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Study of Shock Index for Early Detection of Septic Shock and Prediction of Mortality in Emergency Units

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ABSTRACT

Background: Early identification of septic shock is crucial to reduce mortality, as sepsis and septic shock remain major causes of morbidity and mortality worldwide. Shock Index (SI), calculated as heart rate divided by systolic blood pressure, is an immediate bedside marker of circulatory compromise. To determine the predictive value of SI for 28-day mortality; to assess its prognostic value in severe sepsis/septic shock; and to evaluate its correlation with serum lactate and total leukocyte count (TLC).

Methods: This prospective observational study included 50 patients presenting to the emergency unit with severe sepsis or septic shock. SI was calculated at presentation. Mortality, ICU admission/stay, inotropic support, ventilator support, lactate, and TLC were analyzed. Chi-square test, Pearson correlation, and ROC curve analysis were performed.

Results: The mean age was 50.8 ± 15.5 years. Overall mortality was 38.0% (19/50). Mortality increased significantly across SI categories ($\chi^2 = 42.78$, df = 3, $p < 0.001$). SI correlated strongly with mortality (r = 0.88), ventilator support (r = 0.80), inotropic support (r = 0.77), lactate (r = 0.91), and TLC (r = 0.85). ROC analysis showed excellent mortality prediction by SI (AUC = 0.988).

Conclusion: Shock Index is a rapid, non-invasive, and cost-effective predictor of mortality and severity in sepsis. Its strong correlation with lactate and TLC supports its role as a bedside surrogate for hypo perfusion and inflammatory burden. Furthermore, it may serve as an effective screening tool for early recognition of clinical deterioration in septic patients.

Key-words: Shock Index; sepsis; Septic shock; Mortality; Lactate; Emergency department; ROC curve

INTRODUCTION

Sepsis and septic shock remain major causes of morbidity and mortality worldwide. Early recognition and prompt resuscitation are central to improving outcomes [1-4]. Serum lactate is widely used as a marker of tissue hypoperfusion and is strongly associated with mortality in infected patients [1]. However, lactate and other laboratory parameters may be delayed in emergency settings, particularly in resource-limited hospitals.

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Shock Index (SI), defined as heart rate divided by systolic blood pressure, is a simple bedside parameter that integrates tachycardia and hypotension into a single hemodynamic marker [2,3]. Previous work has shown that SI may identify acutely ill patients earlier than conventional vital signs alone [4]. In severe sepsis and septic shock, Mohd Yussof et al. reported that SI after resuscitation predicted death with AUC 0.889, sensitivity 80.8%, and specificity 79.2% [5]. Berger *et al.* found that SI was useful for early recognition of sepsis, hyperlactatemia, and 28-day mortality in the emergency department [6].

Sepsis is currently recognized as a life-threatening organ dysfunction caused by a dysregulated host response to infection and remains a major public health challenge worldwide. Despite advances in antimicrobial therapy, intensive care management, and sepsis treatment

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protocols, mortality rates remain high, particularly among patients who progress to septic shock [7]. According to the Sepsis-3 consensus definition, septic shock is characterized by profound circulatory, cellular, and metabolic abnormalities that substantially increase the risk of death. Delayed recognition of hemodynamic instability may lead to inadequate tissue perfusion, multiple organ dysfunction syndrome, prolonged intensive care unit (ICU) stay, and increased healthcare costs [8].

Early risk stratification in the emergency department is therefore essential for timely initiation of fluid resuscitation, vasopressor therapy, and appropriate monitoring. Although several scoring systems, such as the Sequential Organ Failure Assessment (SOFA), quick SOFA (qSOFA), and Acute Physiology and Chronic Health Evaluation (APACHE), have been developed to predict outcomes in septic patients, these tools often require multiple clinical variables, laboratory investigations, or repeated assessments [9,10]. Their routine use may be challenging in busy emergency settings and resource-constrained healthcare facilities.

In contrast, the SI is a readily available physiological parameter that can be calculated within seconds from routine vital signs without the need for laboratory testing. Because SI reflects the combined effects of tachycardia and hypotension, it may detect early circulatory compromise before overt shock becomes clinically apparent [11]. Recent studies have demonstrated the usefulness of SI in predicting mortality, identifying patients at risk of deterioration, and guiding triage decisions in critically ill patients. However, evidence regarding its relationship with biochemical markers such as serum lactate and total leukocyte count in septic patients remains limited, particularly in the Indian emergency care setting.

The present study evaluates SI for early detection of septic shock and prediction of mortality in emergency units, with additional assessment of its relationship with ICU stay, ventilator support, inotropic support, lactate, and total leukocyte count.

MATERIALS AND METHODS

Study design and setting- Prospective observational study conducted in the emergency unit of a tertiary care hospital. 50 adult patients diagnosed with severe sepsis or septic shock was included. Patients presenting to the emergency department with clinical features suggestive of severe sepsis or septic shock were screened consecutively for eligibility. After obtaining informed consent from the patient or a legally authorized representative, demographic details, medical history, and clinical examination findings were recorded. At presentation, vital parameters including heart rate, systolic blood pressure, respiratory rate, temperature, and mean arterial pressure were measured. SI was calculated by dividing the heart rate by the systolic blood pressure. Baseline laboratory investigations, including serum lactate levels and TLC, were obtained as part of routine clinical management. Patients received standard treatment according to institutional sepsis management protocols and were monitored throughout their hospital stay. Information regarding ICU admission, duration of ICU stay, requirement for inotropic support, need for mechanical ventilation, and other relevant clinical outcomes was documented prospectively. All enrolled patients were followed for 28 days or until death/discharge, and mortality outcomes were recorded for analysis. Age, sex, temperature, respiratory rate, heart rate, systolic blood pressure, mean arterial pressure, serum lactate, TLC, ICU admission/stay, inotropic support, ventilator support, and 28-day outcome were recorded. SI was calculated as heart rate/systolic blood pressure.

Inclusion Criteria

1. Adult patients aged 18 years and above**.**
2. Patients presenting to the emergency department with a clinical diagnosis of severe sepsis or septic shock based on Sepsis-3 criteria.
3. Patients admitted to the emergency unit within the study period.
4. Patients in whom baseline vital signs, including heart rate and systolic blood pressure, could be accurately recorded at presentation.

Exclusion Criteria

1. Patients aged <18 years and pregnant women.
2. Patients with significant cardiac arrhythmias. affecting heart rate measurement.
3. Patients receiving medications that significantly alter heart rate or blood pressure, such as beta-blockers or drugs causing marked atrioventricular blockade.
4. Patients with implanted cardiac pacemakers.

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Statistical Analysis- Continuous variables were summarized as mean ± SD. Categorical variables were summarized as frequencies and percentages. Pearson correlation assessed associations between SI and outcomes/biochemical markers. The chi-square test assessed the association between SI categories and mortality. ROC curve analysis evaluated discrimination for mortality and organ-support outcomes. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1 summarizes the demographic and baseline characteristics of the study population. A total of 50 patients were included, with a mean age of 50.8 ± 15.5 years and a median age of 52 years (range: 19–82 years). The cohort comprised 23 males (46%) and 27 females (54%). Overall, 19 patients (38%) died during hospitalization, while 31 patients (62%) survived.

Table 1: Demographic characteristics

Characteristic	Value
Total patients	50
Mean age ± SD	50.8 ± 15.5 years
Median age (range)	52 (19-82) years
Male	23 (46%)
Female	27 (54%)
Deaths	19 (38%)
Survivors	31 (62%)

Table 2 demonstrates the relationship between SI categories and in-hospital mortality. Patients with an SI <0.9 (n = 11) had no deaths (0%), whereas those with an SI of 0.9–1.1 (n = 20) had a mortality rate of 5.0% (1/20). Mortality increased markedly in patients with an SI of 1.1–1.3 (3/4, 75.0%) and reached 100% (15/15) among patients with an SI > 1.3. Chi-square analysis demonstrated a highly significant association between increasing Shock Index category and in-hospital mortality (χ^2 = 42.78, df = 3, p < 0.001), indicating that higher Shock Index values were strongly associated with an increased risk of death.

Table 2: Shock Index categories and mortality

Shock Index range	Total	Deaths	Survivors	Mortality (%)
<0.9	11	0	11	0%
0.9-1.1	20	1	19	5%
1.1-1.3	4	3	1	75%
>1.3	15	15	0	100%

Fig. 1 illustrates the progressive increase in in-hospital mortality with increasing SI category. Patients with an SI <0.9 had 0% mortality, while those with an SI of 0.9–1.1 had a mortality rate of 5.0%. Mortality increased sharply to 75.0% in patients with an SI of 1.1–1.3, and all patients with an SI >1.3 died during hospitalization (100% mortality). The graph demonstrates a clear dose-response relationship between increasing Shock Index and mortality, indicating that higher SI values were associated with worse clinical outcomes. This trend was supported by a highly significant chi-square analysis (χ^2 = 42.78, df = 3, p < 0.001), confirming Shock Index as a strong predictor of in-hospital mortality.

Table 3 shows significant positive correlations between SI and clinical outcomes. SI demonstrated very strong correlations with in-hospital mortality (r = 0.879), serum lactate (r = 0.911), and total leukocyte count (r = 0.847) (all p < 0.001). Higher SI was also significantly associated with ventilator support (r = 0.797), inotropic support (r = 0.765), and ICU admission (r = 0.613) (all p < 0.001). Although the correlation with ICU stay duration was comparatively weaker (r= 0.305), it remained statistically significant (p = 0.031). Overall, higher SI was associated with greater disease severity, organ dysfunction, and adverse clinical outcomes.

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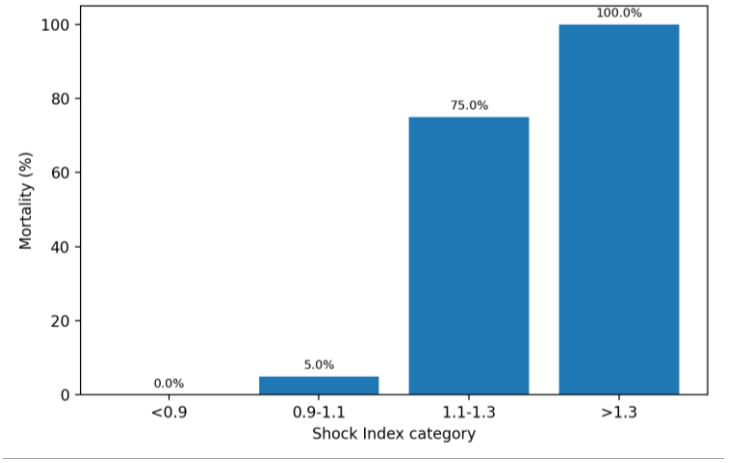

Fig 1: Impact of Shock Index on Mortality

Table 3: Correlation of Shock Index with clinical outcomes and biochemical markers

Variable correlated with SI	Pearson r	p-value
Mortality	0.879	<0.001
ICU admission	0.613	<0.001
Inotropic support	0.765	<0.001
Ventilator support	0.797	<0.001
ICU stay duration	0.305	0.031
Lactate	0.911	<0.001
Total leukocyte count	0.847	<0.001

Fig. 2 compares the receiver operating characteristic (ROC) curves of Shock Index, serum lactate, and total leukocyte count for predicting in-hospital mortality. All three ROC curves lie close to the upper-left corner of the graph, indicating excellent diagnostic performance. Serum lactate demonstrated the highest predictive accuracy (AUC = 1.000), followed closely by Shock Index (AUC = 0.988) and TLC (AUC = 0.977). Each marker performed substantially better than the reference diagonal line, which represents random prediction. Although lactate achieved the greatest overall accuracy, the minimal difference between lactate and Shock Index suggests that SI is an excellent bedside predictor of mortality despite requiring no laboratory investigations.

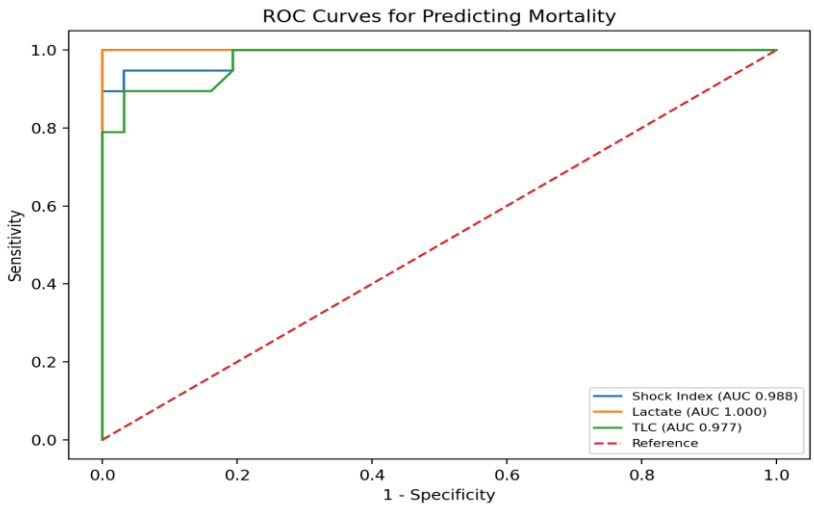

Fig. 2: ROC curve analysis

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Fig. 3 represents the ROC curves evaluating the ability of Shock Index to predict major clinical outcomes. Shock Index demonstrated excellent discriminatory ability for predicting in-hospital mortality (AUC = 0.988), ICU admission (AUC = 0.984), ventilator support (AUC = 0.983), and inotropic support (AUC = 0.982). All ROC curves remain close to the upper-left corner, indicating consistently high sensitivity and specificity across different clinical outcomes. The minimal variation among the AUC values highlights the reliability of Shock Index as a single bedside parameter for predicting disease severity and the likelihood of requiring advanced critical care interventions. These findings support the routine clinical use of Shock Index for early risk stratification and timely decision-making in patients with sepsis. ROC curves of Shock Index for mortality, ICU admission, inotropic support, and ventilator support.

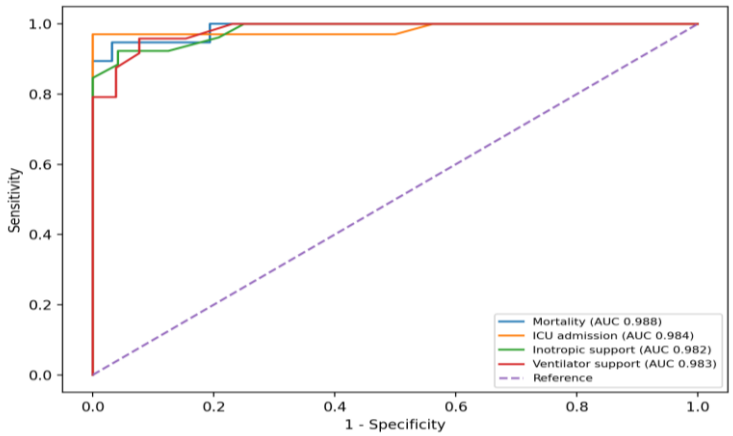

Fig. 3: ROC Curves of Shock Index for Clinical Outcomes

DISCUSSION

This study demonstrates that Shock Index is strongly associated with mortality and organ-support requirements in patients with severe sepsis/septic shock. Mortality increased significantly across SI categories, and chi-square analysis confirmed a highly significant association between SI category and death. The ROC curve showed excellent discrimination for mortality, supporting SI as an early bedside risk-stratification tool [13,14].

The strong association between SI and lactate suggests that SI reflects tissue hypoperfusion. This is clinically important because lactate may not be immediately available in all emergency units. The strong correlation between SI and TLC further suggests that higher SI tracks with systemic inflammatory burden. These findings are consistent with prior studies showing the prognostic value of SI in sepsis and acute circulatory failure [7-10].

Compared with laboratory markers, SI is immediate, non-invasive, inexpensive, repeatable, and easily calculated from routine vital signs [15]. Therefore, it may be especially useful in resource-limited emergency units for early detection of septic shock, triage decisions, and communication of risk.

This study has several limitations that should be considered while interpreting the findings. The sample size was relatively small, comprising only 50 patients, which may limit the generalizability of the results [12-14]. Additionally, the study was conducted at a single center using an observational design, potentially introducing selection bias and limiting external validity. The findings may also have been influenced by confounding factors such as variations in patient comorbidities and treatment approaches. Furthermore, multivariable regression analysis was not performed to adjust for potential confounders, and the results were not externally validated in an independent patient population [15-18].

Despite these limitations, the study provides a foundation for future research. Larger prospective multicenter studies are needed to validate the prognostic utility of the SI in sepsis. Future investigations should also assess the value of serial SI monitoring in conjunction with established biomarkers such as lactate clearance to improve risk stratification and outcome prediction. Additionally, the integration of SI into emergency department sepsis triage protocols warrants further evaluation to determine its effectiveness in

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facilitating early identification and management of high-risk patients.

CONCLUSIONS

Shock Index is a simple, rapid, and reliable predictor of mortality and severity in severe sepsis and septic shock. It is significantly associated with mortality, ICU admission, ventilator support, and inotropic support. Its strong correlation with lactate and total leukocyte count supports its use as a bedside surrogate marker for hypoperfusion and systemic inflammation in emergency units. Furthermore, the findings of this study suggest that Shock Index may serve as an effective screening tool for early recognition of clinical deterioration in septic patients before the onset of overt hemodynamic collapse. Regular monitoring of Shock Index during emergency department stay may help clinicians identify patients who require closer observation, aggressive resuscitation, or early transfer to intensive care. Consequently, Shock Index has the potential to enhance patient management strategies and optimize resource utilization in emergency care settings.

CONTRIBUTION OF AUTHORS

Research concept: Akhilesh Kumar A, Anand BS, Nagendra Prasad
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Literature search: Akhilesh Kumar A, Anand BS
Writing article: Akhilesh Kumar A, Anand BS
Critical review: Akhilesh Kumar A, Nagendra Prasad
Article editing: Akhilesh Kumar A, Anand BS, Nagendra Prasad
Final approval: Akhilesh Kumar A, Anand BS, Nagendra Prasad

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