Section: Emergency Medicine

Original Research Article

SYSTOLIC AND DIASTOLIC SHOCK INDEX AS A PREDICTOR FOR SEPTIC SHOCK IN PATIENTS PRESENTING TO THE EMERGENCY DEPARTMENT IN SEPSIS - PROSPECTIVE OBSERVATIONAL STUDY

Sruthi N Anandhan[1], K. Dhilipan[2], Ramesh K.[3]

[1]Post Graduate Resident, Department of Emergency Medicine, Kovai Medical Center and Hospital, Coimbatore, Tamil Nadu, India
[2]Associate Professor, Department of Emergency Medicine, KMCH institute of Health Sciences and Research, Coimbatore, Tamil Nadu, India
[3]Senior Resident, Department of Emergency Medicine, KMCH institute of Health Sciences and Research, Coimbatore, Tamil Nadu, India

Received *: 12/01/2026*
Received in revised form *: 04/03/2026*
Accepted *: 20/03/2026*

Corresponding Author:
Dr. Sruthi N. Anandhan,
Post Graduate Resident, Department of Emergency Medicine, Kovai Medical Center and Hospital, Coimbatore, Tamil Nadu, India.
Email: sruthinanandhan@gmail.com

DOI: 10.70034/ijmedph.2026.1.578

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 3374-3380

ABSTRACT

Background: Shock indices may aid in the early identification of patients with sepsis who are at risk of progression to septic shock. This study evaluated the predictive accuracy of the systolic shock index (SSI) and diastolic shock index (DSI) at triage.

Materials and Methods: A hospital-based prospective observational study was conducted among 100 adults with sepsis who presented to the Emergency Department of Kovai Medical Centre and Hospital from April 2025 to March 2026. Demographic details, clinical features, triage vital signs, and laboratory parameters were recorded, and the SSI and DSI were calculated. Patients were followed up for progression to septic shock, vasopressor requirement, ICU admission, and in-hospital mortality.

Results: Of the 100 patients, 21% progressed to septic shock, 23% required vasopressors, 30% required ICU admission, and the in-hospital mortality rate was 8%. Patients who progressed to septic shock had significantly higher triage DSI ($p = 0.015$) and lactate levels ($p = 0.001$). All patients who developed septic shock required vasopressors and ICU admission and had a higher mortality rate ($p = 0.003$). Using ROC-derived cut-offs, SSI $\geq$0.82 was present in 85.7% of patients with shock versus 31.6% of patients without shock, while DSI $\geq$1.3 was present in 81.0% versus 50.6% of patients, respectively. SSI showed better discrimination than DSI (AUC 0.812 vs. 0.726), with a sensitivity of 85.7%, specificity of 68.4%, NPV of 94.7%, and accuracy of 72.0%.

Conclusion: SSI and DSI at triage supported early risk stratification for septic shock progression, with SSI demonstrating superior overall predictive performance.

Keywords: Sepsis, Septic Shock, Shock Index, Blood Pressure, Heart Rate, Vasoconstrictor Agents.

INTRODUCTION

Sepsis remains a major cause of morbidity and mortality in hospitals worldwide. In emergency units, sepsis can progress rapidly to septic shock, leaving limited time for care.[1] With mortality rates of up to 54%, depending on illness and treatment, clinicians aim for early risk identification. Standard diagnostics rely on laboratory values and scoring systems that are unavailable at first contact. Early assessment of haemodynamic parameters helps identify high-risk patients.[2] The Shock Index (SI), calculated as heart rate divided by systolic pressure, is a quick tool for unstable circulation. Studies have assessed SI in trauma, myocardial infarction, and haemorrhage.[3] In sepsis, an increased heart rate with falling systolic pressure indicates worse outcomes and greater vasopressor need. A study found SI above 1.2 at arrival predicted vasopressor need within 24 hours.[4] The SI does not include diastolic pressure, which may better reflect vascular tone changes in distributive states such as sepsis. Loss of vascular tone may lead

to early diastolic pressure decline before systolic hypotension.[5] Due to this limitation, the Diastolic Shock Index (DSI) and Systolic Shock Index (SSI) were introduced. DSI, defined as heart rate divided by diastolic pressure, DSI is associated with mortality in septic shock.[6,7] Since diastolic pressure better reflects arterial compliance and systemic vascular resistance, it may better indicate loss of vascular tone in distributive shock.[5]

A prospective study found that both had prognostic value, with DSI showing a stronger association with adverse outcomes while remaining calculable from vital signs.[8] Their findings indicated that DSI increase is a marker for early vasopressor need. An Indian ICU study showed that repeated DSI measurements provide more prognostic information than baseline values.[7] Diastolic pressure decline in sepsis often results from the loss of arterial tone, reducing coronary flow during diastole and increasing the cardiac burden. Echocardiographic studies have confirmed that systolic and diastolic impairment are common in patients with sepsis. LV diastolic dysfunction is common in sepsis and is linked with mortality.[9,10] A paediatric study showed that the minimum diastolic-to-systolic blood pressure ratio in the first 24 hours predicted progression to septic shock within 48 hours and 28-day mortality.[11] Systolic and diastolic impairment could reverse with treatment, resembling human septic shock patterns, supporting these bedside indices.[12]

Shock indices remain useful in many resource-limited settings such as rural hospitals. In Indian EDs, delays in lactate/procalcitonin reporting may occur, whereas SI and its related indices require only simple vital signs. An SI of ≥ 0.875 tended to identify patients who later developed haemodynamic failure, with reasonable test performance.[13] In another cohort, an SI cut-off of 0.7 came close to the SIRS criteria for spotting raised lactate and for gauging 28-day mortality in patients assessed for suspected infection.[14] Prospective ED evidence in India is limited, and SSI/DSI cut-offs remain, and fixed cut-off values for SSI or DSI before overt hypotension remain unsettled. Early sepsis, before shock onset, still lacks fully validated bedside thresholds for these indices.

Aim

This study aimed to evaluate the predictive accuracy of SSI and DSI in identifying patients with sepsis at risk of progressing to septic shock.

MATERIALS AND METHODS

A hospital-based prospective observational study was conducted among 100 patients at the Department of Emergency Medicine, Kovai Medical Centre and Hospital (KMCH), from April 2025 to March 2026 (12 months). The study was performed after obtaining clearance from the Institutional Ethics Committee. Informed consent was obtained from all patients before enrolment in the study.

Inclusion Criteria

Adults (≥18 years) presenting to the ED with suspected or confirmed sepsis, with complete triage vital signs (HR, SBP, DBP), and SBP ≥100 mmHg at presentation.

Exclusion Criteria

Patients referred on vasopressors, SBP <100 mmHg at presentation, haemodynamic instability due to non-sepsis causes (acute myocardial infarction, haemorrhagic shock, cardiac arrest), end-stage organ failure (advanced cirrhosis or terminal malignancy), and pregnant women were excluded.

Methods

Demographic details, presenting complaints, comorbidities, and provisional diagnoses were recorded using a structured form. Vital signs, including heart rate, systolic and diastolic blood pressure, respiratory rate, temperature, oxygen saturation, and mean arterial pressure, were measured using standard monitoring devices. SSI (HR/SBP) and DSI (HR/DBP) were calculated during triage. Baseline laboratory parameters, including serum lactate, white blood cell count, serum creatinine, and procalcitonin levels, were obtained as part of routine evaluation.

Patients were monitored for progression to septic shock, defined as vasopressor requirement to maintain MAP ≥65 mmHg after fluid administration. For those who developed shock, the time of onset, time of vasopressor initiation, and corresponding vital signs were recorded, and the SSI and DSI were recalculated. BP was recorded using an automated cuff monitoring system during triage. Sepsis was defined as a suspected infection with ≥2 qSOFA or SOFA increase ≥2. The outcomes assessed included vasopressor requirement, ICU admission, in-hospital mortality, and total hospital stay. Clinical care followed standard sepsis protocols, and no research-specific interventions were performed.

Statistical Analysis

Data were summarised as mean ± SD and frequency (%). Continuous variables were compared using the independent samples t-test, and categorical variables were compared using the chi-square test. ROC curve analysis was used to determine the optimal SSI and DSI cut-offs and to report the AUC. The diagnostic performance (sensitivity, specificity, PPV, NPV, and accuracy) was calculated using cross-tabulation. Statistical significance was set at $p < 0.05$. Analyses were performed using IBM SPSS Statistics version 29.0.

RESULTS

A total of 100 patients with sepsis were enrolled in the study. The study population showed a male predominance, with 57 (57.0%) males and 43 (43.0%) females. The most common provisional diagnosis in the ED was abdominal sepsis (25.0%), followed by urinary tract infection (UTI) (19.0%). The most frequent presenting complaint was general

weakness (21.0%). The most prevalent comorbidities were ischaemic heart disease (36.0%) and chronic kidney disease (34.0%). Overall, 21 patients (21.0%) progressed to septic shock, 23 (23.0%) required vasopressor support, 30 (30.0%) required ICU admission, and the in-hospital mortality rate was 8.0%. [Table 1]

Table 1: Baseline demographic characteristics, clinical profile, comorbidities, and outcomes

Variable	Category	Frequency (%)
Sex	Male	57 (57%)
	Female	43 (43%)
ER provisional diagnosis	Abdominal sepsis	25 (25%)
	UTI	19 (19%)
	COVID sepsis	17 (17%)
	Meningitis	17 (17%)
	Cellulitis	11 (11%)
	Pneumonia	11 (11%)
Clinical complaints	Weakness	21 (21%)
	Vomiting	16 (16%)
	Fever	15 (15%)
	Altered sensorium	15 (15%)
	Dysuria	13 (13%)
	Cough	11 (11%)
	Breathlessness	9 (9%)
Comorbidity	Diabetes mellitus	31 (31%)
	Hypertension	16 (16%)
	Ischemic heart disease	36 (36%)
	Chronic kidney disease	34 (34%)
	Malignancy	14 (14%)
	Other comorbidities	25 (25%)
Progression to septic shock		21 (21%)
Vasopressor requirement		23 (23%)
ICU admission		30 (30%)
In-hospital mortality		8 (8%)

The mean age of the study population was 60.67 ± 16.38 years, and the mean duration of symptoms prior to presentation was 2.29 ± 1.38 days. On arrival, the mean respiratory rate was 26.04 ± 5.01 breaths/min, mean oxygen saturation (SpO_2) was 92.89 ± 4.12%, and mean baseline lactate level was 2.08 ± 1.08 mmol/L. The triage vital signs showed a mean systolic shock index (SSI) of 0.80 ± 0.19 and a mean diastolic shock index (DSI) of 1.37 ± 0.33.

At the time of vasopressor initiation, patients had hypotension (MAP 66.00 ± 5.38 mmHg) with tachycardia (heart rate 121.33 ± 10.71 bpm), with elevated SSI (1.32 ± 0.15) and DSI (2.35 ± 0.36) levels. Laboratory evaluation showed leukocytosis (WBC 14.44 ± 4.21 $\times 10^9$ /L), elevated procalcitonin (9.91 ± 4.78 ng/mL), and raised creatinine (1.42 ± 0.66 mg/dL). Clinical deterioration was rapid, with progression to shock occurring at 2.76 ± 1.70 h and vasopressor initiation at 3.71 ± 1.15 h after presentation. [Table 2]

Table 2: Baseline demographic profile, clinical parameters, laboratory values, and time-to-event measures

Parameter	Mean ± SD
Age (years)	60.67 ± 16.38
Duration of symptoms (days)	2.29 ± 1.38
Baseline lactate (mmol/L)	2.08 ± 1.08
Hospital stay (days)	6.34 ± 3.32
Vitals on arrival	
Temperature (°C)	37.76 ± 0.75
Respiratory rate (breaths/min)	26.04 ± 5.01
SpO_2 (%)	92.89 ± 4.12
MAP (mmHg)	86.77 ± 9.55
Triage vitals and indices	
Heart rate (bpm)	96.61 ± 19.15
SBP (mmHg)	122.16 ± 14.99
DBP (mmHg)	71.78 ± 9.38
SSI	0.80 ± 0.19
DSI	1.37 ± 0.33
At vasopressor initiation (for patients requiring vasopressors)	
Temperature (°C)	38.03 ± 0.36
SBP (mmHg)	92.90 ± 8.54
DBP (mmHg)	52.62 ± 6.74
Heart rate (bpm)	121.33 ± 10.71
Respiratory rate (breaths/min)	28.19 ± 5.60
SpO_2 (%)	91.90 ± 3.92

MAP (mmHg)	66.00 ± 5.38
SSI	1.32 ± 0.15
DSI	2.35 ± 0.36
WBC ($\times 10^9$ /L)	14.44 ± 4.21
Creatinine (mg/dL)	1.42 ± 0.66
Procalcitonin (ng/mL)	9.91 ± 4.78
Time-to-event measures	
Time to progression into shock (hours)	2.76 ± 1.70
Time to vasopressor initiation (hours)	3.71 ± 1.15

Patients who progressed to septic shock had significantly higher DSI values at triage (1.62 ± 0.37 vs. 1.30 ± 0.29; p = 0.015) and significantly higher lactate levels (3.72 ± 0.95 vs. 1.64 ± 0.58 mmol/L; p = 0.001). Patients who developed septic shock also had a higher heart rate (110.48 ± 21.19 vs. 92.92 ± 16.87 bpm; p = 0.060) and lower systolic blood pressure (114.76 ± 12.21 vs. 124.13 ± 15.11 mmHg; p = 0.074), although these were not significant. Age, symptom duration, temperature, respiratory rate, oxygen saturation, WBC count, creatinine, and procalcitonin levels were comparable between the groups. [Table 3]

Table 3: Comparison of baseline clinical and laboratory parameters between patients with and without progression to septic shock

Variable	Progressed to septic shock		p value
	Yes	No	
Age (years)	61.81 ± 15.29	60.37 ± 16.74	0.682
Duration of symptoms (days)	2.67 ± 1.59	2.19 ± 1.31	0.124
Temperature (°C)	37.74 ± 0.69	37.77 ± 0.77	0.516
Hospital stay (days)	10.48 ± 2.91	5.24 ± 2.45	0.214*
HR at triage (bpm)	110.48 ± 21.19	92.92 ± 16.87	0.06
SBP at triage (mmHg)	114.76 ± 12.21	124.13 ± 15.11	0.074
DBP at triage (mmHg)	68.95 ± 6.98	72.53 ± 9.83	0.142
RR (breaths/min)	25.43 ± 4.85	26.20 ± 5.07	0.402
SpO_2 (%)	93.29 ± 4.64	92.78 ± 4.00	0.142
SSI at triage	0.97 ± 0.19	0.76 ± 0.17	0.658*
DSI at triage	1.62 ± 0.37	1.30 ± 0.29	0.015
Lactate (mmol/L)	3.72 ± 0.95	1.64 ± 0.58	0.001
WBC ($\times 10^9$ /L)	13.20 ± 4.72	14.77 ± 4.03	0.185
Creatinine (mg/dL)	1.30 ± 0.64	1.46 ± 0.66	0.778
Procalcitonin (ng/mL)	11.09 ± 4.77	9.59 ± 4.76	0.802

Sex distribution was similar between the groups (male: 52.4% vs. 58.2%; p = 0.630). The ED provisional diagnoses and presenting complaints were comparable between patients who progressed to septic shock and those who did not (p = 0.627 and p = 0.989, respectively). Comorbidities, including diabetes mellitus, hypertension, ischaemic heart disease, chronic kidney disease, malignancy, and other comorbidities, were not significantly associated with progression to septic shock (all p > 0.05). Patients who progressed to septic shock had significantly higher vasopressor requirements (100.0% vs. 2.5%; p < 0.0001), higher ICU admission rates (100.0% vs. 11.4%; p < 0.0001), and higher in-hospital mortality rates (23.8% vs. 3.8%; p = 0.003). [Table 4]

Table 4: Comparison of demographic characteristics, clinical presentation, comorbidities, and outcomes between patients with and without progression to septic shock

Variable	Category	Progressed to septic shock		p value
		Yes	No	
Sex	Male	11 (52.4)	46 (58.2)	0.63
	Female	10 (47.6)	33 (41.8)	
ED provisional diagnosis	Abdominal sepsis	6 (28.6)	19 (24.1)	0.627
	Cellulitis	1 (4.8)	10 (12.7)	
	COVID sepsis	3 (14.3)	14 (17.7)	
	Meningitis	4 (19.0)	13 (16.5)	
	Pneumonia	1 (4.8)	10 (12.7)	
	UTI	6 (28.6)	13 (16.5)	
Presenting complaints	Altered sensorium	4 (19.0)	11 (13.9)	0.989
	Breathlessness	2 (9.5)	7 (8.9)	
	Cough	3 (14.3)	8 (10.1)	
	Dysuria	2 (9.5)	11 (13.9)	
	Fever	3 (14.3)	12 (15.2)	
	Vomiting	3 (14.3)	13 (16.5)	
	Weakness	4 (19.0)	17 (21.5)	
Diabetes mellitus	Yes	7 (33.3)	24 (30.4)	0.795
	No	14 (66.7)	55 (69.6)	
Hypertension	Yes	5 (23.8)	11 (13.9)	0.272

	No	16 (76.2)	68 (86.1)	
Ischaemic heart disease	Yes	5 (23.8)	31 (39.2)	0.19
	No	16 (76.2)	48 (60.8)	
Chronic kidney disease	Yes	6 (28.6)	28 (35.4)	0.555
	No	15 (71.4)	51 (64.6)	
Malignancy	Yes	1 (4.8)	13 (16.5)	0.17
	No	20 (95.2)	66 (83.5)	
Other comorbidities	Yes	4 (19.0)	21 (26.6)	0.478
	No	17 (81.0)	58 (73.4)	
Vasopressor use	Yes	21 (100.0)	2 (2.5)	<0.0001
	No	0 (0.0)	77 (97.5)	
ICU admission	Yes	21 (100.0)	9 (11.4)	<0.0001
	No	0 (0.0)	70 (88.6)	
In-hospital mortality	Died	5 (23.8)	3 (3.8)	0.003
	Survived	16 (76.2)	76 (96.2)	

Using ROC-derived cut-offs, a high SSI (>0.83) was observed in 85.7% of patients who progressed to septic shock, compared with 31.6% of those who did not. A high DSI (>1.4) was present in 81.0% of patients with septic shock compared with 50.6% of patients without shock. [Table 5]

Table 5: Distribution of patients based on SSI and DSI cut-off values

Index		Progressed to septic shock	
		Yes	No
SSI triage	>0.83	18 (85.7%)	25 (31.6%)
	<0.82	3 (14.3%)	54 (68.4%)
DSI triage	>1.4	17 (81.0%)	40 (50.6%)
	<1.3	4 (19.0%)	39 (49.4%)

On ROC curve analysis, SSI demonstrated better predictive performance than DSI (AUC 0.812 vs. 0.726). The optimal cutoff values were 0.82 for SSI and 1.3 for DSI. SSI had a sensitivity of 85.71% and specificity of 68.35%, whereas DSI had a sensitivity of 80.95% and specificity of 49.37%. PPV was low for both indices, whereas NPV was high (SSI, 94.74%; DSI, 90.70%). The accuracy was higher for the SSI (72.00%) than for the DSI (56.00%). [Table 6, Figure 1]

Table 6: Diagnostic accuracy of SSI and DSI in predicting progression to septic shock

Parameter	SSI	DSI
Cut-off	0.82	1.3
AUC	0.812	0.726
p value	<0.0001	0.002
Sensitivity	85.71%	80.95%
Specificity	68.35%	49.37%
PPV	41.86%	29.82%
NPV	94.74%	90.70%
Accuracy	72.00%	56.00%

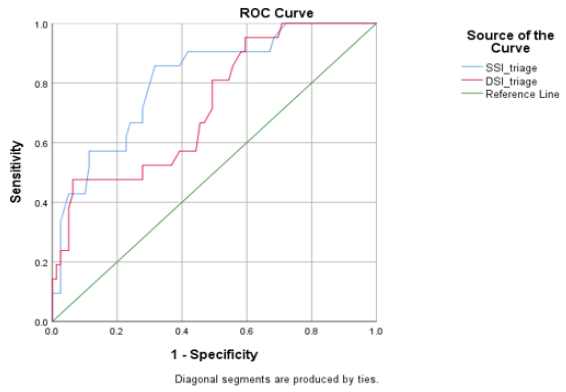

Figure 1: ROC curve analysis for SSI and DSI at triage

DISCUSSION

In this prospective ED cohort, 21% of patients progressed to septic shock. The DSI at triage and baseline lactate levels were significantly higher in patients who developed septic shock, whereas the SSI showed better overall ROC performance. Age distribution and sex were comparable between patients who progressed to septic shock and those who did not, with no significant associations. Studies show that 60-65% of patients with sepsis are over 60 years of age, and 64.7% are aged 50-80 years. While older age and male sex correlated with worse outcomes, age alone did not independently predict adverse events.[15,16] Rose et al. found that sepsis mainly affected those aged 50-70 years, noting a complex age-sex interaction.[17] In this cohort, age and sex were not significantly associated with progression to septic shock.

The patterns of presenting complaints, symptom duration, and emergency diagnoses were similar between the groups and did not predict shock progression. The presenting symptoms did not differ significantly between the groups in this cohort. Jeon et al. found baseline clinical presentation did not differ between patients who developed shock and those remaining stable.[18] Zhang et al. demonstrated

that while haemodynamic indices correlated with mortality, presenting symptoms did not significantly differ between survivors and non-survivors.[19]

In the current study, chronic illnesses were common, but comorbidities were not significantly associated with progression to septic shock in this cohort. This suggests that while comorbidities may increase vulnerability, they do not independently cause early haemodynamic deterioration. Kang et al. noted hypertension and diabetes were prevalent but didn't predict early clinical deterioration; only conditions like chronic kidney disease, liver disease, and malignancy were more linked to mortality than shock.[20] Holder et al. found triage diastolic blood pressure and hypoalbuminaemia were stronger predictors of early severe sepsis or shock, not chronic comorbidities.[21] Javier et al. suggested CKD and long-standing hypertension may predispose to septic complications, but their impact depends more on acute organ dysfunction severity than sepsis progression.[22] These observations support that the shift from sepsis to septic shock is driven by acute physiological changes.

Higher triage DSI and lactate levels were associated with progression to septic shock, whereas HR and SBP showed non-significant trends. Heart rate and systolic blood pressure showed greater derangement in patients progressing to shock; however, isolated parameters were insufficient predictors. The DSI at triage was significantly associated with shock development. Ospina-Tascón et al. reported median heart rates of 103–104 bpm, systolic pressures of 92–100 mmHg, diastolic pressures of 45–52 mmHg, and DSI values of 1.97–2.28 at vasopressor initiation, showing haemodynamic indices' stronger prognostic value.6 Puskarich et al. found overt shock patients had lower systolic pressure (85 vs 108 mmHg, $p<0.01$), while cryptic shock patients showed higher heart rate (114 vs. 102 beats/min, $p=0.04$) and respiratory rate (26 vs. 22 breaths/min, $p=0.01$).[23] Wira et al. confirmed isolated vital signs lacked predictive accuracy, but patients with sustained shock index elevation had lower systolic pressure and higher heart rate versus those without.[24]

SSI was higher in the septic shock group, but this was not statistically significant.[Table 3] Baseline lactate levels were higher among patients who progressed to septic shock, whereas WBC count, creatinine, and procalcitonin levels were not predictive. Jansen et al. showed elevated lactate correlated with early haemodynamic compromise.[25] Mikkelsen et al. demonstrated lactate levels predicted 28-day mortality without hypotension.26 Kim et al. identified lactate as an independent predictor of vasopressor requirement.[27]

At vasopressor initiation, patients who progressed to septic shock showed tachycardia, hypotension, reduced oxygen saturation, and fever, with elevated SSI and DSI. These values represent the time of vasopressor initiation, rather than the triage measurements. Ospina-Tascón et al. reported similar profiles with systolic pressures of 92–100 mmHg, diastolic pressures of 45–52 mmHg, mean arterial pressures of 63–66 mmHg, heart rates of 103–104 bpm, and median DSI values of 1.97–2.28.6 Puskarich et al. observed lower systolic pressures in overt shock versus cryptic shock (85 vs 108 mmHg, $p<0.01$), with both groups achieving resuscitation targets.23 Wira et al. showed patients with elevated shock index required more vasopressors (38.6% vs 11.6%, $p=0.0001$) and had higher mortality (41.7% vs 7.2%, $p=0.0001$).[24]

In this study, 21% of the patients progressed to septic shock, requiring vasopressors and ICU admission. The mortality rate was higher in the septic shock group. Hospital stay was longer in the septic shock group, although the reported p-value was not significant. Similar findings by Vucelić et al. and Naqvi et al. noted increased ICU mortality and vasopressor use in shock patients.[28,29] Mostafa et al. showed over 50% mortality in septic shock with cardiovascular failure, confirming that septic shock worsens survival and increases critical care burden.[30]

Both SSI and DSI showed high NPV for excluding progression to septic shock. The SSI exhibited superior diagnostic performance, with higher sensitivity, specificity, AUC, and accuracy at triage. This aligns with Zhang et al., who reported better AUC and discrimination for SI over DSI in early mortality prediction.[19] Avci et al. found SI had a higher AUC than dSI in predicting in-hospital outcomes.[31] Prasad et al. also showed better sensitivity and specificity for SI over modified indices, supporting SSI as a reliable screening tool.[32] Using ROC-derived cut-offs (SSI 0.82, DSI 1.3), SSI showed better discrimination than DSI.

Limitations

This single-centre design limits external validity, and the small sample size limits subgroup analyses. The lack of repeated measurements during emergency evaluation restricted the assessment of haemodynamic changes. Echocardiographic parameters, lactate clearance, and long-term outcomes were not captured, limiting the evaluation of cardiac dysfunction. Excluding hypotensive patients limits the applicability of this study to patients presenting with overt shock.

CONCLUSION

Individual vital signs at triage showed limited predictive value for the progression to septic shock. Triage DSI and baseline lactate levels were significantly associated with septic shock progression, whereas SSI showed better overall predictive performance in ROC analysis. Patients who progressed to septic shock had higher vasopressor requirements, ICU admissions, and in-hospital mortality. SSI and DSI may support early risk stratification at triage, but multicentre studies are required for validation and generalisability.

REFERENCES

1. Gavelli F, Castello LM, Avanzi GC. Management of sepsis and septic shock in the emergency department. Intern Emerg Med 2021;16:1649–61. https://doi.org/10.1007/s11739-021-02735-7.
2. Sasko B, Butz T, Prull MW, Liebeton J, Christ M, Trappe H-J. Earliest bedside assessment of hemodynamic parameters and cardiac biomarkers: Their role as predictors of adverse outcome in patients with septic shock. Int J Med Sci 2015;12:680–8. https://doi.org/10.7150/ijms.11720.
3. Sharma A, Naga Satish U, Tevatia MS, Singh SK. Prehospital shock index, modified shock index, and pulse pressure heart rate ratio as predictors of massive blood transfusions in modern warfare injuries: A retrospective analysis. Med J Armed Forces India 2019;75:171–5. https://doi.org/10.1016/j.mjafi.2018.07.002.
4. Prashanth V. N. S. Shock index as a predictor of vasopressor use in patients with sepsis. Int J Adv Med 2019. https://doi.org/10.18203/2349-3933.ijam20193624.
5. Magder SA. The Highs and Lows of Blood Pressure: Toward Meaningful Clinical Targets in Patients With Shock. Crit Care Med 2014;42:1241. https://doi.org/10.1097/CCM.0000000000000324.
6. Ospina-Táscon G, Teboul J, Hernández G, Alvarez I, Sánchez-Ortiz ÁI, Calderón-Tapia LE, et al. Diastolic shock index and clinical outcomes in patients with septic shock. Ann Intensive Care 2020;10. https://doi.org/10.1186/s13613-020-00658-8.
7. Sarkar S, Azim A, Mishra P, Chahar JS, Gurjar M, Poddar B. Temporal Analysis of Diastolic Shock Index in Patients with Septic Shock and its Correlation with Clinical Outcomes in Indian Setting – A Prospective Observational Study. Indian J Crit Care Med Peer-Rev Off Publ Indian Soc Crit Care Med 2025;29:194–194. https://doi.org/10.5005/jaypee-journals-10071-24933.143.
8. Srinivasan A, Saroj B, Prusty K. Comparison of Diastolic Shock Index with That of Lactates in Predicting Outcomes In Septic Shock – A Prospective Study. Indian J Crit Care Med Peer-Rev Off Publ Indian Soc Crit Care Med 2025;29:223–223. https://doi.org/10.5005/jaypee-journals-10071-24933.168.
9. Landesberg G, Gilon D, Meroz Y, Georgieva M, Levin P, Goodman S, et al. Diastolic dysfunction and mortality in severe sepsis and septic shock. Eur Heart J 2012;33:895–903. https://doi.org/10.1093/eurheartj/ehr351.
10. Rolando G, Espinoza EDV, Avid E, Welsh S, Pozo JF del, Vazquez AR, et al. Prognostic value of ventricular diastolic dysfunction in patients with severe sepsis and septic shock. Rev Bras Ter Intensiva 2015;27:333–9. https://doi.org/10.5935/0103-507x.20150057.
11. Mu C-T, Lin Y-J, Chen C-H, Hsia S, Lin J-J, Chan O, et al. Diastolic/systolic blood pressure ratio for predicting febrile children with sepsis and progress to septic shock in the emergency department. BMC Emerg Med 2024;24. https://doi.org/10.1186/s12873-024-00995-y.
12. Natanson C, Fink M, Ballantyne H, MacVittie T, Conklin J, Parrillo J. Gram-negative bacteremia produces both severe systolic and diastolic cardiac dysfunction in a canine model that simulates human septic shock. J Clin Invest 1986;78:259–70. https://doi.org/10.1172/jci112559.
13. Aseri ZAA, Ageel MA, Binkharfi M. The use of the shock index to predict hemodynamic collapse in hypotensive sepsis patients: A cross-sectional analysis. Saudi J Anaesth 2020;14:192–9. https://doi.org/10.4103/sja.sja_780_19.
14. Berger T, Green JP, Horeczko T, Hagar Y, Garg N, Suarez A, et al. Shock Index and Early Recognition of Sepsis in the Emergency Department: Pilot Study. West J Emerg Med 2013;14:168–74. https://doi.org/10.5811/westjem.2012.8.11546.
15. Nasa P, Juneja D, Singh O. Severe sepsis and septic shock in the elderly: An overview. World J Crit Care Med 2012;1:23–30. https://doi.org/10.5492/wjccm.v1.i1.23.
16. Ko RE, Kang D, Cho J, Na SJ, Chung CR, Lim SY, et al. Influence of gender on age-associated in-hospital mortality in patients with sepsis and septic shock: a prospective nationwide multicenter cohort study. Crit Care 2023;27:229. https://doi.org/10.1186/s13054-023-04515-5.
17. Rose N, Agrama I, Nachtigall I, Pletz MW, Rosendahl J, Chung H-Y, et al. Sex differences in sepsis outcomes across the lifespan: a population-based cohort study in Germany. Crit Care 2025;29:408. https://doi.org/10.1186/s13054-025-05657-4.
18. Jeon Y, Kim S, Ahn S, Park J-H, Cho H, Moon S, et al. Predicting septic shock in patients with sepsis at emergency department triage using systolic and diastolic shock index. Am J Emerg Med 2024;78:196–201. https://doi.org/10.1016/j.ajem.2024.01.029.
19. Zhang T-N, Hao P-H, Gao S-Y, Liu C-F, Yang N. Evaluation of SI, MSI and DSI for very early (3-day) mortality in patients with septic shock. Eur J Med Res 2022;27:227. https://doi.org/10.1186/s40001-022-00857-y.
20. Kang C, Choi S, Jang EJ, Joo S, Jeong JH, Oh S-Y, et al. Prevalence and outcomes of chronic comorbid conditions in patients with sepsis in Korea: a nationwide cohort study from 2011 to 2016. BMC Infect Dis 2024;24:184. https://doi.org/10.1186/s12879-024-09081-x.
21. Holder AL, Gupta N, Lulaj E, Furgiuele M, Hidalgo I, Jones MP, et al. Predictors of early progression to severe sepsis or shock among emergency department patients with nonsevere sepsis. Int J Emerg Med 2016;9:10. https://doi.org/10.1186/s12245-016-0106-7.
22. Javier RM, Salim J, Aji BL, Pradipta BPA, Nur C, Muhammad I, et al. Risk factors for chronic kidney disease and septic shock with hypertension in adults and children. Front Nephrol 2025;5:1671763. https://doi.org/10.3389/fneph.2025.1671763.
23. Puskarich MA, Trzeciak S, Shapiro NI, Heffner AC, Kline JA, Jones AE. Outcomes of patients undergoing early sepsis resuscitation for cryptic shock compared with overt shock. Resuscitation 2011;82:1289–93. https://doi.org/10.1016/j.resuscitation.2011.06.015.
24. Wira CR, Francis MW, Bhat S, Ehrman R, Conner D, Siegel M. The shock index as a predictor of vasopressor use in emergency department patients with severe sepsis. West J Emerg Med 2014;15:60–6. https://doi.org/10.5811/westjem.2013.7.18472.
25. Jansen TC, van Bommel J, Schoonderbeek FJ, Sleeswijk Visser SJ, van der Klooster JM, Lima AP, et al. Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial: A multicenter, open-label, randomized controlled trial. Am J Respir Crit Care Med 2010;182:752–61. https://doi.org/10.1164/rccm.200912-1918OC.
26. Mikkelsen ME, Miltiades AN, Gaieski DF, Goyal M, Fuchs BD, Shah CV, et al. Serum lactate is associated with mortality in severe sepsis independent of organ failure and shock. Crit Care Med 2009;37:1670–7. https://doi.org/10.1097/CCM.0b013e31819fcf68.
27. Kim DS, Park JE, Hwang SY, Jeong D, Lee GT, Kim T, et al. Prediction of vasopressor requirement among hypotensive patients with suspected infection: usefulness of diastolic shock index and lactate. Clin Exp Emerg Med 2022;9:176–86. https://doi.org/10.15441/ceem.22.324.
28. Vucelić V, Klobučar I, Đuras-Cuculić B, Gverić Grginić A, Prohaska-Potočnik C, Jajić I, et al. Sepsis and septic shock - an observational study of the incidence, management, and mortality predictors in a medical intensive care unit. Croat Med J 2020;61:429–39. https://doi.org/10.3325/cmj.2020.61.429.
29. Naqvi F, Jain P, Umer A, Rana B, Hadique S. Outcomes of patients with sepsis and septic shock requiring source control: A prospective observational single-center study. Crit Care Explor 2022;4:e0807. https://doi.org/10.1097/CCE.0000000000000807.
30. Mostafa M, Hamed L, Mokhtar S, Arafa M. Analysis of mortality factors in ICU patients with sepsis and septic shock: a retrospective study. Egypt J Crit Care Med 2025;12. https://doi.org/10.1007/s44349-024-00012-y.
31. Avci M, Doganay F. Prognostic performance of shock index, diastolic shock index, age shock index, and modified shock index in COVID-19 pneumonia. Disaster Med Public Health Prep 2022;17:e189. https://doi.org/10.1017/dmp.2022.110.
32. Prasad KJD, Bindu KCH, Abhinov T, Moorthy K, Rajesh K. A comparative study on predictive validity of modified shock index, shock index, and age shock index in predicting the need for mechanical ventilation among sepsis patients in a tertiary care hospital. J Emerg Trauma Shock 2023;16:17–21. https://doi.org/10.4103/jets.jets_118_22.