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EVALUATION OF MODIFIED SHOCK INDEX AS A PROGNOSTIC MARKER OF SEPTIC SHOCK AND SEVERE SEPSIS: A CROSS-SECTIONAL ANALYTICAL STUDY

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ABSTRACT Sepsis is a life-threatening event and early recognition and intervention is important. The development of cost-effective and easily attainable clinical parameters that would effectively prognosticate the outcome of sepsis patients would be invaluable within an emergency department setting. This study aimed to assess the utility of modified shock index (MSI) as a predictor for sepsis and septic shock, and evaluation of MSI as a prognostic marker of sepsis severity and septic shock. This is a cross-sectional analytical study conducted with total of 100 patients diagnosed with sepsis and septic shock. The MSI was calculated using the mean arterial pressure (MAP) and heart rate (HR) at arrival. Results depicted that non-survivor group had significantly higher MSI values (1.33 [interquartile range (IQR): 1.10-1.80] vs. 0.95 [IQR: 0.77-1.20]; p<0.001) and serum lactate levels (4.1 [IQR: 2.8-6.0] vs. 2.2 [IQR: 1.2-3.5] mmol/L; p<0.001) compared to survivor group >1.2 had a sensitivity of 74.3% and specificity of 83.1% for predicting mortality (AUC: 0.84, 95% CI: 0.76-0.92). The combination of MSI and serum lactate showed the highest predictive ability, with a sensitivity of 82.9%, specificity of 89.2%, and an AUC of 0.92 (95% CI: 0.87-0.97). In conclusion, the MSI is a valuable tool for assessing the prognosis of patients with severe sepsis and septic shock. Its incorporation into clinical practice, along with other parameters such as serum lactate may improve risk stratification and guide management strategies. Hence, MSI could be recommended to use as bedside prognostic marker for predicting the severity of sepsis and septic shock.

KEYWORDS : Sepsis, Septic shock, MSI, Prognostic marker, Serum lactate

INTRODUCTION

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. Sepsis and septic shock are major health care problems, affecting millions of people around the world each year and killing as many as one in four.[1] The Global Burden of Disease study estimated that there were 48.9 million sepsis cases worldwide in 2017, which led to 11.0 million sepsis related deaths (19.7% of all global deaths) that same year.[2] The highest burden of sepsis was noted in low- and middle-income countries and represented 85% of all sepsis-related deaths worldwide. The sepsis cases in India alone were estimated to be 11.3 million, with 2.9 million deaths (297.7 per 100,000 population) in 2017.[3]

Early recognition of sepsis and the initiation of appropriate management including antibiotics and supportive care are paramount to improving survival rates. However, the heterogeneity of sepsis manifestations and the lack of universally accepted prognostic markers complicate the early identification of patients at high risk of deterioration.[4,5] Currently for emergency physicians or other healthcare providers responsible for initial management have limited modalities to risk-stratify patients at risk for short-term cardiovascular collapse and escalation of disease.[6]

Shock index was first described by Allgower and Bwriin 1967, and it is defined as heart rate divided by systolic blood pressure. Normal range being 0.5 to 0.7 in healthy adults.[7] It has been reported to be a sensitive marker for shock and for success of resuscitation efforts than the standard vital signs alone and has the advantage of not requiring invasive monitoring.[8] A shock index elevation greater than 0.8 has a reported 95% sensitivity for predicting shock.[9] It is an easily accessible, non-invasive, and non-costly risk stratification tool that may enhance current emergency physicians' methods for differentiating severe sepsis patients at risk for imminent cardiovascular collapse.[6]

The Modified Shock Index (MSI) calculated as the ratio of heart rate to mean arterial pressure, has emerged as a potentially superior metric for assessing the severity of illness in patients with sepsis and septic shock. The rationale behind the MSI is to provide a more sensitive and specific measure of circulatory failure, which is a central feature of sepsis pathophysiology. The prognostic value of the MSI in sepsis has been the subject of recent research with studies suggesting that it may outperform traditional measures of shock in predicting mortality, ICU admission, and the need for mechanical ventilation and vasopressor support.[10-12] Previous studies have highlighted the prognostic value of the MSI in various clinical settings, including trauma and acute heart failure, suggesting its broad applicability.[4,5] Moreover, recent investigations into the utility of the MSI in sepsis have shown promising results.[13,14]

However, application of the MSI in clinical practice is challenging because of dynamic nature of sepsis, characterized by rapid changes in physiological parameters, necessitates continuous monitoring and reevaluation of prognostic markers. In addition, the influence of underlying comorbidities, age, and the cause of sepsis on the MSI's predictive value warrants further investigation. With this background, the present study was designed with main objectives to assess the utility of MSI as a predictor for severe sepsis and septic shock, and evaluation of MSI as a prognostic marker of sepsis severity and septic shock.

METHODOLOGY

Study Design And Patients

This is a cross-sectional analytical study conducted with total of 100 patients diagnosed with sepsis and septic shock at Department of General Medicine, Bapuji hospital and Chigateri hospital attached to J. J. M. Medical College, Davangere, Karnataka. The ethical committee approval was obtained before the conduct of study.

Inclusion Criteria

1. Age: ≥ 17 years
2. Patients fulfilling requirements at least two of the four Systemic Inflammatory Response Syndrome (SIRS) criteria for either severe sepsis or septic shock
3. Who are ready to sign informed consent form

Exclusion Criteria

1. Age: <17 years old
2. Sepsis patients with low blood pressure that were started on inotropes immediately (< 2 hours before arrival to the emergency department)
3. Taking medications that have significant atrioventricular blockage effect *viz.* beta blockers, calcium channel blockers, digoxin, and amiodarone
4. End-stage malignancies
5. Internal pacemakers
6. Acute coronary syndrome
7. Atrial fibrillations
8. Upper gastrointestinal bleeding
9. Immuno-compromised states

Data Collection And Assessment Parameters

After obtaining informed consent, a detailed history and demographic data were collected from qualifying patients using a pre-designed structured proforma. The MSI was calculated using the mean arterial pressure and heart rate at arrival. Patient assessment was thorough, incorporating clinical evaluation and laboratory investigations including CBC, random blood sugar Levels (RBS), urine culture and sensitivity, blood culture and sensitivity.

Statistical Analysis

Data were entered in Microsoft Excel 2021 and statistical analysis was done using IBM Statistical Software for Social Sciences (SPSS) version 22. Categorical variables were represented in the form of percentages and frequencies. Continuous variables were presented as descriptive statistics (Mean and Standard deviation). Data were analyzed to ascertain the MSI's value in predicting mortality among patients with severe sepsis and septic shock. Due to the small sample size and the assumption of non-normality, median values along with minimum and maximum were used for numerical variables. Outcomes were categorized as death (negative outcome) or survival (positive outcome) to discharge. The Mann-Whitney U test was utilized for analyzing significant differences among clinical parameters. Sensitivity and specificity analyses were conducted to find the optimal cut-off point for predicting outcomes. The analysis included area under curve (AUC) with confident intervals (CI) for significant parameters and combinations thereof. $p \leq 0.05$ was considered statistically significant.

RESULTS

The mean age of the study subjects was significantly different between survivor and non-survivor group (p=0.002). The mean age of overall study subjects was found to be 58.20 and 54.70 & 65.10 years of survivor & non-survivor group respectively. Male predominance was observed in both survivor (58.50%) and non-survivor group (68.60%). There was no significant (p>0.05) difference between the groups was observed with regards to distribution of comorbid conditions like diabetes and hypertension (Table 1).

Table 1. Distribution Of Study Subjects Based On Demographic And Baseline Characteristics

Variables	Overall (n=100)	Survivors (n=65)	Non-Survivors (n=35)	p-value
Age (years)	58.20 ± 16.40	54.70 ± 15.80	65.10 ± 15.30	0.002
Gender				
Male, n (%)	62 (62.00)	38 (58.50)	24 (68.60)	0.319
Female, n (%)	38 (38.00)	27 (41.50)	11 (31.40)	
Comorbidities				
Diabetes, n (%)	32 (32.00)	18 (27.70)	14 (40.00)	0.212
Hypertension, n (%)	45 (45.00)	26 (40.00)	19 (54.30)	0.167

Values are expressed as mean ± standard deviation (SD) unless otherwise stated

The results of distribution study subjects based on SIRS criterions was represented in Table 2. Results depicted that heart rate (p=0.001), respiratory rate (p<0.001), WBC count (p=0.008) were significantly different between survivor and non-survivor groups. Whereas, the immature neutrophil band form was non-significantly (p=0.061) different between survivor and non-survivor groups.

Table 2. Distribution Of Study Subjects Based On SIRS Criterions

SIRS Criterions	Overall (n=100)	Survivors (n=65)	Non-Survivors (n=35)	p-value
<36°C, n (%)	18 (18.00)	9 (13.80)	9 (25.70)	0.135
36-38°C, n (%)	52 (52.00)	38 (58.50)	14 (40.00)	
>38°C, n (%)	30 (30.00)	18 (27.70)	12 (34.30)	
Heart rate, bpm	110 (95-125)	105 (92-120)	120 (105-135)	0.001
Respiratory rate, breaths/min	24 (20-28)	22 (18-26)	28 (24-32)	<0.001
WBC count ($x10^9$/L)	14.20 (9.80-19.50)	13.50 (9.20-18.10)	16.8 (11.40-22.30)	0.008
Immature neutrophil band forms, n (%)	65 (65.00)	38 (58.50)	27 (77.10)	0.061

Values are expressed as median (IQR) unless otherwise stated

SIRS, Systemic Inflammatory Response Syndrome; IQR, Interquartile range

The results of distribution study subjects based on haemodynamic parameters was represented in Table 3. Results implied that all the haemodynamic parameters *viz.* MAP, SBP, DBP, and MSI were significantly (p<0.001) different between survivor and non-survivor groups. The non-survivors had significantly (p<0.001) lower MAP (63.80 ± 14.70) compared to survivors (77.20 ± 12.60). The median SBP and DBP were also significantly lower in non-survivors at 90mmHg and 50mmHg, respectively, compared to survivors at 110mmHg and 65mmHg, respectively (p<0.001 for both). The median MSI was significantly (p<0.001) higher in non-survivors at 1.33 compared to survivors at 0.95.

Table 3. Distribution Of Study Subjects Based On Haemodynamic Parameters

Haemodynamic parameters	Overall (n=100)	Survivors (n=65)	Non-Survivors (n=35)	p-value
MAP, mmHg, mean ± SD	72.40 ± 14.80	77.20 ± 12.60	63.80 ± 14.70	<0.001
SBP, mmHg	100 (90-120)	110 (100-130)	90 (80-100)	<0.001
DBP, mmHg	60 (50-70)	65 (60-75)	50 (40-60)	<0.001
MSI	1.10 (0.83-1.50)	0.95 (0.77-1.20)	1.33 (1.10-1.80)	<0.001

Values are expressed as median (IQR) unless otherwise stated

SD. Standard deviation; MAP, Mean arterial pressure; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; MSI, Modified shock index; IQR, Interquartile range

There statistically significant (p<0.05) difference was observed between survivor and non-survivor groups with regards to all the sepsis severity parameters *viz.* end-organ damage, serum lactate, hypertension, adequate fluid resuscitation, severe sepsis, and septic shock. The non-survivors had a significantly (p=0.002) higher proportion of patients with regards to evidence of end-organ damage (68.60%) compared to survivors (36.90%). The median serum lactate level was also significantly (p<0.001) higher in non-survivors at 4.10 mmol/L compared to survivors at 2.20 mmol/L. Furthermore, incidences of septic shock were significantly (p<0.001) higher in non-survivors (57.10%) compared to survivors at (23.10%) (Table 4).

Table 4. Distribution Of Study Subjects Based On Sepsis Severity Parameters

Sepsis severity parameters	Overall (n=100)	Survivors (n=65)	Non-Survivors (n=35)	p-value
Evidence of end-organ damage, n(%)	48 (48.00)	24 (36.90)	24 (68.60)	0.002
Serum lactate, mmol/L	2.80 (1.50-4.50)	2.20 (1.20-3.50)	4.10 (2.80-6.00)	<0.001
Presence of hypotension, n(%)	62 (62.00)	32 (49.20)	30 (85.70)	<0.001
Adequate fluid resuscitation, n(%)	38 (38.00)	33 (50.80)	5 (14.30)	<0.001
Severe sepsis, n(%)	65 (65.00)	50 (76.90)	15 (42.90)	<0.001
Septic shock, n(%)	35 (35.00)	15 (23.10)	20 (57.10)	<0.001

Values are expressed as median (IQR) unless otherwise stated
IQR, Interquartile range

The results of predictive ability of MSI and other parameters *viz.* serum lactate, MAP, and combined MSI and serum lactate were represented in Table 5. Results depicted that the MSI showed good predictive ability for mortality with an optimal cut-off value of >1.2, achieving a sensitivity of 74.30% and specificity of 83.10%. The area under the curve (AUC) for MSI was 0.84 (Figure 1). Serum lactate levels showed cut-off value of >3.5 mmol/L, and had a sensitivity of 71.4% and specificity of 80.0% with an AUC of 0.81. Furthermore, the combination of MSI and serum lactate showed the highest predictive ability for mortality. These findings suggest that MSI, along with other clinical and laboratory parameters could be useful in assessing the prognosis of patients with severe sepsis and septic shock.

Table 5. Predictive Ability Of MSI And Other Parameters

Parameters	Cut-off	Sensitivity (%)	Specificity (%)	AUC (95% CI)
MSI	>1.20	74.30	83.10	0.84 (0.76-0.92)
Serum lactate, mmol/L	>3.50	71.40	80.00	0.81 (0.72-0.90)
MAP, mmHg	<70.00	77.10	75.40	0.79 (0.70-0.88)

Combined MSI and Serum lactate	-	82.90	89.20	0.92 (0.87-0.90)

MSI, Modified shock index; MAP, Mean arterial pressure; AUC, Area under curve; CI, Confidence interval

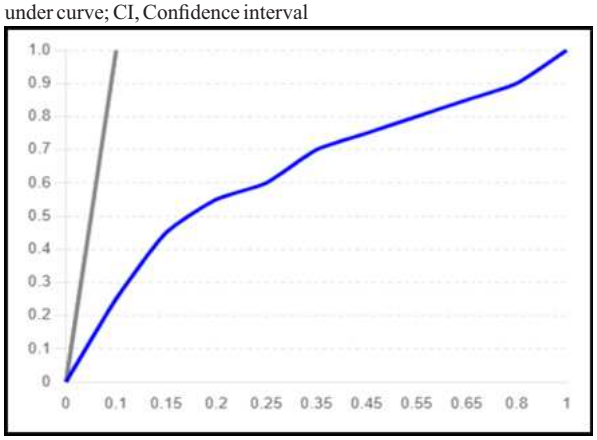

Figure 1. ROC Curve For Modified Shock Index (MSI)

DISCUSSION

Sepsis is a life-threatening event and early recognition and intervention is important. Simple bedside tools at the emergency department like shock index helps to identify the risk of clinical deterioration. Clinicians evaluate the heart rate and systolic blood pressure on every patient but rarely analyze them in a unified way. This can be easily accomplished by calculating a shock index. The management of sepsis is closely related to the availability of relevant equipment and efficacy of clinical and serological indices, which is used as a guide for the prognostication and effective treatment goals. The development of cost effective and easily attainable clinical parameters that would effectively prognosticate the outcome of sepsis patients would be invaluable within an emergency department setting.[15]

Moreover, recent studies have examined the prognostic value of the MSI in sepsis and found that it may be more accurate than conventional measures of shock in predicting mortality, ICU admission, and the need for vasopressor support and mechanical ventilation.[10-12] Hence, in this cross-sectional analytical study we aimed to assess the utility of MSI as a predictor for sepsis and septic shock, and evaluation of MSI as a prognostic marker of sepsis severity and septic shock.

The results of our study revealed that the mean age of survivor and non-survivor group was found to be 54.70 and 65.10 years respectively. The age distribution between survivor and non-survivor groups statically significant. Male predominance was observed in both survivor (58.50%) and non-survivor group (68.60%). These findings were comparable with the previous studies reported in the literature, that have reported age as a risk factor for mortality in sepsis and septic shock. Martin et al., reported patients aged 65 years or older had a significantly higher mortality rate compared to younger patients (38.4% vs. 27.4%, p<0.001).[16] Fleischmann et al., reported in a meta-analysis study that older age was associated with increased mortality in sepsis (OR: 1.03 per year increase).[17]

The results on distribution study subjects based on SIRS criterions in our study found that there were significant differences in heart rate, respiratory rate, and WBC count between survivor and non-survivor groups. These findings were in accordance with findings of Kaukonen et al., wherein researchers have reported that higher heart rates (>90 bpm) and respiratory rates (>20 breaths/min) were associated with increased mortality. The elevated heart rate and respiratory rate in non-survivors may reflect the severity of the septic process and the body's attempt to compensate for the hemodynamic and metabolic derangements. Tachycardia and tachypnea are common manifestations of the systemic inflammatory response and may be driven by the release of cytokines, endogenous catecholamines, and other mediators. Moreover, these vital sign alterations may indicate the presence of underlying organ dysfunction, such as cardiovascular compromise or respiratory failure, which are associated with worse outcomes in sepsis.[18]

In our study there were significant differences in all the haemodynamic parameters *viz.* MAP, SBP, DBP, and MSI between survivor and non-survivor groups. The median MSI was significantly (p<0.001) higher in non-survivors (1.33) compared to survivors (0.95). In concurrence with our study findings Jayaprakash et al., that MSI >1.3 was associated with increased mortality (OR: 2.96; p=0.004).[19] In another study, Wira et al., demonstrated that MSI >1.4 was a predictor of mortality (OR 2.7; p=0.016).[6] These findings implied that the MSI which incorporates both heart rate and MAP provides a more comprehensive assessment of the hemodynamic status compared to individual vital signs. A higher MSI indicates a greater degree of shock and may suggest the need for more aggressive resuscitation and vasopressor support.

In our study non-survivors had a significantly (p=0.002) higher proportion of patients with evidence of end-organ damage (68.60%) compared to survivors (36.90%). The median serum lactate level was also significantly (p<0.001) higher in non-survivors (4.10 mmol/L) compared to survivors (2.20 mmol/L). Furthermore, incidences of septic shock were significantly (p<0.001) higher in non-survivors (57.10%) compared to survivors at (23.10%). These findings were in consistence with various other research studies reported in the literature. In a study by Mikkelsen et al., demonstrated that serum lactate levels >4 mmol/L were associated with increased mortality (OR: 4.87; p=0.015).[20] In another meta-analysis study conducted by Suetrong et al., also established that that elevated serum lactate levels were associated with increased mortality in sepsis with odds ratio of 2.66.[21]

In our study, MSI showed good predictive ability for mortality with an optimal cut-off value of >1.2, achieving a sensitivity of 74.30% and specificity of 83.10% with 0.84 AUC. Furthermore, the combination of MSI and serum lactate also showed the highest predictive ability for mortality. These findings suggest that MSI along with other clinical and laboratory parameters could be useful in assessing the prognosis of patients with severe sepsis and septic shock. These findings were comparable with the literature findings reported by various other research investigators. Gupta et al., reported AUC of 0.73 for MSI.[22] Similarly Zhou et al., reported 0.64 AUC for MSI.[23] These discrepancies in the performance of MSI across studies may be ascribed to differences in study populations, sample sizes, and the definition of outcomes.

This cross-sectional analytical study has several limitations. First, the single-center design and relatively small sample size may limit the generalizability of the study outcomes. Second, the study did not account for potential confounding factors such as comorbidities, severity of underlying infections, and timing of interventions, which may have influenced the study outcomes. Third, the study did not evaluate the performance of MSI in comparison to other widely used severity scores such as APACHE II, SOFA, and qSOFA. Hence, future studies should address these limitations by conducting prospective, multicenter studies with larger sample sizes and by adjusting for potential confounders.

CONCLUSION

The results of the current study clearly established that MSI is a valuable tool in assessing the prognosis of patients with severe sepsis and septic shock. MSI, along with other clinical and laboratory parameters such as serum lactate could help identify patients at higher risk of mortality. The combination of MSI and serum lactate showed the highest predictive ability for mortality in this study. Hence, MSI could be recommended to use as a bedside prognostic marker for predicting the severity of sepsis and septic shock.

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