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Original Article

The role of plasma presepsin levels in determining the incidence of septic shock and mortality in patients with sepsis

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Abstract

Introduction: The present study aimed to investigate the role of plasma presepsin in the early detection of septic shock and in determining the prognosis and mortality of patients with sepsis.

Methodology: The study was conducted in the emergency department between 1 January 2017 and 1 July 2017. A total of 106 patients 18 years of age or older who were diagnosed with sepsis according to the quick sequential organ failure assessment (qSOFA) criteria were included in this prospective study. The patients' symptoms, vital signs, additional diseases, demographic attributes, laboratory results, Mortality in Emergency Department Sepsis (MEDS) scores, imaging findings and treatments were recorded. Moreover, the patients' blood samples were collected to measure plasma presepsin, procalcitonin and CRP levels.

Results: In total, 55.7% of the patients were female. The median age of the patients was 78 (24–103) years, and their 30-day mortality rate was 67%. The presepsin level was significantly higher in the sepsis group than in the healthy control group ($p < 0.001$). The presepsin levels did not differ significantly between the sepsis and septic shock groups ($p = 0.12$). Similarly, the procalcitonin levels did not differ significantly between the sepsis and septic shock groups ($p > 0.05$). There was no significant difference in the presepsin, procalcitonin and CRP levels between survivor and non-survivor patients ($p = 0.74$).

Conclusions: The plasma presepsin level was found to be ineffective in determining the incidence of septic shock and mortality in patients with sepsis in the emergency department.

Key words: Sepsis; septic shock; presepsin; prognosis; mortality.

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Introduction

Sepsis is a syndrome involving organ dysfunction due to a dysregulated host response to infection [1-3]. It is a significant cause of mortality worldwide and is an important health problem due to its high mortality rates. The mortality rates vary between 20% and 50% in patients with sepsis and are often > 50% in patients with septic shock [4-7]. Bacteraemia is detected in only 50%-60% of patients with clinically suspected sepsis [8,9]. In routine laboratory practice, a specific diagnosis of bacteraemia is made by culturing the causative agent using blood and tissue fluid samples. This diagnostic method is still regarded as the gold standard. A certain period of time is needed to detect the active agent in blood culture, even in the most advanced systems [10,11]. However, immediate initiation of treatment following the early diagnosis of sepsis is associated with a good prognosis [11]. Antimicrobial treatment is generally initiated on the basis of the clinical findings and on detecting a high level of inflammatory markers, such as leukocytes, C-reactive protein (CRP) and procalcitonin (PCT), in the peripheral venous blood [4]. Unfortunately, neither clinical findings nor inflammatory markers are sensitive or specific enough for the diagnosis of sepsis [4].

Many biomarkers have been investigated to diagnose to this life-threatening clinical syndrome at an early stage, to enable timely initiation of the appropriate treatment and to make a clinical prediction regarding the prognosis and follow-up course. Presepsin, a

recently proposed sepsis marker, is a 13-kDa glycoprotein (soluble CD14 subtype, sCD14-ST) cut from the N-terminal end of CD14 [11]. CD-14 is an approximately 55-kDa protein that is present in the membranes of mononuclear cells; it serves as a high-affinity receptor to lipopolysaccharides (LPS) and the LPS/LPS-binding protein complex (LPBP). The LPS–LPBP–CD14 complex is separated from the cell membrane and released into the circulation to form sCD14. Presepsin is another form of sCD14 produced by plasma protease activity. Presepsin does not have LPS-binding properties; therefore, it cannot be captured by anti-CD14 antibodies and can be used as a biomarker. Several studies have revealed increased presepsin levels in the early stages of bacteraemia and sepsis and have reported that presepsin can be routinely used a biomarker [10-14]. The Mortality in Emergency Department Sepsis (MEDS) score was developed to determine the mortality rate in patients at risk of infection in emergency departments, to identify independent determinants of death and to finalise the treatment plans by classifying emergency infection patients based on mortality risk rates [2]. According to the MEDS score, patients with sepsis are evaluated with a score of 0–27 points. The expected 28-day mortality rate in patients scoring ≥15 is between 40% and 50%. The present study aimed to investigate the role of the plasma presepsin level in the early detection of septic shock and in determining the prognosis and mortality of patients with sepsis. In addition, we wanted to compare the plasma presepsin level with plasma procalcitonin and C-reactive protein levels.

Methodology

This prospective study included patients 18 years of age or older who were diagnosed with sepsis and septic shock at the Emergency Medicine Department between 1 January 2017 and 1 July 2017 and whose treatment was started or followed in the intensive care unit. In addition, 44 healthy individuals were included in the study (healthy control group). Three groups were formed in total: sepsis, septic shock and healthy control.

Sepsis group

The quick sequential organ failure assessment (qSOFA) criteria were used for the diagnosis of sepsis [1]. The criteria included respiratory rate ≥ 22/min, mental deterioration Glasgow coma score (GCS) ≤ 13 and systolic blood pressure ≤ 100 mmHg. Each criterion was regarded as one point, and patients with a score of ≥ 2 were included in the sepsis group [1].

Septic shock group

The patients who needed a vasopressor to maintain the mean arterial pressure at > 65 mmHg and whose lactate level was > 2 mmol/L were included in the septic shock group.

Healthy control group

The healthy control group consisted of volunteer patients' relatives and emergency department staff who did not have any disease.

Individuals who were 18 years of age or younger, who did not have 30-day results, who were not citizens of the Republic of Turkey, who had malignant disease or who received immunosuppressive treatment were excluded from the study.

The patients' complaints on arrival, vital signs, additional diseases, demographic features, symptoms, physical examination findings, laboratory results, imaging findings and treatments were recorded in a previously formed study draft. Moreover, the patients' MEDS scores were calculated and recorded. The MEDS score was calculated by recording the presence of terminal illness, tachypnoea or hypoxaemia, septic shock, platelet count < 150,000 cells/mm^3, band count as a percentage of total white blood cell count > 5%, age > 65 years, lower respiratory infection, nursing home residence and altered mental status.

Based on the patients' symptoms, tests were performed to determine the foci of infection. Microscopic examination of urine samples, abdominal and urogenital imaging (by x-ray, ultrasound and/or tomography) and thoracic radiological imaging (by x-ray and/or tomography) were performed, in addition to the evaluation of urine and blood cultures.

Blood samples were collected to measure the presepsin levels within 1 h after the diagnosis of sepsis and septic shock. In total, 5 mL of blood was drawn from the patients. Next, the blood samples were centrifuged at 3000 rpm for 15 min. The serum was then transferred into Eppendorf tubes using a serum pipette and frozen at −80°C until examination. To measure the presepsin levels, blood samples were collected from a total of 106 patients and 40 healthy individuals. Presepsin levels were measured in the hospital's biochemistry laboratory using the Human Presepsin ELISA Kit (#MBS766136) having a detection range of 0.156–10 ng/mL and sensitivity of < 0.094 ng/mL (BioVision, California, US). The erythrocyte sedimentation rate (ESR), procalcitonin level and CRP level were measured in the hospital laboratory using routine blood samples. ESR (range: 0–20 mm/h) was measured on the Test 1 device (Alifax SpA, Padova,

Italy) using the Westergren method. The CRP level (range: 0–0.5 mg/dL) was measured on the Beckman Coulter Immage 800 device (Beckman Coulter, CA, USA) using the nephelometric method. The procalcitonin level was measured on the AQT90 FLEX analyser (Radiometer Medical Aps, Copenhagen, Denmark) using the time-resolved fluorescence method (range: < 0.05 μg/L).

The patients were observed for 30 days, and the mortality rate was recorded.

The present study was conducted in accordance with the principles of the World Medical Association Declaration of Helsinki after receiving approval from the Ethics Committee (dated 06.11.2017 and numbered 42/29) at Ankara Diskapi Yildirim Beyazit Training and Research Hospital.

Statistical Analysis

The SPSS statistical programme (IBM Statistical Package for the Social Sciences Statistics for Windows,

Table 1. The comparison of sepsis and septic shock groups.

	All patients	**Sepsis (N = 53)**	**Septic shock (N = 53)**	**p**
Age, years (mean ± SD)	77.4 ± 12.9	78.3 ± 11.8	76.5 ± 14.1	0.91
Gender (n, %)				0.17
Female	59 (55.7%)	33 (62.3%)	26 (49.1%)	
Male	47 (44.3%)	20 (37.7%)	27 (50.9%)	
Symptoms (n, %)				
Altered of consciousness	81 (%76.4)	37 (69.8%)	44 (83%)	0.11
Coughing	53 (50%)	31 (58.5%)	22 (41.5%)	0.08
Sputum	53 (50%)	18 (34%)	30 (56.6%)	0.02
Dyspnea	50 (47.2%)	26 (49.1%)	20 (45.3%)	0.69
Nausea-Vomiting	23 (21.7%)	11 (20.8%)	12 (22.6%)	0.81
Abdomen pain	15 (14.2%)	5 (9.4%)	10 (18.9%)	0.16
Skin Erythema-Pain	11 (10.4%)	7 (13.2%)	4 (7.5%)	0.34
Dysuria	10 (9.4%)	6 (11.3%)	4 (7.5%)	0.50
Diarrhea	9 (8.5%)	4 (7.5%)	5 (8.5%)	0.73
Co-morbidity (N, %)				
Hypertension	43 (40.6%)	22 (41.5%)	28 (52.8%)	0.24
Diabetes mellitus	26 (24.5%)	13 (24.5%)	13 (24.5%)	1.00
Alzheimer	25 (23.6%)	18 (34%)	7 (13.2%)	**0.01**
COPD	22 (20.8%)	6 (11.3%)	14 (26.4%)	0.05
CHF	18 (17%)	6 (11.3%)	12 (22.6%)	0.12
CRF	15 (14.2%)	6 (11.3%)	9 (17%)	0.40
CVD	13 (12.3%)	6 (11.3%)	7 (13.2%)	0.67
Vital signs (mean ± SD)				
Fever, ^{0}C	37.7 ± 1.0	37.8 ± 0.9	37.7 ± 1.0	0.30
Systolic BP, mmHg	95.9 ± 21.4	109 ± 20.9	82.9 ± 12.1	< 0.001
Dyastolic BP, mmhg	57.8 ± 12.1	65.1 ± 11.3	50.6 ± 7.8	< 0.001
Pulse/min	110.1 ± 25.2	108.6 ± 19.1	111.6 ± 30.3	0.94
RR/min	25.4 ± 4.7	25.3 ± 4.4	25.7 ± 5.1	0.89
QSOFA, median (IQR)	2 (1)	2 (1)	3 (1)	0.002
MEDS, median (IQR)	10 (4.5)	8 (3.5)	11 (4)	< 0.001
Biomarkers (mean±SD)				
WBC ($\times 10^9$/L)	15.9 ± 9.6	16.4 ± 7.7	15.5 ± 11.2	0.20
ESR (mm/h)	34.5 ± 29.3	32.2 ± 26.1	36.9 ± 32.3	0.53
Lactate (mmol/L)	4.2 ± 2.9	4.3 ± 3.1	4.4 ± 2.8	0.99
Presepsin (ng/mL)	6.7 ± 3.7	6.2 ± 3.7	7.3 ± 3.6	0.12
CRP (mg/L)	207.4 ± 128.4	181.5 ± 118.6	233.3 ± 133.6	**0.03**
Procalcitonin (ng/mL)	19.7 ± 27.7	16.9 ± 25.3	22.6 ± 29.9	0.35
Result (n, %)				
Clinical Hospitalization	6 (5.7%)	3 (5.7%)	3 (5.7%)	1.00
ICU Hospitalization	100 (94.3%)	50 (94.3%)	50 (94.3%)	1.00
30-days Mortality	71(67%)	34 (64.2%)	37 (69.8%)	0.54

RR: Respiratory rate; COPD: Chronic obstructive pulmonary disease; CHF: Congestive Heart Failure; CRF: Chronic renal failure; CVD: Cerebral Vascular Disease; IUC: Intensive Care Unit.

version 17.0, SPSS Inc., Chicago, IL) was used for statistical analysis of the acquired data. The data were tested for normality using the Kolmogorov–Smirnov test. The Student's t-test was used to compare the data conforming to the normal distribution, while the Mann–Whitney U test was used to compare the data not conforming to the normal distribution. $p < 0.05$ was considered statistically significant. The chi-square test was used to compare the frequency of the data among the groups. Correlation was tested using Spearman's rank correlation coefficient. The Kruskal–Wallis test was used to compare the three groups (sepsis, septic shock and healthy control).

Results

A total of 106 patients who visited the emergency department between 1 January 2017 and 1 July 2017 were included in the present study. Fifty-nine of the patients were female (55.7%), while the remaining 47 were male (44.3%). Fifty percent of the patients (N = 53) had complaints related to cough and sputum. Hypertension was the most common comorbidity (Table 1). In the healthy control group, 52.5% of the

Table 2. The comparison of non-survivor and survivor patients.

	Non-survivors	Survivors	P
Age, years, median (IQR)	80 (12)	79 (11)	0.43
Gender (n, %)			0.30
Female	42 (59.2%)	17 (48.6%)	
Male	29 (40.8%)	18(51.4%)	
Symptoms (n, %)			
Altered of consciousness	58 (81.7%)	23 (65.7%)	0.06
Coughing	36 (50.7%)	17 (48.6%)	0.84
Sputum	41 (57.7%)	17 (48.6%)	0.37
Dyspnea	37 (52.1%)	13 (37.1%)	0.14
Nausea-Vomiting	16 (22.5%)	7 (20.0%)	0.77
Abdomen pain	9 (12.7%)	6 (17.1%)	0.53
Skin Erythema-Pain	8 (11.3%)	3 (8.6%)	0.67
Dysuria	7 (9.9%)	3 (8.6%)	0.83
Diarrhea	6 (8.5%)	3 (8.6%)	0.98
Comorbidity (n, %)			
Hypertension	31 (43.7%)	19 (54.3%)	0.30
Diabetes mellitus	21 (29.6%)	5 (14.3%)	0.08
Alzheimer	14 (19.7%)	11 (31.4%)	0.18
COPD	13 (18.3%)	7 (20.0%)	0.82
CHF	13 (18.3%)	5 (14.3)	0.60
CRF	12 (16.9%)	3 (8.6%)	0.24
CVD	12 (16.9%)	1 (7.7%)	**0.04**
Vital signs (mean±SD)			
Fever, ^{0}C	37.7 ± 0.9	37.8 ± 0.9	0.52
Systolic BP, mmHg	96.6 ± 21.5	94.5 ± 21.7	0.59
Dyastolic BP, mmHg	58.2 ± 12.5	57.1 ± 11.4	0.89
Pulse / min	109.7 ± 24.1	110.9 ± 27.7	0.98
RR / min	25.0 ± 4.7	26.3 ± 4.8	0.23
QSOFA, median (IQR)	2 (1)	2 (2)	0.63
MEDS, median (IQR)	10 (5)	10 (5)	0.57
Biomarkers (mean±SD)			
WBC ($\times 10^9$/L)	16.1 ± 10.5	15.5 ± 7.5	0.82
ESR (mm/h)	31.5 ± 30.3	40.8 ± 26.6	**0.03**
Lactate (mmol/L)	4.4 ± 3.1	3.9 ± 2.5	0.48
Presepsin (ng/mL) (mean±SD)	6.7 ± 3.5(0.39-13.9)	6.9 ± 4.1(0.79-15.4)	0.74
CRP (mg/L) (mean±SD)	197.8 ± 133.2(9.1-544)	227 ± 117.5(7.4-471)	0.09
Procalcitonin (ng/mL) (mean±SD)	17.6 ± 26.7(0.1-100)	24.0 ± 29.6(0.22-100)	0.19
Patients Groups (n, %)			0.53
Sepsis	34 (47.9%)	19 (54.3%)	
Septic shock	37 (52.1%)	16 (45.7)	

RR: Respiratory rate; COPD: Chronic obstructive pulmonary disease; CHF: Congestive Heart Failure; CRF: Chronic renal failure; CVD: Cerebral Vascular Disease.

Table 3. The comparison of biomarkers according to blood culture results.

	Blood Culture (+) (n = 28)	Blood Culture (-) (n = 78)	P
Presepsin (ng/mL)	5.8 ± 3.2 (1.2-3.3)	7.1 ± 3.8 (0.8-15.4)	0.15
CRP (mg/L)	211.8 ± 136.6 (9.1-4739	205.9 ± 126.2 (7.4-544)	0.9
Procalcitonin (ng/mL)	19.6 ± 24.2 (0.6-100)	19.8 ± 29 (0.1-100)	0.45

p-values were calculated by Mann–Whitney U test.

individuals were female; the mean age was 44 ± 14 years. In total, 50% (N = 53) of the patients were diagnosed with sepsis, while the remaining 50% (N = 53) were diagnosed with septic shock. The qSOFA score was 2 in 55 patients and 3 in 51 patients. The most common reservoir of infection was the lung (48.1%) and urinary tract (48.1%). The 30-day mortality rate of the patients was 67% (N = 71). The average plasma presepsin level of the healthy controls was 0.50 ± 0.1 ng/mL (0.41–1.01 ng/mL), while that of the patients with sepsis and septic shock was 6.7 ± 3.7 ng/mL; the difference was statistically significant ($p < 0.001$) (Figure 1). The average presepsin levels did not differ significantly between the sepsis and septic groups ($p = 0.12$). Among the other inflammatory markers studied, a significant difference was noted only in CRP levels ($p = 0.03$) between these groups (Table 1). The plasma presepsin levels did not differ significantly between the survivor and non-survivor patients during the 30-day period ($p = 0.74$). Among the other inflammatory markers studied, only erythrocyte sedimentation rate (ESR) differed significantly between these patients ($p = 0.03$) (Table 2). The presepsin levels did not differ significantly between positive and negative blood cultures ($p = 0.15$). No significant differences were detected in the levels of other inflammatory markers as well (Table 3). The qSOFA and MEDS scores were significantly higher in the septic shock group than in the sepsis group ($p < 0.05$) (Table 1). There was no significant difference in the qSOFA and MEDS scores between deceased survivor and non-survivor patients ($p > 0.05$) (Table 2). The relationship between plasma biomarker levels and qSOFA and MEDS scores is shown in Table 4. A significant relationship was noted only between the CRP level and qSOFA score. In 51 patients, lung imaging findings were indicative of pneumonia. The urine culture of 30 patients and blood culture of 28 patients were positive. All the patients received crystalloid resuscitation and antibiotic therapy. Epinephrine infusion was performed in 50% (N = 53) of the patients, while corticosteroid drug treatment was administered to 3% (N = 3). There was no statistical significance in univariate and multivariate analyzes of inflammatory markers for 30-day mortality (Table 5).

Discussion

Sepsis is associated with a high mortality rate, and as it progresses towards septic shock, the mortality rate increases. The 30-day mortality rate was 67% in the present study. In the literature, the 30-day mortality rates have been reported to vary between 39% and 74% [3,14-16]. An early diagnosis of sepsis is therefore crucial. Presepsin has been found to be a potential biomarker among different molecules used recent studies for the early diagnosis of sepsis [14, 17]. We

Figure 1. The mean plasma presepsin level of healthy controls was 0.50 ± 0.1 ng / mL (0.41-1.01 ng / mL), and that of patients with sepsis and septic shock was 6.7 ± 3.7 ng / mL. The difference between the two groups was statistically significant (p < 0.001).

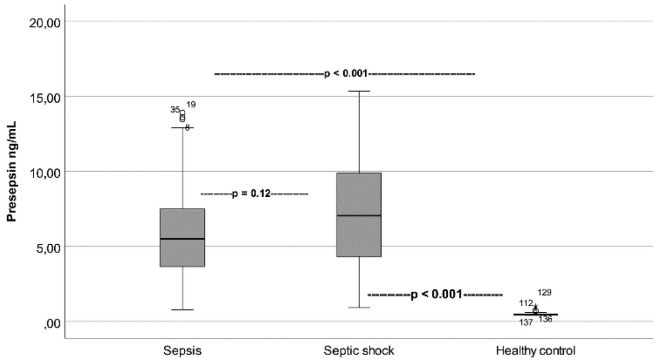

Table 4. The relationship of plasma CRP, PCT, and presepsin levels with the qSOFA and MEDS score.

	qSOFA	MEDS
Presepsin, r	0.06	-0.04
p	0.53	0.68
Procalcitonin, r	0.11	-0.037
p	0.28	0.71
CRP, r	0.232	0.069
p	0.02	0.48

Spearman's correlation test.

Table 5. Univariate and multivariate analysis of biomarker levels for 30-day mortality.

	B	SE	Wald	df	p	OR	95% CI	
							Lower	Upper
Univariate								
Presepsin	0.039	0.034	1.301	1	0.254	1.040	0.972	1.111
Procalcitonin	0.001	0.005	0.064	1	0.800	1.001	0.991	1.012
CRP	0.000	0.001	0.131	1	0.718	1.000	0.997	1.002
Multivariate								
Presepsin	0.009	0.058	0.024	1	0.876	1.009	0.901	1.130
Procalcitonin	0.006	0.008	0.610	1	0.435	1.006	0.991	1.021
CRP	0.001	0.002	0.627	1	0.428	1.001	0.998	1.005

found that the plasma presepsin level in the sepsis group was higher than that in the healthy control group, in accordance with the findings of Bo Liu et al. [14] and Marcoulla et al. [18] In their study conducted on 859 patients, Bo Liu *et al.* reported significantly higher presepsin levels in patients with septic shock than in those with sepsis [14]. However, in another study, they did not detect a significant difference in plasma presepsin levels between patients with septic shock and those with severe sepsis [11]. Yamamoto et al. did not report a significant difference in presepsin levels between patients with sepsis and those with septic shock [3], similar to the findings of the present study. Our study included a less number of patients, of which many had serious sepsis. This situation may have resulted in the absence of a significant difference. Independently of sepsis, Nakamura et al. found an increase in the severity of injury and plasma presepsin levels in patients with acute kidney injury. Hence, they reported that not only sepsis but also accompanying kidney injuries contribute to an increase in presepsin levels [19]. In the present study, we did not separate the patients with or without acute kidney injury. Therefore, a significant difference may not have appeared between the sepsis and septic shock groups. We did not observe significant differences in procalcitonin and lactate levels and ESR between sepsis and septic shock groups. Only the CRP level was significantly higher in the septic shock group than in the sepsis group. Similarly, Kweon et al. did not report a difference in procalcitonin levels among sepsis, severe sepsis and septic shock groups [11]. In addition, they reported a significant difference in high-sensitivity CRP (hsCRP) levels between serious sepsis and sepsis groups but no significant difference between serious sepsis and septic shock groups [11]. The levels of inflammatory markers may differ depending on the profile of the patients. We did not observe a significant difference in presepsin levels between survivor and non-survivor patients. Similarly, Kweon et al. did not ascertain a relationship between the plasma presepsin levels examined on day 1 and 30-day mortality [11]. However, Brodska *et al.* reported significantly higher presepsin levels in deceased patients [20]. In another study, Kim et al. reported higher mortality rates in patients with high presepsin levels [21]. The fact that the findings of the reported studies are incompatible necessitates further research. In addition, we did not observe a significant difference in plasma CRP levels between survivor and non-survivor patients. The relationship between the CRP level and mortality has been confirmed in previous studies on sepsis [22,23]. The findings of the present study can be attributed to the fact that the patients with sepsis included in the study had a serious clinical status.

Moreover, we did not observe a significant difference in plasma procalcitonin levels between survivor and non-survivor patients. Jedynak *et al.* revealed that a high procalcitonin level was associated with 28-day mortality in patients with sepsis [24]. In their study involving 109 patients with septic shock and 50 control subjects, Spoto et al. identified a high procalcitonin level to be significantly effective in determining both diagnosis and mortality [25]. Barre *et al.* examined 136 patients with sepsis or septic shock and reported that procalcitonin levels on days 1, 3 and 8 were not significant in determining 30-day mortality [26]. In the present study, no significant difference was noted between presepsin levels according to whether there was a reproduction in the blood culture of the patients whose blood cultures were taken. Similarly, in another study conducted on 106 patients, the level of presepsin was identified as non-significant according to the foci of infection [18]. The most important limitation of the present study is the low number of patients. This may have resulted in the weak effect of plasma biomarkers on the prognosis and mortality. In addition, a different group with infection only could be included in the study. Another limitation is the lack of serial measurements of biomarkers. These limitations may have affected our results.

Conclusions

In the present study, the plasma presepsin level was not found to be effective in determining the incidence of septic shock and mortality in patients with sepsis in the emergency department. The CRP level was more successful in detecting septic shock compared to the presepsin and procalcitonin levels. New studies with a larger number of patients are needed to predict the prognosis and mortality.

Authors' Contributions

All authors contributed equally to this study.

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