ORIGINAL ARTICLE

Risk Factors for Septic Shock in Adult Patients: Evidence from a General Hospital in Southern Thailand

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Background: Sepsis and septic shock remain major global health concerns, characterized by dysregulated host responses to infection leading to organ failure and high mortality. In Thailand, the incidence is increasing, however, evidence regarding specific risk factors for septic shock in general hospital settings remains limited.

Objective: To identify independent risk factors for septic shock among adult patients with sepsis at Sichon Hospital and to describe associated clinical characteristics and outcomes.

Materials and Methods: A retrospective cohort study was conducted using medical records of adult patients diagnosed with sepsis or septic shock at Sichon Hospital between January and December 2023. Eligible cases were identified through ICD-10 coding and screened according to predefined inclusion and exclusion criteria. Demographic, clinical, and laboratory data were analyzed to determine predictors of septic shock using multivariable logistic regression.

Results: Of the 584 screened patients, 484 met the inclusion criteria and 146, or 30.2%, developed septic shock. Multivariable logistic regression identified six independent predictors of septic shock, which are body temperature of less than 37.5°C (OR 2.83, 95% CI 1.70 to 4.69), respiratory rate greater than 22 per minute (OR 1.79, 95% CI 1.10 to 2.92), activated partial thromboplastin time (aPTT) greater than 29.9 seconds (OR 2.06, 95% CI 1.27 to 3.36), creatinine level greater than 1.25 mg/dL (OR 2.30, 95% CI 1.39 to 3.79), albumin of less than 3.7 g/dL (OR 2.26, 95% CI 1.37 to 3.72), and bilirubin greater than 0.92 mg/dL (OR 2.51, 95% CI 1.51 to 4.18). Hypertension (OR 0.54, 95% CI 0.32 to 0.91) and positive hemoculture (OR 0.46, 95% CI 0.28 to 0.76) were inversely associated. The model demonstrated good discrimination (AUC 0.804).

Conclusion: About one-third of septic patients developed shock. Six readily available parameters, body temperature, respiratory rate, serum creatinine, aPTT, albumin, and total bilirubin, independently predicted septic shock, indicating early multi-organ dysfunction. In contrast, hypertension and positive blood cultures were inversely associated, reflecting earlier recognition and source control. Routine, low-cost clinical and laboratory data may thus support early risk stratification and timely management, especially in resource-limited settings.

Keywords: Sepsis; Septic shock; Risk factors

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Sepsis is a critical medical emergency that requires prompt and effective intervention. It arises when the immune system overreacts to an infection, often culminating in the failure of essential organ systems. Septic shock represents a more severe progression of sepsis, characterized by profound circulatory, cellular, and metabolic abnormalities. It can be diagnosed clinically using criteria defined by the necessity of vasopressor support to sustain a mean arterial pressure above 65 mmHg, in conjunction with elevated serum lactate levels exceeding 2 mmol/L despite sufficient fluid resuscitation[1]. In addition, patients with septic shock may exhibit abnormal signs and laboratory findings, which are a consequence of decreased oxygen delivery to tissues. Such abnormalities include arterial hypoxemia, acute oliguria, increased creatinine levels, coagulation abnormalities, thrombocytopenia, hyperbilirubinemia, and others[2].

Globally, sepsis affected an estimated 49 million individuals in 2017, resulting in 11 million fatalities[3]. Within Thailand, the Health Data Center of the Ministry of Public Health reported that in fiscal years 2021, 2022, and 2023, the number of patients with severe sepsis (ICD-10 code R65.1) and septic shock (ICD-10 code R57.2) due to community-acquired infections were 72,647, 79,088, and 90,178 cases,

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respectively. The corresponding mortality rates were 34.09%, 35.35%, and 29.73%[4].

Research conducted in tertiary hospitals in Thailand has highlighted the severity of septic shock. A study at Songklanagarind Hospital in 2009 found that the mortality rate among patients diagnosed with septic shock and admitted to the intensive care unit (ICU) was 54.1%[5]. Similarly, a 2019 study at Siriraj Hospital reported that 30.5% of patients diagnosed with sepsis progressed to septic shock, with a corresponding mortality rate of 45.1% among those who developed shock[6].

Sichon Hospital, a large general hospital located in Nakhon Si Thammarat Province, a major province in southern Thailand, has 400 beds, including 14 ICU beds, and recognizes sepsis as one of its top three major health challenges. Data collected from fiscal years 2021 and 2022 showed that the hospital treated 229 and 184 patients with severe community-acquired sepsis, respectively, with mortality rates of 17.09% and 26.63%[4]. In response to these figures, the Ministry of Public Health had set a goal to reduce the mortality rate to below 26%.

Over the past 16 years, global studies have identified risk factors for septic shock. A 2008 study in Türkiye found advanced age, female gender, lymphopenia, and hyperglycemia increased the risk of septic shock in patients with ventilator-associated pneumonia[7]. A 2011 South Korean study on bacteremic acute pyelonephritis revealed gender, cirrhosis, platelet count, albumin levels, acute kidney failure, and healthcare-associated infections as risk factors[8]. A 2015 Taiwanese study on urinary tract infections highlighted age, coronary artery disease (CAD), heart failure, acute kidney failure, and stage 3 chronic kidney disease[9]. Lastly, a 2020 South Korean study on obstructive urolithiasis identified the absence of hypertension, low platelet count, low erythrocyte sedimentation rate (ESR), high blood urea nitrogen (BUN), and positive blood cultures as predictive factors[10].

The studies mentioned above have identified various risk factors and predictive indicators for the development of septic shock, with these varied factors depending on the patient population studied. In Thailand, however, there is limited research on this topic. Consequently, investigating the risk factors associated with septic shock in patients at Sichon Hospital could provide valuable insights. The present research would enable medical personnel to identify high-risk patients and closely monitor them for the development of septic shock, potentially reducing the incidence of organ dysfunction and mortality rates. Moreover, the findings from the present study could serve as the foundation for creating clinical tools and developing treatment guidelines for sepsis at Sichon Hospital.

Therefore, the objective of the present study was to identify the risk factors associated with septic shock in adult patients admitted to Sichon Hospital, and to analyze their clinical characteristics, treatment outcomes, length of hospital stay, and mortality rates.

Materials and Methods

Study design and study population

The present study was a retrospective cohort study, which included adult patients aged 18 years and older who were treated at Sichon Hospital, Nakhon Si Thammarat Province, between January 1 and December 31, 2023. Eligible patients were identified using a diagnosis coded according to the International Classification of Diseases (ICD-10) codes A40.0-A41.9, which is all bacterial infections except melioidosis, R65.1, which is severe sepsis, R57.2, which is septic shock, A24.0-A24.4, which is melioidosis, and A27.0-A27.9, which is leptospirosis, listed as primary diagnoses, co-diagnoses, or complications. Case selection was performed based on inclusion and exclusion criteria for sample selection. Patients whose data could not be retrieved from medical records, those who were pregnant, or those whose medical records did not meet the diagnostic criteria for sepsis or septic shock were excluded.

Patient details potentially related to the development of septic shock, including gender, age, underlying conditions, infection type and site, acute respiratory failure, acute kidney failure, laboratory results at the time of diagnosis, blood culture results, as well as additional information such as weight, ward location, length of hospital stay, and discharge status were retrieved from the medical records.

The present study was approved by the Human Research Ethics Committee, Public Health Office, Nakhon Si Thammarat Province (93/2567), prior to data collection. Additionally, formal permission was requested from the director of Sichon Hospital and relevant stakeholders for permission to use medical records. As a retrospective observational study, it posed no risk to patients, and data were collected exclusively from medical records.

Sample size

A 2008 study in Türkiye on patients with

ventilator-associated pneumonia identified gender as a significant risk factor for septic shock[7]. These data were used to calculate the sample size using n4Studies software, specifically the module for testing two independent population proportions, which determined that each group should consist of 89 participants, for a total of 178 participants. To accommodate for potential data loss, the sample size was increased by 20%, adding 18 participants per group, resulting in 107 participants per group. Accordingly, the total sample size for the present study was 214 participants.

Definitions and diagnostic criteria

Sepsis was diagnosed with a suspected or confirmed infection and systemic inflammatory response syndrome (SIRS), indicated by two or more of the following: body temperature greater than 38°C or less than 36°C, heart rate greater than 90 bpm, respiratory rate greater than 24 breaths/minute or $PaCO_2$ of less than 32 mmHg, and white blood cell (WBC) count outside 4,000 to 12,000 cells/mm^3 or band forms greater than 10%. A positive blood culture was required, except in immunocompromised patients with no identifiable infection source. Treatment with appropriate antibiotics for five to seven days and clinical improvement were needed for diagnosis unless the patient died or was transferred[11]. Community-acquired sepsis arises from infections contracted outside healthcare settings, while hospital-acquired or healthcare-associated sepsis came from infections acquired during hospitalization or healthcare-related events within three months[12]. Septic shock was defined as requiring vasopressors to maintain mean arterial pressure of 65 millimeters of mercury (mmHg) or greater and lactate of greater than 2 mmol/L, despite adequate fluid resuscitation[1].

Acute respiratory failure was defined by altered mental status, cyanosis, respiratory distress, PaO_2 of less than 55 mmHg or $PaCO_2$ greater than 45 mmHg, or SpO_2 of less than 90% if arterial blood gas is unavailable, and the need for invasive ventilation or ambu bag support[11]. Acute kidney injury (AKI) was defined by a serum creatinine increased greater than 0.3 mg/dL within 48 hours, a 1.5x increase in one week, or urine output of less than 0.5 mL/kg/hour for more than six hours[13]. Chronic diseases, including diabetes, hypertension, dyslipidemia, and chronic obstructive pulmonary disease (COPD), were those with documented diagnoses and treatment. Chronic kidney disease (CKD) was defined as abnormal kidney function lasting more than three months, confirmed by albuminuria, hematuria, electrolyte abnormalities, radiological changes, or an estimated glomerular filtration rate (eGFR) of less than 60 mL/minute/1.73 m^2[14]. Obesity was defined as body mass index (BMI) of 25 kg/m^2 or more[15]. Myocardial infarction (MI) was confirmed by electrocardiogram (ECG) showing Q waves or regional wall motion abnormalities[11]. Cirrhosis was diagnosed based on clinical findings (e.g., jaundice, splenomegaly, ascites) and confirmed by liver function tests or radiological evidence[11].

Statistical analysis

Categorical variables were analyzed using the chi-square test, while continuous data were assessed using the Wilcoxon rank-sum test. A predictive model for shock in patients with sepsis was constructed using a backward, stepwise manual multivariable logistic regression analysis, including variables from univariate analysis with a p-value of less than 0.2. The model's discriminatory performance was assessed using the AUROC metric. Data was analyzed using Stata Statistical Software, version 15.1 (StataCorp LLC, College Station, TX, USA).

Results

Using the ICD-10 code, 584 patients were initially identified. After applying the exclusion criteria, 100 patients were excluded from the analysis. The reasons for exclusion were 55 patients did not meet the criteria for sepsis or septic shock, three patients were pregnant, three patients had diagnosed unrelated to infections, twenty patients did not undergo blood culture testing, eleven patients had missing medical records, and eight patients who experienced cardiac arrest were excluded due to insufficient documentation regarding whether the arrest was attributable to sepsis or other etiologies. In addition, laboratory values obtained after cardiac arrest were excluded, as they could potentially distort study outcomes due to extreme values introducing bias. Consequently, 484 patients were included in the final analysis.

Among the 484 patients, 146 (30.17%) presented with shock. There were no significant differences in age at 66 versus 65.5 years (p=0.855), gender distribution (p=0.138), weight (p=0.361), or BMI (p=0.400) between the two groups (Table 1).

Regarding baseline vital signs, patients with shock exhibited significantly lower systolic at 93 versus 127.5 mmHg, diastolic at 58 versus 73 mmHg, and mean arterial pressure at 69 versus 90.83 mmHg,

Table 1. Comparison of demographic, clinical, and laboratory characteristics between patients with and without shock (n=484)

Variables		Total cases (n=484)	Without shock (n=338, 69.83%)	Shock (n=146, 30.17%)	p-value
Age (years); median (IQR)	484	66 (52.5 to 77)	66 (51 to 78)	65.5 (55 to 76)	0.855
Sex; n (%)	484				0.138
Female		237 (48.97)	173 (51.18)	64 (43.84)	
Male		247 (51.03)	165 (48.82)	82 (56.16)	
Weight (kg); median (IQR)	484	56.95 (49.5 to 67.75)	56.95 (50 to 68)	56.5 (48 to 65)	0.361
BMI (kg/m^2); median (IQR)	481	22.22 (19.53 to 26.04)	22.22 (19.71 to 26.30)	22.17 (19.15 to 25.39)	0.400
Vital signs; median (IQR)					
Systolic blood pressure (mmHg)	484	118 (96 to 142)	127.5 (108 to 150)	93 (81 to 116)	<0.001
Diastolic blood pressure (mmHg)	484	68 (56.5 to 84)	73 (61 to 87)	58 (47 to 69)	<0.001
Mean arterial pressure (mmHg)	484	85.67 (70.83 to 102.83)	90.83 (78.67 to 107.33)	69 (59 to 87.67)	<0.001
Body temperature (°C)	484	38.2 (37 to 39)	38.4 (37.4 to 39.1)	37.5 (36.7 to 38.6)	<0.001
Heart rate (bpm)	484	107 (94 to 120)	105.5 (94 to 118)	110 (92 to 121)	0.413
Respiratory rate (breaths/minute)	481	22 (20 to 24)	22 (20 to 24)	22 (20 to 28)	0.001
Lab investigations; median (IQR)					
WBC (cells/μL)	482	12,640 (8,700 to 16,610)	12,710 (9,100 to 16,470)	12,230 (7,220 to 17,870)	0.991
Lactate (mmol/L)	379	2.8 (1.8 to 4.6)	2.55 (1.6 to 3.5)	4 (2.4 to 6.6)	<0.001
Hb (g/dL)	482	11.25 (9.5 to 12.7)	11.5 (9.9 to 12.7)	10.6 (8.9 to 12.4)	0.002
Platelet (cells/μL)	482	231,000 (155,000 to 312,000)	237,000 (171,000 to 312,000)	202,000 (118,000 to 302,000)	0.007
PT (seconds)	347	12.7 (11.8 to 13.8)	12.3 (11.6 to 13.3)	13.75 (12.6 to 15.9)	<0.001
aPTT (seconds)	337	27.1 (24.8 to 30.5)	26.6 (24.45 to 29.2)	30.5 (26.1 to 34.9)	<0.001
BUN (mg/dL)	472	19 (13 to 32)	17 (13 to 27)	28 (16 to 45)	<0.001
Cr (mg/dL)	472	1 (0.73 to 1.6)	0.96 (0.7 to 1.32)	1.33 (0.9 to 2.23)	<0.001
HCO_3 (mEq/L)	472	23 (20 to 26)	24 (21 to 26)	21.8 (17.9 to 25.5)	<0.001
Albumin (g/dL)	415	3.7 (3.2 to 4.2)	3.9 (3.5 to 4.3)	3.3 (2.9 to 3.8)	<0.001
Total bilirubin (mg/dL)	411	0.73 (0.5 to 1.18)	0.71 (0.49 to 1.1)	0.84 (0.5 to 1.69)	0.016
Direct bilirubin (mg/dL)	411	0.32 (0.2 to 0.6)	0.3 (0.19 to 0.49)	0.42 (0.23 to 0.89)	<0.001
Underlying disease; n (%)	484				
Diabetes type II		121 (25.00)	98 (28.99)	23 (15.75)	0.002
Hypertension		190 (39.26)	144 (42.60)	46 (31.51)	0.022
Chronic kidney disease		86 (17.77)	65 (19.23)	21 (14.38)	0.200
COPD		38 (7.85)	25 (7.40)	13 (8.90)	0.571
Coronary artery disease		34 (7.02)	25 (7.40)	9 (6.16)	0.626
Cerebrovascular accident		55 (11.36)	44 (13.02)	11 (7.53)	0.081
Cirrhosis		18 (3.72)	10 (2.96)	8 (5.48)	0.179
HIV		16 (3.31)	12 (3.55)	4 (2.74)	0.647
Solid malignancy		49 (10.12)	34 (10.06)	15 (10.27)	0.943
Hematologic malignancy		6 (1.24)	4 (1.18)	2 (1.37)	0.865
Rheumatological disease		6 (1.24)	5 (1.48)	1 (0.68)	0.469
Steroid treatment		4 (0.83)	3 (0.89)	1 (0.68)	0.821
Splenectomy		1 (0.21)	0 (0.0)	1 (0.68)	0.128

IQR=interquartile range; BMI=body mass index; WBC=white blood cell; Hb=hemoglobin; PT=prothrombin time; aPTT=activated partial thromboplastin time; BUN=blood urea nitrogen; Cr=creatinine; COPD=chronic obstructive pulmonary disease

all p<0.001. Body temperature was also lower at 37.5°C versus 38.4°C (p<0.001), and respiratory rate was higher at 22 versus 22 breaths/minute (p=0.001). Heart rate did not differ significantly (p=0.413) (Table 1).

With respect to laboratory findings, shock was associated with higher lactate at 4.0 versus 2.55 mmol/L (p<0.001), BUN at 28 versus 17 mg/dL (p<0.001), creatinine (1.33 versus 0.96 mg/dL (p<0.001), total and direct bilirubin (p=0.016 and <0.001, respectively), and prolonged prothrombin time (PT) and activated partial thromboplastin time

(aPTT) (both p<0.001). Hemoglobin, platelet count, bicarbonate, and albumin were significantly lower in the shock group (all p<0.01). WBC counts did not differ (p=0.991) (Table 1).

Type II diabetes mellitus at 28.99% versus 15.75% (p=0.002) and hypertension at 42.60% versus 31.51% (p=0.022) were significantly more prevalent among patients without shock compared to those with shock. Other comorbidities, including CKD, COPD, CAD, cerebrovascular accident (CVA), cirrhosis, HIV, and malignancies showed no significant differences (Table 1).

Infections were community-acquired at 81.6%, with a non-significant difference between patients with and without shock at 76.7% versus 83.7% (p=0.067). The distribution of infection sources differed significantly between groups (p=0.002). Respiratory, at 32.9% versus 19.8%, and hepatobiliary infections at 8.9% versus 2.7% were more common in shock patients, while urinary tract infections were more frequent in the non-shock group at 22.5% versus 15.8% (Table 2).

Of the 484 patients, 207 (42.8%) had positive blood cultures, with no significant difference between the shock and non-shock groups at 34.2% versus 46.4% (p=0.443). Gram-negative bacteria were the most common pathogens, led by *Escherichia coli* at 27.1%. Although not statistically significant, *Klebsiella pneumoniae* at 14.0% versus 6.4% (p=0.087) and *Acinetobacter baumannii* at 10.0% versus 4.5% (p=0.144) appeared more frequently in patients with shock.

Gram-positive organisms were also identified, particularly coagulase-negative Staphylococci (CoNS) at 17.9% and *Staphylococcus aureus* at 5.8%, with similar distribution between groups. However, the clinical significance of CoNS was limited, as most cases involved only a single positive culture, often without risk factors such as intravascular devices, immunosuppression, or positive repeat cultures, suggesting contamination rather than true bacteremia (Table 2).

In the total cohort of 484, patients with shock met the SIRS criterion for abnormal body temperature of more than 38°C or less than 36°C, less frequently than those without shock at 39.04% versus 63.02% (p<0.001). The proportion of patients meeting the heart rate criterion of more than 90 bpm was similar between groups at 76.03% versus 81.95% (p=0.134). In contrast, more patients with shock met the respiratory rate criterion of more than 20 breaths/minute compared to those without shock at 69.18% versus 59.76% (p=0.049). The frequency of abnormal WBC count was comparable between groups at 60.27% versus 58.88% (p=0.774) (Table 3).

SIRS score distributions differed significantly between groups (p=0.040). A greater proportion of shock patients had lower SIRS scores of 0 or 1, while non-shock patients were more likely to have higher scores of 3 or 4. Specifically, 12.33% of shock patients had a score of 1 compared to 4.73% in the non-shock group, while 34.93% of shock patients had a score of 3 versus 40.53% in the non-shock group (Table 3).

Significant differences were observed between patients with and without shock in terms of organ dysfunction and clinical interventions. Intubation was required in 52.7% of shock patients, compared to only 13.3% of non-shock patients (p<0.001). AKI was also more prevalent in the shock group at 54.8% versus 16.3% (p<0.001). However, there was no significant difference in the need for renal replacement therapy (RRT) between the groups with 0.7% in shock patients versus 0% in non-shock patients (p=0.128) (Table 4).

Lactate levels of 2 mmol/L or more, an indicator of tissue hypoperfusion, were significantly more common in shock patients at 78.8% versus 47.9% (p<0.001). Similarly, a platelet count of less than 100,000 cells/μL was more frequently observed in the shock group at 17.1% versus 4.7% (p<0.001). Shock patients were more likely to require ICU admission at 68.5% versus 10.1% (p<0.001) (Table 4).

In terms of clinical outcomes, shock patients had a significantly higher mortality rate at 30.8% versus 7.7% (p<0.001) and were less likely to be discharged home at 59.6% versus 89.4% (p<0.001). The proportion of patients referred to another hospital was higher in the shock group at 6.9% versus 1.8% (p=0.013). Length of hospital stay did not differ significantly between the two groups at eight days (p=0.513) (Table 4).

Multivariable logistic regression identified several independent predictors of septic shock in patients with sepsis (Table 5). Body temperature of less than 37.5°C (OR 2.83, 95% CI 1.70 to 4.69), respiratory rate of more than 22 breaths/minute (OR 1.79, 95% CI 1.10 to 2.92), aPTT of more than 29.9 seconds (OR 2.06, 95% CI 1.27 to 3.36), serum creatinine greater than 1.25 mg/dL (OR 2.30, 95% CI 1.39 to 3.79), albumin of less than 3.7 g/dL (OR 2.26, 95% CI 1.37 to 3.72), and total bilirubin of more than 0.92 mg/dL (OR 2.51, 95% CI 1.51 to 4.18) were each significantly associated with higher odds

Table 2. Type, source, and microbiological profile of infections in patients with and without shock (n=484)

Variables	Total cases (n=484) n (%)	Without shock (n=338, 69.83%) n (%)	Shock (n=146, 30.17%) n (%)	p-value
Type of infection				0.067
Community-acquired	395 (81.61)	283 (83.73)	112 (76.71)	
Hospital-acquired	89 (18.39)	55 (16.27)	34 (23.29)	
Source of infection				0.002
Bone and joint infection	4 (0.83)	3 (0.89)	1 (0.68)	0.821
Central nervous system infection	3 (0.62)	2 (0.59)	1 (0.68)	0.905
ENT infection	4 (0.83)	4 (1.18)	0 (0.00)	0.187
Gastrointestinal infection	37 (7.64)	24 (7.10)	13 (8.90)	0.493
Hepatobiliary infection	22 (4.55)	9 (2.66)	13 (8.90)	0.002
Respiratory infection	115 (23.76)	67 (19.82)	48 (32.88)	0.002
Skin infection	35 (7.23)	25 (7.40)	10 (6.85)	0.831
Tropical infection	16 (3.31)	13 (3.85)	3 (2.05)	0.312
Urinary tract infection	99 (20.45)	76 (22.49)	23 (15.75)	0.092
Unknown	108 (22.31)	79 (23.37)	29 (19.86)	0.395
Other	15 (3.10)	12 (3.55)	3 (2.05)	0.384
Primary bacteremia	26 (5.37)	24 (7.10)	2 (1.37)	0.010
Hemoculture	484			0.013
Positive	207 (42.77)	157 (46.4)	50 (34.25)	
Negative	277 (57.23)	181 (53.55)	96 (65.75)	
Pathogen	207	157 (75.85)	50 (24.15)	0.443
Gram-negative bacteria				
• *Acinetobacter baumannii*	12 (5.80)	7 (4.46)	5 (10.00)	0.144
• *Acinetobacter lwoffii*	3 (1.45)	3 (1.91)	0 (0.00)	0.325
• *Aeromonas* spp.	2 (0.97)	2 (1.27)	0 (0.00)	0.423
• *Burkholderia pseudomallei*	3 (1.45)	2 (1.27)	1 (2.00)	0.708
• *Escherichia coli*	56 (27.05)	47 (29.94)	9 (18.00)	0.098
• *Enterobacter cloacae*	3 (1.45)	2 (1.27)	1 (2.00)	0.708
• *Klebsiella pneumoniae*	17 (8.21)	10 (6.37)	7 (14.00)	0.087
• *Pseudomonas aeruginosa*	10 (4.83)	7 (4.46)	3 (6.00)	0.658
• Non-fermenting gram-negative bacilli	6 (2.90)	5 (3.18)	1 (2.00)	0.664
Gram-positive bacteria				
• *Staphylococcus aureus*	12 (5.80)	9 (5.73)	3 (6.00)	0.944
• Coagulase-negative Staphylococci	37 (17.87)	27 (17.20)	10 (20.00)	0.652
• *Staphylococcus epidermidis*	4 (1.93)	3 (1.91)	1 (2.00)	0.968
• *Staphylococcus haemolyticus*	2 (0.97)	2 (1.27)	0 (0.00)	0.423
• *Staphylococcus saprophyticus*	1 (0.48)	0 (0.00)	1 (2.00)	0.076
• *Stenotrophomonas maltophilia*	6 (2.90)	4 (2.55)	2 (4.00)	0.594
• *Streptococcus agalactiae*	4 (1.93)	4 (2.55)	0 (0.00)	0.254
• *Streptococcus dysgalactiae*	6 (2.90)	4 (2.55)	2 (4.00)	0.594
• *Streptococcus gallolyticus*	2 (0.97)	2 (1.27)	0 (0.00)	0.423
• *Streptococcus pneumoniae*	6 (2.90)	4 (2.55)	2 (4.00)	0.594
• *Streptococcus pyogenes*	8 (3.86)	7 (4.46)	1 (2.00)	0.432
• Other *Streptococcus* spp.	6 (2.90)	6 (3.82)	0 (0.00)	0.161
Other pathogens				
• *Vibrio cholerae*	1 (0.48)	0 (0.00)	1 (2.04)	0.076

of shock. In contrast, hypertension (OR 0.54, 95% CI 0.32 to 0.91) and positive hemoculture (OR 0.46, 95% CI 0.28 to 0.76) were associated with lower odds of shock. The model demonstrated good discriminative

Table 3. Systemic inflammatory response syndrome (SIRS) criteria and scores among patients with and without shock (n=484)

Variables	Total cases (n=484) n (%)	Without shock (n=338, 69.83%) n (%)	Shock (n=146, 30.17%) n (%)	p-value
SIRS criteria				
Body temperature <36°C or >38°C	270 (55.79)	213 (63.02)	57 (39.04)	<0.001
Heart rate >90/minute	388 (80.17)	277 (81.95)	111 (76.03)	0.134
Respiratory rate >20/minute	303 (62.60)	202 (59.76)	101 (69.18)	0.049
WBC count <4,000 or >12,000 cells/μL	287 (59.30)	199 (58.88)	88 (60.27)	0.774
SIRS score				0.040
0	7 (1.45)	4 (1.18)	3 (2.05)	
1	34 (7.02)	16 (4.73)	18 (12.33)	
2	185 (38.22)	130 (38.46)	55 (37.67)	
3	188 (38.84)	137 (40.53)	51 (34.93)	
4	70 (14.46)	51 (15.09)	19 (13.01)	

WBC=white blood cell

Table 4. Organ dysfunction, interventions, and clinical outcomes in patients with and without shock (n=484)

Variables	Total cases (n=484)	Without shock (n=338, 69.83%)	Shock (n=146, 30.17%)	p-value
Organ dysfunction; n (%)				
Intubation	122 (25.21)	45 (13.31)	77 (52.74)	<0.001
Acute kidney injury	135 (27.89)	55 (16.27)	80 (54.79)	<0.001
Renal replacement therapy	1 (0.21)	0 (0.00)	1 (0.68)	0.128
Lactate ≥2 mmol/L	277 (57.23)	162 (47.93)	115 (78.77)	<0.001
Platelet <100,000 cells/μL	41 (8.47)	16 (4.73)	25 (17.12)	<0.001
Intensive care unit admission; n (%)	134 (27.69)	34 (10.06)	100 (68.49)	<0.001
Length of hospital stay (days); median (IQR)		8 (5 to 12)	8 (4 to 14)	0.513
Discharge status; n (%)				<0.001
Expired	71 (14.67)	26 (7.69)	45 (30.82)	
Discharged to home	389 (80.37)	302 (89.35)	87 (59.59)	
Transferred to another hospital	16 (3.31)	6 (1.78)	10 (6.85)	
Left against medical advice	8 (1.65)	4 (1.18)	4 (2.74)	

IQR=interquartile range

Table 5. Fitted regression model for predicting shock in sepsis patients based on initial clinical signs and laboratory investigations

Variables	Odds ratio	95% CI	p-value
Hypertension	0.54	0.32 to 0.91	0.020
Body temperature <37.5°C	2.83	1.70 to 4.69	<0.001
Respiratory rate >22/minute	1.79	1.10 to 2.92	0.019
aPTT >29.9 seconds	2.06	1.27 to 3.36	0.004
Cr >1.25 mg/dL	2.30	1.39 to 3.79	0.001
Albumin <3.7 g/dL	2.26	1.37 to 3.72	0.001
Total bilirubin >0.92 mg/dL	2.51	1.51 to 4.18	<0.001
Positive hemoculture	0.46	0.28 to 0.76	0.003

aPTT=activated partial thromboplastin time; Cr=creatinine; CI=confidence interval

performance, with an area under the ROC curve of 0.804 (95% CI 0.757 to 0.851) (Figure 1).

Discussion

The present study highlights the clinical, laboratory, and prognostic differences between septic patients with and without shock. Approximately one-third (30.2%) developed shock, consistent with global prevalence[1,16]. Moreover, the findings provide region-specific evidence from a large general hospital in southern Thailand, thereby expanding the limited literature on sepsis epidemiology and risk stratification in middle-income healthcare settings, which remain underrepresented in global analyses of sepsis outcomes[17]. Despite similar baseline characteristics, shock patients exhibited more severe physiological compromise, organ dysfunction, and worse outcomes.

Hemodynamic and physiological changes

Shock patients had significantly lower body

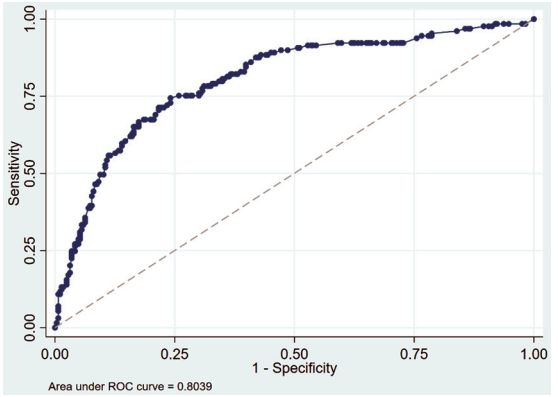

Figure 1. ROC curves for predicting shock in sepsis patients based on initial clinical signs and laboratory investigations (ROC area 0.804, 95% CI 0.757 to 0.851).

ROC, receiver operating characteristic

temperature and higher respiratory rates. Notably, the observation that a substantial proportion of patients with shock were afebrile, with a median of 37.5°C, underscores that fever is not a universal feature of severe infection. This atypical presentation is attributed to impaired thermoregulation and immune responses, as noted by Young et al.[18] and Shimazui et al.[19], and may delay diagnosis and treatment. Although heart rate did not differ significantly, respiratory rate was markedly higher in the shock group, reinforcing its role as an early clinical indicator[3,20]. Given its simplicity and accessibility, respiratory rate remains a valuable bedside tool for detecting early deterioration in resource-limited hospitals.

Biochemical abnormalities and organ dysfunction

Shock patients showed elevated lactate, creatinine, BUN, bilirubin, and prolonged PT/aPTT, along with lower hemoglobin, albumin, bicarbonate, and platelet levels, indicative of systemic hypoperfusion and multi-organ failure. Elevated lactate is a well-established marker of poor prognosis[22]. Importantly, lactate measurement is also practical and accessible in most hospitals, as point-of-care testing enables rapid bedside assessment. Nevertheless, its interpretation requires caution, since lactate elevation may occur in non-hypoperfusion states such as hepatic dysfunction, β-agonist therapy, or seizures[23,24]. Renal dysfunction, reflected by increased creatinine and AKI prevalence, aligns with KDIGO guidelines and studies by Bellomo et al. and Schrier et al.[13,25,26]. Liver dysfunction and coagulopathy further confirm severe organ impairment[26]. In addition, hypoalbuminemia, more frequent in the shock group, is established severity markers linked to systemic inflammation[27].

Comorbidities and infection profiles

An unexpected finding was that diabetes mellitus and hypertension were more common among patients without shock, which contrasts with findings from Lee et al.[8] and Hsiao et al.[9], who reported these comorbidities as significant risk factors for septic shock. One plausible explanation is that individuals with chronic conditions may be more likely to seek medical care promptly due to increased health awareness and routine monitoring. Additionally, healthcare providers are often trained to maintain a high index of suspicion for infections in diabetic patients, even when classical symptoms are absent, facilitating earlier interventions such as timely antibiotic administration and fluid resuscitation, both critical in preventing progression to shock.

Respiratory and hepatobiliary infections predominated in shock patients[3,9,16], whereas urinary tract infections, typically milder, were more frequent in non-shock cases[10].

Despite their critical condition, shock patients had lower blood culture positivity rates, consistent with Rudd et al.[16] and Tancharoen et al[6]. Explanations include prior antibiotics, occult infections, or non-bacterial etiologies. *Klebsiella* spp. and *A. baumannii* were more frequently isolated in shock patients, raising concerns due to their association with drug resistance and healthcare-associated infections[28].

Clinical outcomes and implications

Septic shock was significantly associated with higher mortality, ICU admission, mechanical ventilation, and multi-organ dysfunction, consistent with findings from larger multicenter and international studies[3,28]. Although hospital length of stay did not differ significantly, the markedly greater utilization of critical care resources among patients with shock underscores its substantial clinical and economic burden. These findings reaffirm the importance of early identification and aggressive management, including prompt initiation of appropriate antimicrobial therapy, adequate hemodynamic resuscitation, and timely source control, interventions consistently shown to reduce sepsis-related mortality. Comparable observations from Thailand and other Southeast Asian cohorts further emphasize the persistent burden of septic shock in resource-limited settings and the need for locally adapted, evidence-based sepsis

protocols that prioritize rapid triage and early goal-directed therapy[5,6].

Appraisal of the SIRS criteria

In the present study, sepsis was identified according to the SIRS criteria, which rely on readily available parameters, vital signs, and complete blood count. This approach is particularly practical for general hospitals, where advanced investigations such as arterial blood gas analysis, required for calculating the SOFA score, are not routinely performed. Nonetheless, our findings underscore the limitations of SIRS in capturing disease severity. Patients with septic shock met the abnormal temperature criteria less frequently at 39.0% versus 63.0% ($p<0.001$) and exhibited lower overall SIRS scores. Similar observations by Young et al.[18], Shimazui et al.[19], and Taniguchi et al.[20] indicate that SIRS may underestimate illness severity, particularly among elderly or immunocompromised patients.

Compared with the SOFA score proposed in Sepsis-3, which provides greater specificity for organ dysfunction[16,29], SIRS remains a feasible and time-efficient screening tool in resource-limited settings, where early recognition often outweighs diagnostic precision. In such contexts, integration of straightforward early warning systems, such as the National Early Warning Score (NEWS) or Modified Early Warning Score (MEWS), both incorporating vital parameters similar to SIRS, may further enhance bedside detection and facilitate prompt clinical intervention, as recommended by the Surviving Sepsis Campaign[3].

Predictors of septic shock

The multivariable analysis identified body temperature lower than 37.5°C, respiratory rate of more than 22 breaths/minute, aPTT greater than 29.9 seconds, serum creatinine greater than 1.25 mg/dL, albumin of less than 3.7 g/dL, and total bilirubin greater than 0.92 mg/dL as independent predictors of septic shock. Together, these parameters reflect early physiological and biochemical derangements associated with circulatory compromise, coagulopathy, and multi-organ dysfunction. Patients presenting without fever were more likely to develop shock, suggesting that an attenuated febrile response may accompany severe immune dysregulation and delayed infection recognition[18-20]. Elevated respiratory rate remains a simple yet sensitive marker of metabolic distress and tissue hypoxia[3,21]. The observed associations of elevated aPTT, creatinine, and bilirubin with shock underscore the interplay of coagulation, renal, and hepatic dysfunction in the progression of sepsis. Likewise, lower serum albumin concentrations indicate systemic inflammation, endothelial injury, and increased vascular permeability[27]. Collectively, these findings highlight that fundamental clinical and biochemical variables can serve as practical and accessible tools for early risk stratification and clinical decision-making in sepsis.

Interestingly, hypertension and positive blood culture were inversely associated with the occurrence of shock. The protective association observed among hypertensive patients may reflect greater healthcare engagement, such as regular follow-up and home blood pressure monitoring, enabling earlier recognition and treatment. In addition to these behavioral factors, prior studies by Yeo et al.[30] demonstrated that patients with pre-existing hypertension had improved outcomes in septic shock, due to adaptive vascular remodeling and enhanced tolerance to transient hypotension. This finding aligns with the present study, suggesting that chronic hypertension may not necessarily predispose patients to worse outcomes but could confer a degree of hemodynamic resilience during infection. Positive blood culture was likewise inversely associated with shock. Identification of a causative organism often facilitates pathogen-directed antimicrobial therapy and early source control, improving outcomes, as noted by Vincent & De Backer[31]. Conversely, culture-negative sepsis, frequently resulting from prior antibiotic exposure, occult infections, or diagnostic delay, has been linked to poorer prognosis[32-34]. Prior administration of antibiotics has been shown to reduce the likelihood of blood culture positivity by nearly half, potentially impairing diagnostic accuracy and delaying appropriate treatment[32]. A large multicenter cohort further demonstrated that patients with culture-negative septic shock experienced outcomes comparable to or worse than those with culture-positive disease, underscoring the prognostic significance of early pathogen identification and targeted therapy[33]. Clinically, these findings underscore the importance of individualized resuscitation targets, maintaining slightly higher mean arterial pressure in hypertensive patients may help prevent shock progression without excessive vasopressor exposure, while ensuring timely culture collection and organism-specific treatment may improve outcomes.

Overall, the regression model demonstrated good

discriminatory capacity (AUC 0.804), indicating that a combination of simple physiological and biochemical parameters can predict the development of septic shock with reasonable accuracy. Incorporating such variables into routine sepsis assessment may enhance early detection and facilitate timely, aggressive management, particularly in resource-limited healthcare environments.

Limitation and recommendation

Limitations should be acknowledged. The retrospective, single-center design may limit external validity and introduce selection bias, particularly given the heterogeneity of sepsis presentations across healthcare settings. The exclusion of patients with incomplete records and missing data, especially for key laboratory parameters, may have affected the robustness of the multivariable analysis. Additionally, the lack of information regarding treatment timing, such as antimicrobial initiation, fluid resuscitation, and source control procedures, restricted evaluation of how early interventions influenced outcomes. Serial measurements of lactate and organ function were unavailable, precluding assessment of dynamic changes that might better predict clinical deterioration or recovery. Moreover, residual confounding from unmeasured clinical or socioeconomic variables cannot be excluded.

Future investigations should employ prospective, multicenter study designs with standardized data collection to validate these predictors across diverse hospital contexts. Incorporating dynamic parameters, such as serial lactate trends, hemodynamic responses, and biomarker trajectories, may enhance prognostic accuracy. In addition, extending this work toward diagnostic prediction research, including the development of a point-based clinical scoring system derived from these predictors, may improve the clinical applicability of the model for bedside identification of septic shock. Integration of predictive models or risk-scoring systems with electronic medical records or digital early-warning systems could facilitate real-time risk stratification and guide timely interventions in sepsis and septic shock.

Conclusion

The present study provides comprehensive, region-specific insight into the clinical, biochemical, and prognostic characteristics of sepsis and septic shock in a large general hospital in southern Thailand. Approximately one-third of patients with sepsis developed shock, a prevalence aligned with international data. Simple physiological and laboratory parameters, specifically body temperature, respiratory rate, serum creatinine, aPTT, albumin, and bilirubin, were identified as independent predictors of septic shock, underscoring the diagnostic utility of readily obtainable indicators for early risk stratification. The inverse associations of hypertension and positive hemoculture with shock highlight the potential impact of consistent healthcare engagement and timely infection management on preventing clinical deterioration.

Taking them together, these findings reinforce the importance of prompt recognition, early antimicrobial therapy, hemodynamic optimization, and timely source control, particularly in resource-constrained environments where advanced diagnostic tools may be limited. Integration of these predictors into locally adapted sepsis protocols or digital early-warning systems could enhance the timeliness and precision of sepsis management. Prospective multicenter validation is warranted to refine predictive thresholds and support the development of evidence-based, regionally applicable sepsis management strategies. Future research should also explore translating these predictors into a practical clinical scoring system to further enhance early diagnostic accuracy and bedside decision-making.

What is already known about this topic?

Septic shock remains a major contributor to global morbidity and mortality, characterized by profound circulatory and metabolic disturbances that frequently lead to multi-organ failure[1-3]. Established prognostic indicators include hypotension, elevated serum lactate, and coagulation abnormalities[3,22,27]. Conventional diagnostic frameworks such as the SIRS criteria demonstrate limited sensitivity in identifying severe sepsis or shock, prompting the development of newer definitions under Sepsis-3[1,29]. Nevertheless, most existing evidence arises from tertiary or high-income healthcare settings, leaving a paucity of region-specific data from general hospitals in middle-income countries, where resource limitations and case heterogeneity may alter clinical presentation and outcomes[5,6,17]. Multidrug-resistant pathogens like *A. baumannii* and *K. pneumoniae* worsen prognosis, with respiratory infections more commonly associated with shock than urinary tract infections[7,8].

What does this study add?

This study identifies a combination of simple,

routinely available physiological and biochemical parameters, body temperature, respiratory rate, serum creatinine, aPTT, albumin, and bilirubin, as independent predictors of septic shock. It also demonstrates that afebrile presentations are common among patients with shock, emphasizing the need for diagnostic vigilance even in the absence of fever[18-20]. Additionally, the inverse associations of hypertension and positive hemoculture with shock suggest that consistent healthcare engagement and timely infection source control may mitigate disease progression[3,28-30]. By offering region-specific data from a large general hospital in southern Thailand, this study contributes to the limited literature from middle-income healthcare systems and supports the integration of fundamental clinical indicators into locally adapted sepsis protocols or digital early-warning tools to enhance early recognition and improve outcomes[5,6,28].

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Conflicts of interest

The author declares no conflict of interest.

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