (≥ 18 years) presenting to the Emergency Department of RSUD Cengkareng between January and December 2025 who met operational screening criteria for sepsis, defined as a quick Sequential Organ Failure Assessment (qSOFA) score ≥ 2 in the presence of suspected or confirmed infection, were eligible for inclusion. A total sampling (consecutive sampling) technique was used, in which all patients meeting the inclusion criteria during the study period were enrolled, rather than a randomly selected subset. This approach was intended to minimize selection bias by capturing the full spectrum of disease severity presenting to the ED during the study period.

Patients were excluded if they presented with cardiac arrest or were declared dead on arrival ($n=3$), had incomplete triage vital sign documentation precluding shock index calculation ($n=6$), or had a documented Do-Not-Resuscitate (DNR) status ($n=2$). Of 211 patients screened during the study period, 200 met eligibility criteria and were included in the final analysis (Figure 1). It is important to note that qSOFA was used in this study as an operational screening criterion for sepsis rather than as the formal Sepsis-3 diagnostic definition, which requires evidence of organ dysfunction via a Sequential Organ Failure Assessment (SOFA) score ≥ 2 in the context of infection. qSOFA remains among the most widely used bedside tools for early sepsis screening in emergency department settings, owing largely to its simplicity and high specificity, despite a comparatively low sensitivity.¹¹ The same operational approach — using qSOFA ≥ 2 as the inclusion criterion for sepsis in a retrospective cohort — has been applied in a prior Indonesian hospital-based study.¹² The potential for misclassification arising from this approach, given qSOFA's known sensitivity limitations, is addressed further in the Limitations.

Data Collection and Variables

Clinical data were obtained from electronic medical records. The independent variable in this study was the admission shock index, calculated as the ratio of heart rate to systolic blood pressure (Shock Index = HR/SBP). Vital signs used for shock index calculation were obtained from the initial triage assessment in the emergency department. Shock

index was analyzed both as a continuous variable, scaled per 0.1-unit increase to improve interpretability of the regression model, and as a categorical variable using a cut-off of ≥ 0.95 , derived empirically from receiver operating characteristic (ROC) curve analysis in this study population (see Data Analysis).

The dependent variables were early clinical outcomes occurring within the first 24 hours of hospital admission, including mortality, vasopressor requirement, and intensive care unit (ICU) or high care unit (HCU) admission. Mortality was defined as death occurring within 24 hours of presentation, as documented in the medical record or hospital mortality registry. Vasopressor requirement was defined as the administration of any vasopressor agent within 24 hours for hemodynamic support, ascertained from medication administration records. ICU or HCU admission was defined as transfer to a critical care unit within 24 hours of emergency department presentation, ascertained from ward transfer documentation. All three outcomes were coded as binary variables (yes/no) based on systematic review of the patient's clinical course by the investigators.

Covariates included age (years), sex (male/female), diabetes mellitus status (yes/no), hypertension status (yes/no), and infection source (pulmonary/non-pulmonary), which were obtained from patient medical records. The infection source was classified according to the primary clinical diagnosis documented by the treating physician at the time of hospital admission. The selection of covariates was constrained by the available events per variable (EPV) given the sample size and outcome

frequencies in this study; the implications of this limitation for residual confounding are discussed further in the Limitations section.

Data Analysis

Data were analyzed using IBM SPSS Statistics version 25.0. Baseline characteristics were compared between the two shock index groups ($SI < 0.95$ and $SI \geq 0.95$) to assess whether they differed meaningfully at presentation. Continuous variables were compared using the independent samples t-test, and categorical variables were compared using the chi-square test.

The association between admission shock index and each early clinical outcome (mortality, vasopressor requirement, and ICU/HCU admission within 24 hours) was first assessed using unadjusted (bivariate) binary logistic regression, followed by multivariable binary logistic regression adjusted for age, sex, diabetes mellitus, hypertension, and infection source. Because logistic regression accommodates only one outcome variable per model, three separate binary logistic regression models were fitted — one each for mortality, vasopressor requirement, and ICU/HCU admission — with admission shock index as the common predictor in each. Shock index was analyzed as a continuous variable scaled per 0.1-unit increase. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs).

To evaluate the discriminatory performance of admission shock index for each outcome, receiver operating characteristic (ROC) curve analysis was performed, and the area under the curve (AUC) with 95% CI was

calculated. The optimal cut-off value for shock index was determined using the Youden index (sensitivity + specificity – 1), which identifies the threshold that maximizes the combined sensitivity and specificity. This cut-off was derived empirically from the study sample and has not been externally validated. A p-value <0.05 was considered statistically significant for all analyses.

Ethical Statement

This study received ethical approval from the Health Research Ethics Committee of Cengkareng General Hospital (Approval No: 001/KEP/RSUD/I/2026).

RESULTS

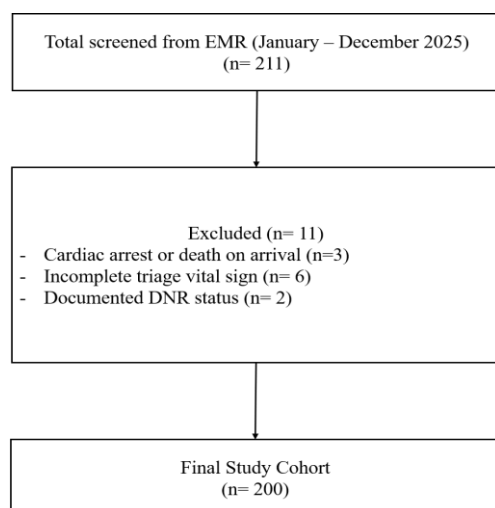


Figure 1. Flowchart of Patients Selection

A total of 200 adult patients with sepsis were included in this study. The mean age was 56.9 ± 15.8 years, and the study population consisted of 95 males (47.5%) and 105 females (52.5%). Diabetes mellitus was present in 63 patients (31.5%), while 86 patients (43.0%) had a history of hypertension. The most common infection source was pulmonary infection (56.5%), while the remaining cases were

categorized as non-pulmonary infection (43.5%). The mean qSOFA score was 2.56 ± 0.50 , with 112 patients (56.0%) scoring 3 and the remainder scoring 2. The mean NEWS2 score was 5.17 ± 3.05 , with 74 patients (37.0%) classified as high risk (NEWS2 ≥ 7), 42 (21.0%) as medium risk, and 84 (42.0%) as low risk. The mean admission shock index was 0.92 ± 0.27 . Using the cut-off of 0.95 derived from receiver operating characteristic (ROC) analysis (see **Table 3**), 84 patients (42.0%) had a shock index ≥ 0.95 and 116 patients (58.0%) had a shock index < 0.95 . Within the first 24 hours of admission, 88 patients (44.0%) died, 106 patients (53.0%) required vasopressor support, and 45 patients (22.5%) were admitted to the ICU or HCU (**Table 1**).

Baseline characteristics including age, sex, diabetes mellitus, hypertension, and infection source did not differ significantly between the two shock index groups (all $p > 0.05$), indicating that the groups were comparable at presentation. In contrast, NEWS2 score was significantly higher in patients with shock index ≥ 0.95 compared with those with shock index < 0.95 (7.65 ± 1.89 vs. 3.37 ± 2.40 , $p < 0.001$), while qSOFA score did not differ significantly between groups (2.55 ± 0.50 vs. 2.57 ± 0.50 , $p = 0.765$), consistent with qSOFA ≥ 2 having been an inclusion criterion for all patients. Both mortality and vasopressor requirement were significantly more frequent among patients with shock index ≥ 0.95 (63.6% vs. 36.4%, $p < 0.001$; and 58.5% vs. 41.5%, $p < 0.001$, respectively), whereas ICU/HCU admission was more common in the SI < 0.95 group (68.9% vs. 31.1%, $p = 0.093$) (**Table 1**).

Table 1. Baseline Characteristics, Severity Markers, and Early Clinical Outcomes Stratified by Admission Shock Index Category (n=200)

Variable	SI <0.95 (n=116)	SI ≥0.95 (n=84)	Total (n=200)	p-value
Demographics				
Age, mean ± SD (years)	57.36 ± 16.17	56.23 ± 15.36	56.90 ± 15.80	0.617
Sex, n (%)				0.977
Female	61 (58.1%)	44 (41.9%)	105 (52.5%)	
Male	55 (57.9%)	40 (42.1%)	95 (47.5%)	
Comorbidities				
Diabetes Mellitus, n (%)				0.169
Yes	41 (65.1%)	22 (34.9%)	63 (31.5%)	
No	75 (54.7%)	62 (45.3%)	137 (68.5%)	
Hypertension, n (%)				0.138
Yes	55 (64.0%)	31 (36.0%)	86 (43.0%)	
No	61 (53.5%)	53 (46.5%)	114 (57.0%)	
Infection Source				
Infection Source, n (%)				0.197
Pulmonary	70 (61.9%)	43 (38.1%)	113 (56.5%)	
Non-pulmonary	46 (52.9%)	41 (47.1%)	87 (43.5%)	
Severity Markers				
qSOFA score, mean ± SDa	2.57 ± 0.50	2.55 ± 0.50	2.56 ± 0.50	0.765
NEWS2 score, mean ± SD	3.37 ± 2.40	7.65 ± 1.89	5.17 ± 3.05	<0.001
NEWS2 Risk Category, n (%)				<0.001
Low (<5)	81 (96.4%)	3 (3.6%)	84 (42.0%)	
Medium (5–6)	20 (47.6%)	22 (52.4%)	42 (21.0%)	
High (≥7)	15 (20.3%)	59 (79.7%)	74 (37.0%)	
Admission Shock Index				
Shock Index, mean ± SD	0.74 ± 0.13	1.18 ± 0.18	0.92 ± 0.27	<0.001
Shock Index Category, n (%)				
SI <0.95	116 (100%)	—	116 (58.0%)	
SI ≥0.95	—	84 (100%)	84 (42.0%)	
Early Clinical Outcomes (within 24 hours)				
Mortality, n (%)	32 (36.4%)	56 (63.6%)	88 (44.0%)	<0.001
Vasopressor Requirement, n (%)	44 (41.5%)	62 (58.5%)	106 (53.0%)	<0.001
ICU/HCU Admission, n (%)	31 (68.9%)	14 (31.1%)	45 (22.5%)	0.093

Bold p-values indicate statistical significance (p <0.05).

In bivariate analysis, each 0.1-unit increase in admission shock index was associated with increased odds of mortality (unadjusted OR=1.38, 95% CI 1.21–1.56, $p<0.001$) and vasopressor requirement (unadjusted OR=1.39, 95% CI 1.22–1.58, $p<0.001$), but not with ICU/HCU admission (unadjusted OR=0.93, 95% CI 0.82–1.06, $p=0.273$). After multivariable adjustment for age, sex, diabetes mellitus, hypertension, and infection source, these associations remained materially unchanged: higher admission shock index remained

independently associated with increased mortality (adjusted OR=1.41, 95% CI 1.24–1.61, $p=0.001$) and vasopressor requirement (adjusted OR=1.38, 95% CI 1.21–1.58, $p=0.001$) within the first 24 hours, while the association with ICU/HCU admission remained non-significant (adjusted OR=0.92, 95% CI 0.81–1.05, $p=0.222$) (**Table 2**). The close similarity between unadjusted and adjusted estimates suggests limited confounding by the covariates included in this analysis.

Table 2. Association Between Admission Shock Index (per 0.1-unit increase) and Early Clinical Outcomes (n=200)

Outcome (24 hours)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	p-value
Mortality	1.38 (1.21–1.56)	1.41 (1.24–1.61)	0.001
Vasopressor Requirement	1.39 (1.22–1.58)	1.38 (1.21–1.58)	0.001
ICU/HCU Admission	0.93 (0.82–1.06)	0.92 (0.81–1.05)	0.222

Adjusted OR: adjusted for age, sex, diabetes mellitus, hypertension, and infection source.

Table 3. Discriminatory Performance of Admission Shock Index for Early Clinical Outcomes (ROC Analysis)

Outcome	AUC	95% CI	Optimal Cut-off	Sensitivity	Specificity
Mortality	0.721	0.648–0.793	SI ≥ 0.95	63.6%	75.0%
Vasopressor Requirement	0.718	0.647–0.788	SI ≥ 0.95	58.5%	76.6%
ICU/HCU Admission	0.445	0.350–0.540	N/A	—	—

Optimal cut-off determined post-hoc using the Youden index (sensitivity + specificity – 1). Youden index: mortality 0.386, vasopressor requirement 0.351.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the discriminatory performance of admission shock index for each outcome. Shock index

demonstrated acceptable discriminatory ability for mortality (AUC=0.721, 95% CI 0.648–0.793, $p<0.001$) and vasopressor requirement (AUC=0.718, 95% CI 0.647–0.788, $p<0.001$),

but not for ICU/HCU admission (AUC=0.445, 95% CI 0.350–0.540, $p=0.262$). Using the Youden index, the optimal cut-off for both mortality and vasopressor requirement was a shock index of ≥ 0.95 , yielding a sensitivity of 63.6% and specificity of 75.0% for mortality, and a sensitivity of 58.5% and specificity of 76.6% for vasopressor requirement (**Table 3**).

DISCUSSION

In this retrospective cohort study of emergency department patients with sepsis, a higher admission shock index was independently associated with an increased risk of early mortality and vasopressor requirement within the first 24 hours of hospital presentation. The relatively high early mortality observed in this cohort may reflect the severity of illness among patients presenting to a secondary referral emergency department, where many patients arrive at advanced stages of sepsis and require urgent hemodynamic stabilization. However, the admission shock index was not significantly associated with ICU or HCU admission. These findings suggest that the shock index may serve as a simple and rapidly available bedside indicator for identifying sepsis patients at risk of early hemodynamic deterioration in the emergency department.

Early clinical deterioration is common among patients with sepsis, particularly during the initial hours after hospital presentation, when circulatory instability and organ dysfunction may rapidly progress if not promptly recognized. Early identification of high-risk patients in the emergency department is therefore critical to guide timely resuscitation, close monitoring, and

appropriate escalation of care. Several studies have demonstrated that delayed recognition and treatment of sepsis are associated with increased mortality, highlighting the importance of rapid risk stratification during the early phase of care.^{13,14} In busy emergency department settings, however, complex scoring systems that require laboratory testing may not be immediately available during triage, which underscores the need for simple bedside predictors that can be rapidly applied using routinely collected vital signs.^{5,15}

The shock index represents a simple hemodynamic parameter that reflects the balance between cardiac compensation and circulatory perfusion. Physiologically, an elevated shock index occurs when heart rate increases in response to reduced effective circulating volume or impaired vascular tone while systolic blood pressure decreases, indicating early circulatory compromise.^{6,7} In patients with sepsis, systemic inflammatory responses lead to vasodilation, capillary leakage, and relative hypovolemia, which may result in tachycardia and hypotension even before overt shock becomes clinically apparent.^{14,16} Consequently, the shock index may detect early hemodynamic instability earlier than isolated vital signs such as heart rate or blood pressure alone, a rationale supported by systematic review evidence demonstrating that the shock index, including its modified variants, tracks clinical deterioration across multiple sepsis cohorts.⁹

The findings of this study are consistent with recent literature demonstrating the prognostic value of shock index in patients with sepsis and septic shock. In a cohort of patients

with septic shock managed by mobile intensive care units, worsening shock index trajectory during prehospital care was independently associated with higher 28-day mortality.⁶ A subsequent study by the same group further demonstrated that the shock index, along with its modified and age-adjusted variants, was significantly associated with 28-day mortality among patients with prehospital septic shock.⁷ A systematic review similarly concluded that the shock index reliably distinguished survivors from non-survivors across multiple sepsis cohorts.⁹ Elevated shock index has also been linked to a greater requirement for vasopressor therapy, reflecting early circulatory compromise in critically ill patients.^{8,17} These findings support the role of shock index as a practical bedside indicator for identifying patients at higher risk of clinical deterioration, consistent with the present study's finding that elevated admission shock index was independently associated with both early mortality and vasopressor requirement.

Compared with composite organ dysfunction scores such as SOFA, the shock index offers the practical advantage of being calculable immediately at triage using only heart rate and blood pressure. This simplicity, however, comes with a trade-off in discriminatory precision. A recent comparative study evaluating the shock index alongside multiple early warning scores for sepsis prediction in the emergency department found that the shock index demonstrated only modest discriminatory performance relative to composite scores such as NEWS2.¹⁰ The AUC values observed in the present study (0.721 for

mortality and 0.718 for vasopressor requirement) likewise indicate acceptable but not excellent discrimination, consistent with the expectation that a single-ratio bedside tool will not match the precision of multi-parameter composite scores. In settings such as ours, where SOFA scoring was not consistently documented, the shock index may nonetheless serve a complementary role: a rapid first-pass screening tool rather than a replacement for more comprehensive severity assessment.

The 24-hour mortality rate observed in this cohort (44.0%) was higher than commonly reported in general emergency department sepsis populations, and this finding warrants careful interpretation. First, RSUD Cengkareng functions as a type B secondary referral hospital, which routinely receives patients transferred from primary care facilities or smaller hospitals after initial stabilization attempts have failed, resulting in a case mix skewed toward more advanced presentations at ED arrival. This is reflected in the severity profile of this cohort: the majority of patients had a qSOFA score of 3 (56.0%), and 37.0% fell into the high-risk NEWS2 category (≥ 7). Second, all patients meeting inclusion criteria during the study period were enrolled through total sampling rather than a randomly selected subset, intended to minimize selection bias by capturing the full spectrum of disease severity presenting to the ED. Comparable mortality figures have been reported in other Indonesian sepsis cohorts: a prospective cohort study at a tertiary hospital in Yogyakarta reported 28-day mortality rates of 65.0% to 92.6% depending on vitamin D status among 88 septic patients with a similar age

profile to the present cohort,¹⁸ while a retrospective cohort at a national referral hospital in Jakarta using the same qSOFA ≥ 2 screening criterion as the present study reported outcomes consistent with a high-acuity inpatient population.¹² These findings suggest that high early mortality in hospital-based sepsis cohorts in Indonesia, particularly at referral centers, may reflect genuine differences in case mix and disease severity rather than methodological artifact, though the absence of serum lactate and complete SOFA scoring limits direct severity comparison with other published cohorts.

The findings of this study have several practical implications for emergency department practice. Because the shock index can be calculated immediately using routinely measured vital signs, it represents a simple and readily available tool for early risk stratification in patients with suspected sepsis. Incorporating the shock index into the initial triage assessment, using the threshold identified in this study (SI ≥ 0.95), may help clinicians identify patients at higher risk of early hemodynamic deterioration, allowing for closer monitoring and timely escalation of care. Particularly in resource-limited emergency settings where rapid laboratory testing may not always be available, the use of simple bedside indicators such as the shock index may provide additional support for early clinical decision-making. Nevertheless, the shock index should not be used as a standalone predictor, but rather as a complementary tool alongside comprehensive clinical assessment and established sepsis management protocols.

For patients identified as high-risk by an elevated shock index, timely initiation of

guideline-directed sepsis management remains the cornerstone of reducing early mortality, including prompt fluid resuscitation, early antimicrobial therapy, and source control where indicated.¹⁶ Regarding vasopressor selection, current international guidelines and evidence consistently recommend norepinephrine as the first-line vasopressor in septic shock, a recommendation adhered to in the large majority of clinical practice surveyed internationally.^{16,19} For patients with profound or refractory hypotension despite norepinephrine, earlier initiation of norepinephrine administered concurrently with fluid resuscitation, rather than only after volume deficits are corrected, may stabilize arterial pressure more rapidly than fluids alone.²⁰ More recently, evidence from a large multicenter cohort suggested that earlier addition of vasopressin to norepinephrine, often at lower norepinephrine doses than typical current practice, was associated with reduced in-hospital mortality compared with usual clinician-directed timing.²¹ While these specific protocols have not been validated in the population studied here, they suggest that patients identified as high-risk by an elevated shock index at triage may benefit from an anticipatory approach to vasopressor escalation, an area warranting further prospective investigation.

In contrast, admission shock index was not significantly associated with ICU or HCU admission in the present study. This contrasts with previous literature showing higher shock index values were associated with increased ICU admission.²² This difference in finding may reflect the multifactorial nature of ICU

admission decisions, which are influenced not only by physiological severity but also by multiple factors including illness acuity, expected benefit, resource availability, clinician judgment, and patient or family preferences.^{23,24} Therefore, ICU admission may not always directly correspond to the degree of hemodynamic instability measured at initial emergency department presentation.

This study has several limitations that should be acknowledged. First, the retrospective design may introduce selection bias and limits the ability to establish causal relationships between admission shock index and clinical outcomes; this risk was partially mitigated through total sampling of all eligible patients during the study period, though residual bias from unmeasured factors cannot be excluded. Second, this study was conducted in a single secondary referral hospital, which may limit the generalizability of the findings to other healthcare settings with different patient populations or resource availability. Third, clinical variables were obtained from medical records, and measurement variability in triage vital signs may have influenced the calculated shock index. In addition, this study did not include other physiological markers of sepsis severity such as serum lactate levels or full SOFA scores, which may provide additional prognostic information beyond the qSOFA and NEWS2 scores captured here; the absence of these markers also limited the ability to directly compare the severity profile of this cohort with other published sepsis populations. The covariates available for multivariable adjustment were similarly limited by the available events per

variable given the sample size and outcome frequencies in this study, which may have left residual confounding unaddressed such as lactate, MAP, Glasgow Coma Scale (GCS), initial fluid use, SOFA score, and other comorbidities. Finally, this study used qSOFA ≥ 2 as an operational screening criterion for sepsis rather than the formal Sepsis-3 diagnostic definition requiring a SOFA score ≥ 2 in the context of infection. While this approach is consistent with prior ED-based sepsis studies that have used qSOFA as an operational screening tool,¹² qSOFA's known low sensitivity for sepsis detection (approximately 28% in pooled literature)¹¹ means that patients with true sepsis but qSOFA < 2 may have been systematically excluded from this cohort, potentially limiting generalizability to milder presentations and contributing to the elevated mortality rate observed. The optimal shock index cut-off identified in this study (≥ 0.95) was derived empirically from this dataset using the Youden index and has not been externally validated. Therefore, future prospective multicenter studies incorporating broader clinical and laboratory parameters, including lactate and full SOFA scoring, are warranted to further validate the role of shock index in early sepsis risk stratification.

CONCLUSION

In conclusion, admission shock index was significantly associated with early mortality and vasopressor requirement among emergency department patients with sepsis. These findings suggest that shock index may serve as a simple bedside indicator for identifying patients at risk

of early hemodynamic deterioration during initial emergency department evaluation. Clinicians encountering sepsis patients with an admission shock index of ≥ 0.95 should consider initiating closer hemodynamic monitoring, preparing for early vasopressor support, and expediting involvement of senior clinical staff, particularly in resource-limited settings where comprehensive severity scoring may not be immediately available. However, shock index was not significantly associated with ICU or HCU admission, highlighting that decisions regarding critical care admission are influenced by multiple clinical and institutional factors beyond hemodynamic parameters alone. Further prospective multicenter studies incorporating broader clinical and laboratory parameters are needed to better define the role of shock index in early sepsis risk stratification.

CONFLICT OF INTEREST

The authors declare that there was no conflict of interest regarding the publication of this paper.

ACKNOWLEDGMENTS

The authors would like to express their gratitude to all individuals involved with this research. No specific acknowledgments are applicable.

REFERENCES

1. Rudd KE, Johnson SC, Agesa KM, et al. Global, regional, and national sepsis incidence and mortality, 1990–2017: analysis for the Global Burden of Disease Study. *The Lancet*. 2020;395(10219):200-211. doi:10.1016/S0140-6736(19)32989-7
2. Hincapié-Osorno C, van der Aart TJ, van Wijk RJ, et al. Early risk assessment of severe outcomes in patients with suspected infection in the emergency department using clinical scores in a multicenter international validation. *J Infect Public Health*. 2026;19(4):103170. doi:10.1016/j.jiph.2026.103170
3. 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. doi:10.1001/jama.2016.0287
4. Seymour CW, Liu VX, Iwashyna TJ, et al. Assessment of Clinical Criteria for Sepsis: For the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*. 2016;315(8):762-774. doi:10.1001/jama.2016.0288
5. Babu MCC, Thomas VK, Chakrapani AT. Diagnostic Accuracy of Triage Shock Index in Predicting Hyperlactatemia in Patients With Sepsis in the Emergency Department. *Cureus*. 2025;17. doi:10.7759/cureus.90971
6. Jouffroy R, Gilbert B, Thomas L, et al. Association between prehospital shock index variation and 28-day mortality among patients with septic shock. *BMC Emerg Med*. 2022;22(1):87. doi:10.1186/s12873-022-00645-1
7. Jouffroy R, Gille S, Gilbert B, et al. Relationship between shock index, modified shock index, and age shock index and 28-day mortality among patients with prehospital septic shock. *J Emerg Med*. 2024;66(2):144-153. doi:10.1016/j.jemermed.2023.11.010
8. Usman S, Bhojwani KM, Raheem A, Khan MA, Khan NU. Shock Indices as Predictors of Outcomes in Emergency Department Patients With Emergency Severity Index Level 3. *Cureus*. 2025;17(5):e83379. doi:10.7759/cureus.83379
9. Diaztagle Fernández JJ, Castañeda-González JP, Trujillo Zambrano JI, Duarte Martínez FE, Saavedra Ortiz MÁ. Assessment of the shock index in septic shock: A systematic review. *Med Intensiva (Engl Ed)*. 2024;48(11):e10-e19. doi:10.1016/j.medine.2024.07.006
10. Lam RPK, Dai Z, Lau EHY, et al. Comparing 11 early warning scores and

- three shock indices in early sepsis prediction in the emergency department. *World J Emerg Med.* 2024;15(4):273-282. doi:10.5847/wjem.j.1920-8642.2024.052
11. Kumar A, Abbenbroek B, Delaney A, Hammond N, Grattan S, Finfer S. Sepsis triggers and tools to support early identification in healthcare settings: An integrative review. *Australian Critical Care.* 2023;36(6):1117-1128. doi:10.1016/j.aucc.2023.01.001
 12. Rinaldi I, Sudaryo MK, Prihartono NA. Disseminated Intravascular Coagulation in Sepsis and Associated Factors. *Journal of Clinical Medicine.* 2022;11(21):6480. doi:10.3390/jcm11216480
 13. Seymour CW, Gesten F, Prescott HC, et al. Time to Treatment and Mortality during Mandated Emergency Care for Sepsis. *N Engl J Med.* 2017;376(23):2235-2244. doi:10.1056/NEJMoa1703058
 14. Cecconi M, Evans L, Levy M, Rhodes A. Sepsis and septic shock. *Lancet.* 2018;392(10141):75-87. doi:10.1016/S0140-6736(18)30696-2
 15. Loots FJ, van Vught LA, van den Brande M, et al. Implementing a sepsis prediction score in out-of-hours primary care: Feasibility and acceptability study. *Eur J Gen Pract.* 2025;31(1):2574869. doi:10.1080/13814788.2025.2574869
 16. Evans L, Rhodes A, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. *Intensive Care Med.* 2021;47(11):1181-1247. doi:10.1007/s00134-021-06506-y
 17. N PV, Sneha. Shock index as a predictor of vasopressor use in patients with sepsis. *International Journal of Advances in Medicine.* 2019;6(5):1488-1492. doi:10.18203/2349-3933.ijam20193624
 18. Asdie RH, Mulya DP, Nainggolan M. Assessment of 28-day survival of patients with sepsis based on vitamin D status: a hospital-based prospective cohort study in Indonesia. *Pan Afr Med J.* 2023;45:76. doi:10.11604/pamj.2023.45.76.36336
 19. Bitton E, Zimmerman S, Azevedo LCP, et al. An international survey of adherence to Surviving Sepsis Campaign Guidelines 2016 regarding fluid resuscitation and vasopressors in the initial management of septic shock. *J Crit Care.* 2022;68:144-154. doi:10.1016/j.jcrc.2021.11.016
 20. Monnet X, Lai C, Ospina-Tascon G, De Backer D. Evidence for a personalized early start of norepinephrine in septic shock. *Crit Care.* 2023;27(1):322. doi:10.1186/s13054-023-04593-5
 21. Kalimoultou A, Kennedy JN, Feng J, et al. Optimal Vasopressin Initiation in Septic Shock: The OVISS Reinforcement Learning Study. *JAMA.* 2025;333(19):1688-1698. doi:10.1001/jama.2025.3046
 22. Surendhar S, Jagadeesan S, Jagtap AB. Complementary value of the Shock Index v. the Modified Shock Index in the prediction of in-hospital intensive care unit admission and mortality: A single-centre experience. *Afr J Thorac Crit Care Med.* 2023;29(2). doi:10.7196/AJTCCM.2023.v29i2.286
 23. James FR, Power N, Laha S. Decision-making in intensive care medicine – A review. *J Intensive Care Soc.* 2018;19(3):247-258. doi:10.1177/1751143717746566
 24. Gopalan PD, Pershad S. Corrigendum to 'Decision-making in ICU – A systematic review of factors considered important by ICU clinician decision makers with regard to ICU triage decisions'. *Journal of Critical Care* 50(2019) pp99–110. *Journal of Critical Care.* 2020;56:326. doi:10.1016/j.jcrc.2019.07.010