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Original Research Article

Diagnostic and prognostic utility of shock index, modified shock index and lactate in early sepsis risk stratification in the emergency department

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ABSTRACT

Background: Early risk stratification of sepsis in the ED is crucial. Simple markers such as Shock Index (SI), Modified Shock Index (MSI) and serum lactate may be useful to identify high-risk patients.
Methods: This was an observational cohort study performed at the KSDC Hospital & Research Centre, Thanjavur, with a study period from 12/2/ 25 to 13/8/25. 200 adults presenting with Sepsis-3 sepsis were analysed of 236 patients screened. The differences in SI, MSI, and initial lactate between severity groups and outcomes were analysed with t-tests and Pearson correlation.
Results: Ninety patients (45.0%) had sepsis, 70 (35.0%) had severe sepsis, and 40 (20.0%) had septic shock. SI, MSI and lactate rose progressively with severity (mean SI 0.84, 1.06, 1.47; MSI 1.13, 1.42, 1.99; lactate 1.8, 3.1, 5.4 mmol/L; all $p<0.001$). In-hospital mortality was 19.0%, 31.0% in the intensive care unit (ICU), and 22.0% were on vasopressors. Non-survivors had higher SI (1.38 vs 0.97), MSI (1.82 vs 1.31) and lactate (5.1 vs 2.5 mmol/L) (all $p<0.001$), and values were also higher with ICU admission and vasopressor use. All three were positively correlated with length of stay ($r=0.34–0.41$).
Conclusions: These are the markers of severity of sepsis and associated mortality - SI, MSI and lactate. SI and MSI are fast bedside tests, and lactate provides additional information, so combining these tools could be beneficial for early risk stratification in the ED. Cut-offs need to be validated in multicentre studies using ROC analysis.

Keywords: Sepsis, Shock index, Modified shock index, Lactate, Emergency department, Risk stratification

INTRODUCTION

Overview and clinical burden of sepsis

Acute sepsis presents significant medical, economic, and operational difficulties. Hospital admissions for septicaemia or sepsis have more than doubled in the last ten years, rising from 811,644 in 2012 to 1,136,889 in 2018, with no significant rise in total hospitalisations over that timeframe.[1] While this accounts for about 2% of hospital admissions, these individuals constitute approximately 17% of fatalities occurring within the hospital. As many as 46% of hospitalised patients with sepsis are admitted via a decreasing number of emergency departments (EDs).[2]

Sepsis is a leading cause of death and sickness worldwide.[3] It is defined as a dangerous failure of organs caused by the body's abnormal response to infection. The ability to detect sepsis quickly and administer treatment immediately is crucial for improving patient survival rates.[4] Because of this, there is a big need for reliable and easy-to-understand

tools that can quickly evaluate patient risk and predict outcomes in places where important decisions are made, like the emergency department (ED). As a result, assessment systems such as qSOFA (rapid SOFA) and SOFA (Sequential Organ Failure Assessment) have been developed. Despite the fact that SOFA is a standard tool for assessing organ damage in ICUs, qSOFA has been promoted for the rapid identification of vulnerable patients in the ED due to its ease of use at the bedside.[5,6] On the flip side, serum lactate concentration is a biomarker that has been used for a long time to evaluate the prognosis of septic shock and sepsis.[7] It indicates that the tissues are not getting enough blood. As a biomarker of haemodynamic instability, the shock index (SI) and its variants have attracted study alongside these established markers. 'The SI (heart rate/systolic blood pressure) has been widely used in trauma and hypovolemic shock cases due to its ease of computation and cost-effectiveness' since its articulation in 1967.[8,9] 'Age-Adjusted Shock Index (ASI), which takes into account the effect of ageing on haemodynamic response, MSI, which adds mean arterial pressure, and Reverse Shock Index (RSI), which changes the original formula' are just a few of the many variations that have evolved over the years.[10,11] Previous research on these indices' prognostic value in sepsis patients has shown mixed results; furthermore, how they stack up against more traditional markers such as SOFA, qSOFA, and lactate is still up for debate.[12,13]

This study stands out because it uses many shock indices in addition to well-known markers (SOFA, qSOFA, and serum lactate) to evaluate multiple clinical outcomes (ward admission, intensive care unit admission, and one-month death) in a single cohort. Previous research lacks comprehensive documentation of this comparative examination across multiple outcomes. It is hypothesised that shock indices (SI, ASI, MSI and RSI) may possess equal or superior predictive capability compared to traditional indicators (SOFA, qSOFA and lactate) in forecasting clinical outcomes for sepsis patients.[14]

Role of serum lactate in sepsis assessment and prognosis

Severe infection can lead to sepsis, which is defined by biochemical, pathologic, and physiological abnormalities and can be fatal.[15,16] When sepsis goes untreated over an extended period of time, it becomes the leading cause of mortality among infection patients and the tenth leading cause of death worldwide. Patients have a better chance of survival and better outcomes when sepsis is treated early.[17]

A biomarker of metabolic stress, serum lactate level is sensitive yet nonspecific.[18] Hypoxia, stress, and several serious disorders all lead to a rise in lactate, a by-product of anaerobic glycolysis.[19] A higher lactate level is associated with a worse outcome and a higher death rate according to recent studies.[20] Some studies have suggested using different lactate thresholds as a predictor of vigorous resuscitation in the early stages.[21,22]

This suggests that people at risk of unfavourable outcomes may be better identified if elevated serum lactate levels are detected early. As a means of gauging organ dysfunction in ICU patients, the Sequential Organ Failure Assessment (SOFA) and the rapid SOFA (qSOFA) were developed. In addition, investigated whether serum lactate level, SOFA and qSOFA are reliable predictive indicators for sepsis mortality.[23]

Existing evidence regarding SI, MSI and lactate

This prospective observational single-centre study included adult sepsis-3 septic shock patients. MSI, SI, and diastolic shock index were measured at 0, 6, 12 and 24 hours. ΔDSI, lactate clearance, and VIS were analysed using ROC analysis, Cox regression, and multivariable logistic regression.[24]

Research gap and rationale

There is limited evidence on the comparative prognostic value of 'Shock Index (SI), MSI and initial serum lactate to predict in-hospital mortality' in specific ED environments amongst patients with sepsis. In the present study, their discriminatory performance is compared to determine their relative prognostic value.

The study objectives include: comparing the use of SI, MSI and initial lactate to predict in-hospital mortality; evaluate and compare their ROC-AUC values; and evaluate the relationships of these parameters with patient outcomes.

METHODS

Study design and setting

An observational cohort study was conducted to assess the diagnostic and prognostic performance of 'Shock Index (SI), MSI and serum lactate' in initial risk stratification of sepsis. The study was performed at the KSDC Hospital and Research Centre Thanjavur, with a study period from 12/2/25 to 13/8/25.

Study population

The study population was adult patients (18 years or older) attending the ED with a clinical diagnosis of or confirmed sepsis. Demographic, clinical, laboratory and clinical outcome data were gathered to evaluate the association between SI/ MSI, serum lactate, the severity of sepsis and adverse clinical events.

Sampling technique and sample selection

Consecutive sampling was used to recruit 200 patients. Patients who met the specified inclusion and exclusion criteria were randomly allocated to the study during the study period.

Inclusion criteria

The study included adults (18 years of age and older). They presented to the Emergency Department with a diagnosis of suspected or confirmed sepsis. Patients had known heart rate and blood pressure measurements, available serum lactate levels, and sufficient clinical data for evaluation of the severity of sepsis and clinical outcome.

Exclusion criteria

Patients younger than 18 years, those with missing or incomplete clinical records or missing essential physiological measurements and those without available Serum lactate levels for lactate-related analyses were excluded. Duplicate patient records and cases with insufficient information to determine the predefined clinical outcomes were excluded.

Data collection and clinical assessment

A structured data collection form was used to gather data. Age, sex, medical history, presenting complaints, heart rate, SBP, DBP, MAP, RR, O2 sat, temperature, serum lactate concentration and laboratory parameters were included. Clinical outcomes such as intensive care unit (ICU) admission, vasopressor use, hospitalisation, and mortality within the hospital are also collected.

Initial vital signs and serum lactate were recorded as per the predetermined assessment period. Where available, pertinent treatment data are documented, such as fluid resuscitation and administration of vasopressors.

Measurement of shock index

Shock Index is determined by the first heart rate and first systolic blood pressure taken at the time they are assessed in the ED.

"Shock index (SI) = Heart rate (beats/min)/Systolic blood pressure (mmHg)"

The relationships between severity of sepsis and clinical outcomes are evaluated based on the SI values. SI values were analysed for their potential correlation with hemodynamic instability and adverse clinical outcomes at higher values.

Calculating MSI

Heart rate and mean arterial pressure are used to determine the MSI. The following computations for MSI:

MSI = Heart rate (beats/min)/Mean arterial pressure (mmHg)

Clinically recorded or computed mean arterial pressure determined from the systolic and diastolic blood pressure readings using the following formula:

"MAP = (Systolic blood pressure + (2 × diastolic blood pressure)) / 3"

The factors that may correlate with sepsis severity and clinical outcomes were assessed for MSI values and compared with SI and serum lactate.

Serum lactate measurement

Laboratory records were used to obtain serum lactate concentration results, expressed in mmol/L, and those obtained during initial assessment at the Emergency Department were used for the primary analysis. Where available, the timing of lactate samples collected in relation to vital sign measurements and treatment initiation was noted.

Serum lactate was measured as a continuous variable and grouped according to clinically accepted levels, if applicable. Association with severity of sepsis, admission to a critical care unit, use of vasopressors and in-hospital mortality was examined.

Acoustic analysis of speech in clinical outcomes

Septic conditions were defined by established clinical criteria and categorized based on the Sepsis-3 definitions. The severity of sepsis and septic shock was determined by recorded clinical parameters, organ dysfunction and treatment data. The main outcome was the correlation of SI, MSI and serum lactate level with predefined adverse clinical outcomes. Secondary outcomes included in-hospital mortality, length of hospital stay and requirement of vasopressors and admission to the ICU. Appropriate statistical methods were used to assess the relationships between the three parameters and clinical outcomes.

RESULTS

Baseline demographic characteristics

Twenty hundred patients were screened, and 200 patients with complete lactate levels and complete follow-up data were analysed (36 had missing vital signs). By Sepsis-3 criteria, 90 (45.0%) had sepsis, 70 (35.0%) had severe sepsis, and 40 (20.0%) had septic shock. The mean age was 56.4±15.2 years; 118 (59.0%) were male, and 82 (41.0%) were female. Mean age was 54.1±15.8 years in sepsis, 57.2±14.6 in severe sepsis and 59.8±14.9 in septic shock. Comparing the ages of patients with septic shock was not statistically significant (t=1.98, p=0.051). Diabetes mellitus (37.0%), hypertension (41.0%) and chronic kidney disease (18.0%) were the commonest comorbidities.

Clinical characteristics and vital signs

At presentation, the mean heart rate was 106.6±17.2 beats/min, SBP 104.9±20.4 mmHg, DBP 65.0±12.6 mmHg, MAP 78.3±13.9 mmHg, respiratory rate 23.8±5.3

/min, SpO_2 93.1±3.9% and temperature 38.5±0.9 °C. Compared with sepsis, septic shock had a significantly higher heart rate (121.3 vs 98.4 beats/min, t=7.65, p<0.001) and respiratory rate (t=10.5, p<0.001). It also had significantly lower SBP (t=13.1, p<0.001), DBP (t=11.9, p<0.001), MAP (t=14.0, p<0.001) and SpO2 (t=8.0, p<0.001). The temperature exhibited no notable variation (t=0.52, p=0.60).

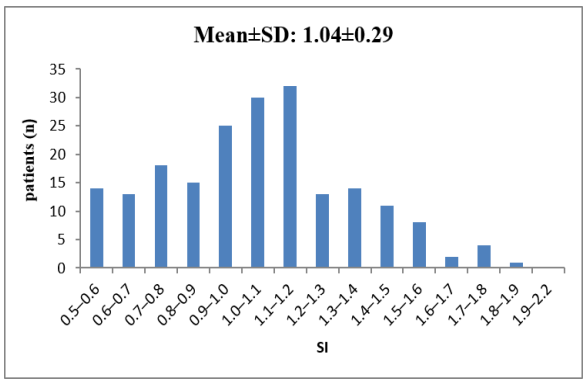


Figure 1: Laboratory and clinical parameters in relation to shock index (SI) at ED presentation (overall SI, mean±SD: 1.04±0.29).

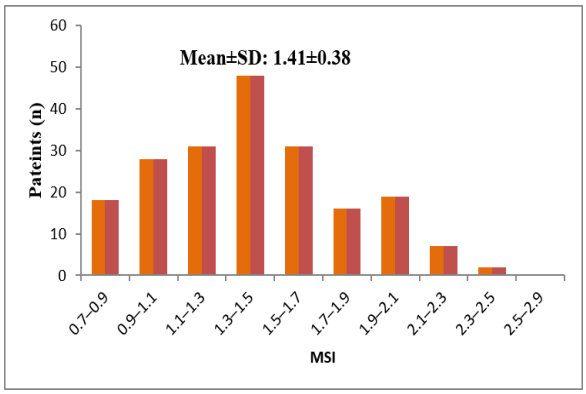


Figure 2: Platelet count in relation to MSI at ED presentation (overall MSI, mean±SD: 1.41±0.38).

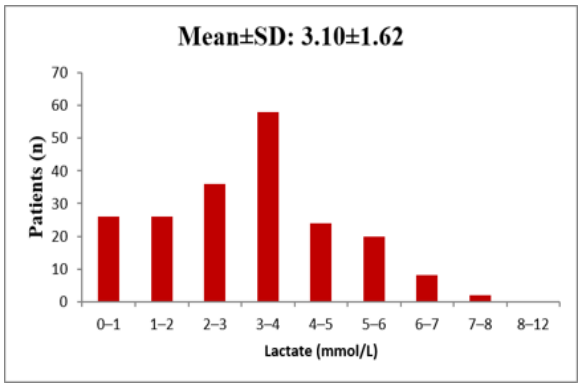


Figure 3: Haemoglobin level in relation to serum lactate at ED presentation (overall lactate, mean±SD: 3.10±1.62 mmol/L).

Distribution of SI, MSI and serum lactate

The mean SI was 1.04±0.30 (range 0.52–2.10), the mean MSI was 1.40±0.40 (range 0.78–2.85), and the mean serum lactate was 2.98±1.85 mmol/L (range 0.6–11.2). SI correlated strongly with MSI (Pearson r=0.91, p<0.001). Lactate correlated moderately with SI (r=0.62, p<0.001) and with MSI (r=0.68, p<0.001).

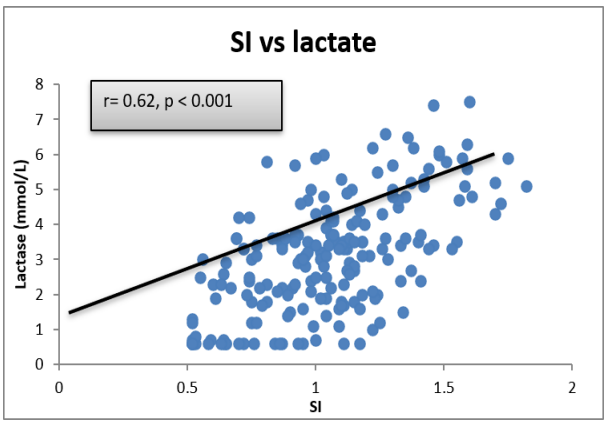


Figure 2: Scatter plots with regression lines for SI vs lactate (r=0.62) and MSI vs lactate (r=0.68).

Comparison across sepsis severity groups

Mean SI was 0.84±0.14 in sepsis, 1.06±0.18 in severe sepsis and 1.47±0.27 in septic shock. Mean MSI was 1.13±0.18, 1.42±0.24 and 1.99±0.36, and mean lactate was 1.8±0.7, 3.1±1.2 and 5.4±2.1 mmol/L, respectively.

All pairwise independent-samples t-tests revealed significant differences between all pairs of groups for all three parameters (all p<0.001; Table 4). Severity grade (coded 1–3) correlated positively with SI (r=0.78), MSI (r=0.81) and lactate (r=0.74) (all p<0.001).

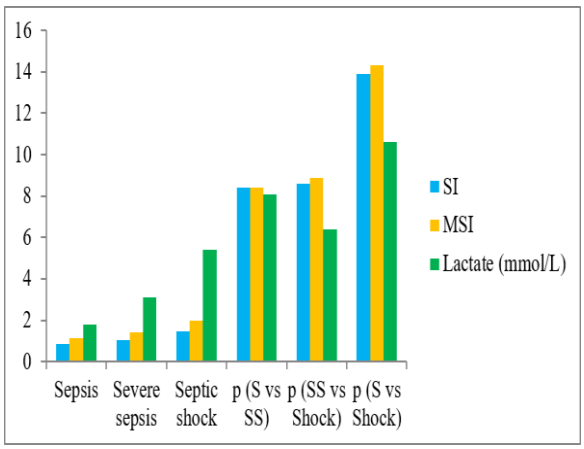


Figure 3: Comparison of serum bilirubin levels between survivors (n=162) and non-survivors (n=38) among ED patients with sepsis.

Relationship with clinical outcomes

Out of the 200 patients, 62 (31.0%) were admitted to the ICU, 44 (22.0%) required vasopressors, and 38 (19.0%) died in hospital. The average length of stay was 7.2±4.1 days.

Non-survivors had significantly higher SI (1.38±0.32 vs 0.97±0.25, p<0.001), MSI (1.82±0.42 vs 1.31±0.33, p<0.001) and lactate (5.1±2.3 vs 2.5±1.4 mmol/L, p<0.001) than survivors. Similar values of all 3 parameters were also observed for patients admitted to the ICU and for those who needed to be given vasopressors (Table 5). There was a positive correlation between length of stay and SI, MSI and lactate (r=0.34, 0.38 and 0.41, respectively, all p<0.001).

Laboratory and clinical parameters

Non-survivors had a significantly higher mean WBC (18.2±6.4 vs 13.6±4.8 ×10³/µl, p<0.001), creatinine (2.6±1.3 vs 1.4±0.8 mg/dl, p<0.001) and bilirubin (2.3±1.6 vs 1.2±0.8 mg/dl, p<0.001). They also had a significantly lower mean platelet count (148±74 vs 214±82×10³/µl, p<0.001) and haemoglobin (10.2±2.1 vs 11.4±1.9 g/dl, p=0.002). Vasopressors were used in 28 (73.7%) non-survivors and 16 (9.9%) survivors, and ICU admission occurred in 30 (78.9%) and 32 (19.8%), respectively.

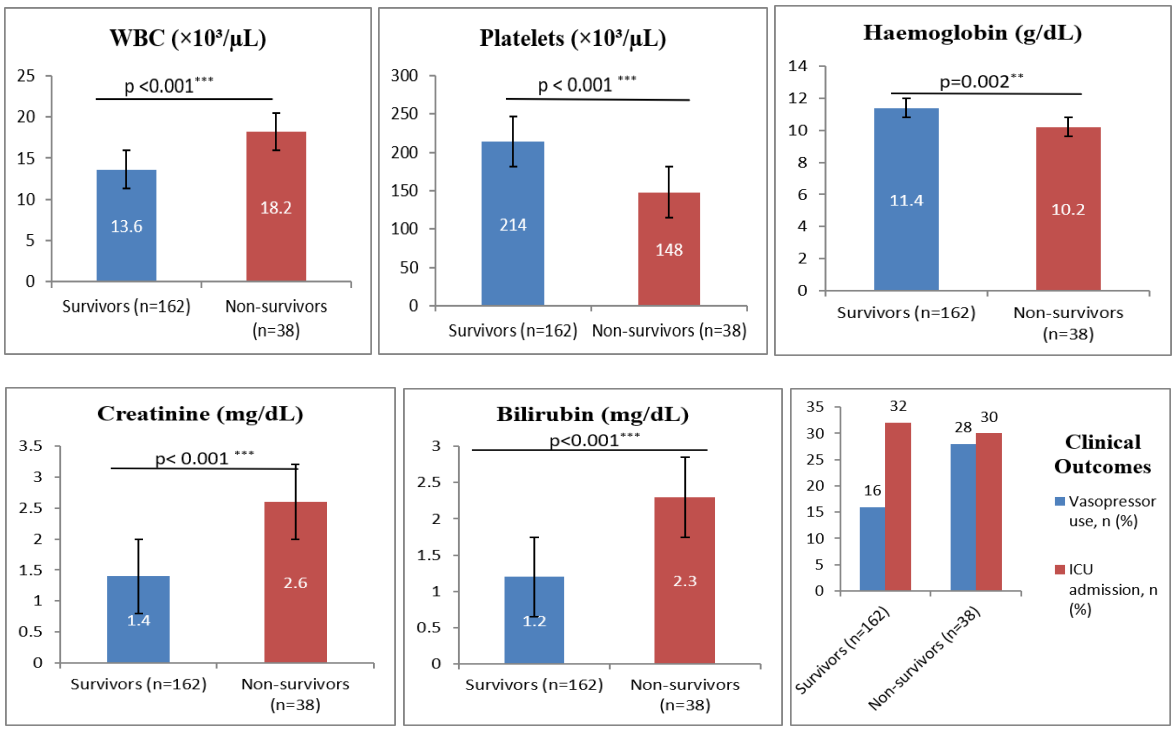


Figure 4: Comparison of laboratory parameters and clinical outcomes between survivors (n=162) and non-survivors (n=38) among ED patients with sepsis.

Statistical significance

Continuous variables are presented as mean±SD. The independent-samples t-test was used to compare differences between two groups (survivors vs non-survivors, ICU vs non-ICU, vasopressor vs no vasopressor, and for each pair of severity groups), and Pearson's correlation coefficient (r) was used to assess the relationship between continuous variables. Categorical variables were reported as n (%) (no inferential testing). P-values from two-tailed tests that are less than 0.05 were considered statistically significant. There was a significant difference in SI, MSI, and lactate between severity groups and between outcome groups (all p<0.001), with the laboratory markers shown in Table 6 (p≤0.002). There were no notable disparities in age (p=0.051) or temperature difference (p=0.60).

Table 1: Baseline demographic characteristics (n=200).

Variables	Overall	Sepsis (n=90)	Severe sepsis (n=70)	Septic shock (n=40)
Age, years (mean±SD)	56.4±15.2	54.1±15.8	57.2±14.6	59.8±14.9
Male, N (%)	118 (59.0)	52 (57.8)	41 (58.6)	25 (62.5)
Female, N (%)	82 (41.0)	38 (42.2)	29 (41.4)	15 (37.5)
Diabetes mellitus, N (%)	74 (37.0)	30 (33.3)	28 (40.0)	16 (40.0)

Continued.

Variables	Overall	Sepsis (n=90)	Severe sepsis (n=70)	Septic shock (n=40)
Hypertension, N (%)	82 (41.0)	34 (37.8)	30 (42.9)	18 (45.0)
Chronic kidney disease, N (%)	36 (18.0)	12 (13.3)	14 (20.0)	10 (25.0)

Table 2: Vital signs at ED presentation (mean±SD).

Parameters	Overall	Sepsis	Severe sepsis	Septic shock	P (Sepsis vs shock)
Heart rate (beats/min)	106.6±17.2	98.4±14.2	108.6±15.1	121.3±16.4	<0.001
SBP (mmHg)	104.9±20.4	116.8±15.6	102.4±16.2	82.6±12.8	<0.001
DBP (mmHg)	65.0±12.6	72.4±10.2	63.8±10.8	50.2±9.6	<0.001
MAP (mmHg)	78.3±13.9	87.2±10.8	76.7±11.2	61.0±9.4	<0.001
Respiratory rate (/min)	23.8±5.3	20.6±3.4	24.8±4.2	29.4±4.8	<0.001
SpO_2 (%)	93.1±3.9	95.2±2.8	92.6±3.4	89.4±4.2	<0.001
Temperature (°C)	38.5±0.9	38.4±0.8	38.6±0.9	38.3±1.1	0.60

Table 3: Distribution and Pearson correlation of SI, MSI and lactate (n=200).

Parameters	Mean±SD	Minimum	Maximum
SI	1.04±0.30	0.52	2.10
MSI	1.40±0.40	0.78	2.85
Lactate (mmol/l)	2.98±1.85	0.6	11.2
Pair	**r**	**P value**	
SI vs MSI	0.91	<0.001	
SI vs Lactate	0.62	<0.001	
MSI vs Lactate	0.68	<0.001	

Table 4: SI, MSI and lactate by sepsis severity (mean±SD).

Parameters	Sepsis	Severe sepsis	Septic shock	p (S vs SS)	p (SS vs Shock)	P (S vs Shock)
SI	0.84±0.14	1.06±0.18	1.47±0.27	<0.001 (t=8.4)	<0.001 (t=8.6)	<0.001 (t=13.9)
MSI	1.13±0.18	1.42±0.24	1.99±0.36	<0.001 (t=8.4)	<0.001 (t=8.9)	<0.001 (t=14.3)
Lactate (mmol/l)	1.8±0.7	3.1±1.2	5.4±2.1	<0.001 (t=8.1)	<0.001 (t=6.4)	<0.001 (t=10.6)

Table 5: SI, MSI and lactate by clinical outcome (mean±SD; independent t-test).

Outcome	Parameters	Yes	No	Mean difference (95% CI)	P value
In-hospital mortality (38 vs 162)	SI	1.38±0.32	0.97±0.25	0.41 (0.30–0.52)	<0.001
	MSI	1.82±0.42	1.31±0.33	0.51 (0.36–0.66)	<0.001
	Lactate	5.1±2.3	2.5±1.4	2.6 (1.82–3.38)	<0.001
ICU admission (62 vs 138)	SI	1.31±0.31	0.91±0.24	0.40 (0.31–0.49)	<0.001
	MSI	1.78±0.40	1.22±0.29	0.56 (0.45–0.67)	<0.001
	Lactate	4.7±2.0	2.1±1.2	2.6 (2.05–3.15)	<0.001
Vasopressor use (44 vs 156)	SI	1.45±0.30	0.93±0.25	0.52 (0.42–0.62)	<0.001
	MSI	1.96±0.38	1.24±0.30	0.72 (0.60–0.84)	<0.001
	Lactate	5.2±2.1	2.3±1.3	2.9 (2.23–3.57)	<0.001
Parameter		**r**	**P value**		
SI		0.34	<0.001		
MSI		0.38	<0.001		
Lactate		0.41	<0.001		

Table 6: Laboratory and clinical parameters by outcome (mean±SD; independent t-test).

Parameters	Survivors (n=162)	Non-survivors (n=38)	t	P value
WBC (×10³/µl)	13.6±4.8	18.2 ± 6.4	4.16	<0.001
Platelets (×10³/µl)	214±82	148 ± 74	4.85	<0.001
Haemoglobin (g/dl)	11.4±1.9	10.2 ± 2.1	3.23	0.002
Creatinine (mg/dl)	1.4±0.8	2.6 ± 1.3	5.45	<0.001
Bilirubin (mg/dl)	1.2±0.8	2.3 ± 1.6	4.12	<0.001
Vasopressor use, N (%)	16 (9.9)	28 (73.7)	–	–
ICU admission, N (%)	32 (19.8)	30 (78.9)	–	–

DISCUSSION

In the present study, 200 patients who were admitted to the ED were evaluated to see if there was any association between SEPSIS, Shock Index (SI), MSI, serum lactate levels, and survival. The results showed a gradual rise in all three parameters in the three groups: sepsis, severe sepsis and septic shock. The mean SI values were 0.84 to 1.06 and 1.47, respectively, while mean MSI values were 1.13 to 1.42 and 1.99, respectively. Similarly, the mean lactate levels in the serum rose from 1.8 to 3.1 and 5.4 mmol/L. All the above-mentioned differences were statistically significant (all pairwise $p<0.001$). SI ($r=0.78$), MSI ($r=0.81$) and lactate ($r=0.74$) also correlated positively with severity of sepsis, suggesting that there are links between tissue hypoperfusion and haemodynamic instability and increasing severity of sepsis.

All three parameters were significantly higher among non-survivors than survivors (SI: 1.38 vs. 0.97; MSI: 1.82 vs. 1.31; lactate: 5.1 vs. 2.5 mmol/L; $p<0.001$). Higher scores were also found in patients who needed intensive care unit (ICU) admission and vasopressor support. In addition, there were positive correlations between lactate, length of hospital stay, and SI, with SI being the strongest ($r=0.41$). The strong correlation between SI and MSI ($r=0.91$) suggests that both indices give related haemodynamic information and that lactate measurement may provide additional information about metabolic disturbances and tissue hypoperfusion, as there were moderate correlations between lactate and both indices. This slight relationship between MSI and severity of sepsis may be due to the fact that it includes mean arterial pressure (MAP), which is a combination of the systolic and diastolic blood pressures.

Levels of white blood cells, creatinine, and bilirubin were also higher in the non-survivors, while platelet and haemoglobin levels were lower. These results suggest that the systemic inflammatory activity and organ dysfunction were greater in patients with adverse outcomes. The findings are consistent with those of Andersson et al who found a correlation between higher prehospital lactate levels and higher 30-day mortality in suspected sepsis. They also advocated the use of lactate as an additional component to early risk stratification to better detect patients with higher risk of adverse events.[25] Likewise, Lu et al in a systematic review and meta-analysis of 41,469 patients, showed that lactate and clinical scoring systems were important parameters to use when assessing prognosis in patients with sepsis, suggesting that multiple clinical parameters should be used when assessing prognosis.[26]

The relevance of lactate in clinical practice was again confirmed by the systematic review and meta-analysis conducted by Li et al in 34 studies with 46,300 patients, which showed that higher serum lactate levels were correlated with higher mortality rates in patients with sepsis. The authors pointed out, however, that lactate is a poor single variable to use for predicting mortality and suggested that it be used in conjunction with other clinical factors.[27] These results are in line with the results of the present study, with lactate having the largest difference between survivors and non-survivors and having a higher correlation with length of hospital stay than SI and MSI.

Besides lactate levels at admission, Bhat et al showed that lactate clearance was a risk factor for mortality in ED sepsis patients with severe sepsis.[28] The results indicate that serial lactate measurements can offer further information about treatment response and the dynamics of clinical condition. However, the current study only looked at the admission lactate level, and serial lactate levels and clearance were not assessed.

The results of the present study are also similar to Wang et al., who studied 187 ED patients with sepsis and found a mortality rate similar to that of patients (19.8% vs. 19.0%). Their study showed that there was higher lactate, lower SI, and lower MSI in non-survivors. In addition, in the adjusted analysis, MSI ≥1.3 and lactate >2.0 mmol/L were both independent risk factors for mortality.[29] Wang et al reported an area under the curve (AUC) of 0.77 for SI and 0.79 for MSI, which indicates moderate discriminatory performance. In the present study, however, the study did not use ROC analysis; hence, discriminatory power and optimum cut-off values of these parameters were not determined.

Lopes et al investigated 365 ED patients with shock (with most of them having septic shock) and found a higher mortality compared to the present study. Like these results,

the study found that there was a significant difference between the scores for SI and MSI for survivors and non-survivors.[30] They also found that they had better mortality discrimination with MSI than SI, especially for patients with septic shock, with an AUC of 0.700 for MSI. This is in agreement with the slightly higher correlation between MSI and the severity of sepsis that the study found. The performance of these indices may vary by patient, disease severity or other factors; the values of these cut-offs may not be directly applied to other groups of patients without validation.

In a tertiary care ED in North India, Uppal et al have studied patients with Sepsis-3 and found that increased MSI was correlated with poor clinical outcomes, such as death and admission to the ICU.[31] Their mean MSI was similar to that in the present study (1.41 vs. 1.40). They had a higher mortality and higher rates of admission to critical care than the cohort. However, they also found that the discriminatory ability of MSI was better than qSOFA (AUC 0.946). The disparate findings in the studies could be related to sample size, patient characteristics, disease severity and study setting. Hence, the best level of MSI should be determined for each population and needs to be confirmed.

From a clinical perspective, SI and MSI are simple bedside parameters calculated using routinely measured heart rate and blood pressure. They can help in the early identification of patients with haemodynamic instability, especially in emergency settings where laboratory results are likely to be delayed. Lactate levels in the blood can give some additional information about tissue hypoperfusion and changes in metabolism. Hence, the combined assessment of these parameters could assist in early clinical assessment and monitoring and management decisions. However, they should be read and understood in relation to the patient's clinical context and not as stand-alone decision makers.

Overall, the present study shows that increased admission SI, MSI and serum lactate level are associated with increased severity of sepsis and poor clinical outcomes such as in-hospital mortality, admission to an intensive care unit, requirement for vasopressors and extended hospital stays. SI and MSI give easily available haemodynamic information while lactate give complementary metabolic information. They need to be tested in prospective studies in larger and more heterogeneous, numbers of patients with appropriate cut-off values, including validation and ROC analysis to determine their individual and combined prognostic performance.

Clinical implications of the findings

SI and MSI only measure heart rate and blood pressure, so they are free, instant and repeatable. They can identify patients with a high risk of being a hit at triage before lab results are received. Septic shock, mortality, ICU admission, and use of vasopressors were all related to high SI, MSI and lactate. These patients might require early senior review, intensive resuscitation and early admission to the ICU. Each marker provides additional information, as lactate was only moderately correlated with SI and MSI. It is also advisable to measure lactate, if possible, whenever it is measured.

MSI might be better than SI when using MAP because it had slightly better associations with severity and outcomes. Low values might allow forward-level care that is repeated and assessed again. These markers are intended to complement and not supplant clinical judgment.

Strengths

Structured data collection form consecutive recruitment of 200 patients. The same cohort of patients, with sepsis-3 criteria, was used for comparing SI, MSI and lactate. Multiple outcomes were measured: severity, hospitalization to the intensive care unit (ICU), use of vasopressors and mortality and length of stay.

Limitations

Limited generalisability due to single centre design, with a small number of children. There were no reports of ROC-AUC, sensitivity, specificity or cut-offs, and individual-level predictive accuracy remains unknown. No adjustment was made for multiple variables like age, any other medical condition, or treatment. Markers were only measured once at presentation, and only in-hospital mortality was assessed.

CONCLUSION

Increased Shock Index, MSI and serum lactate were independently found to be significantly associated with increased sepsis severity and clinical adverse events such as in-hospital mortality, ICU admission and vasopressor requirement in the present study. These findings support the potential use of SI and MSI as easily accessible bedside variables and serum lactate as a secondary variable to give additional information about tissue hypoperfusion and metabolic disturbance. Together, their evaluation may be useful in early clinical evaluation and risk stratification of sepsis patients. They need further validation to confirm their prognostic role and to determine their clinical cut-off values.

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