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The Value of Five Scoring Systems in Predicting the Prognosis of Patients with COVID-19

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Abstract: Scoring systems are routinely used in the intensive care unit (ICU) to evaluate disease prognosis. However, the value of these scoring systems for COVID-19 patient assessment is unclear. This study was conducted to identify the optimal scoring system for predicting the prognosis of COVID-19 patients. All data were obtained from the fourth version of the Medical Information Mart for Intensive Care (MIMIC-IV) database. Patients were grouped into two groups according to survival status at 28 days after admission. Independent risk factors for death in hospitals were identified by Logistic and Cox regression analysis. The scores of five scoring systems were calculated and collected. The predictive value of each of the five scoring systems was evaluated by the area under the receiver operating characteristic curve (AUROC). Multiple subgroup analyses were performed with respect to 28-day mortality according to age and sex. A total of 4274 COVID-19 patients were included. The median patient age was 67 (57,77) years, and 2507 patients (58.7%) were men. The median SIRS, SOFA, OASIS, SAPSII, and APSIII scores were higher in the nonsurvival group than in the survivor group. The discrimination for 28-day mortality using the SAPSII (AUROC 0.774, 95% confidence interval (CI): 0.755–0.793) and APSIII (AUROC 0.767, 95% CI: 0.748–0.786) models was superior to that using the SOFA (AUROC 0.727, 95% CI: 0.707–0.748), OASIS (AUROC 0.740, 95% CI: 0.721–0.760), SIRS (AUROC 0.617, 95% CI: 0.595–0.640), and Charlson (AUROC 0.666, 95% CI: 0.644–0.688) models. The Youden index of the SAPSII model was 0.407, which was the highest among the models. The results of subgroup analyses were similar to the overall results. The SAPSII and APSIII models was superior than other scoring systems with regards to the discrimination of 28-day mortality in COVID-19 patients.

1. Introduction

Since it first emerged in late 2019, the COVID-19 pandemic which was caused by the novel coronavirus SARS-CoV-2, has presented unprecedented challenges to worldwide healthcare systems[1,2]. This pandemic disease presents with a variety of forms which may cause multiorgan failure and high mortality. It is necessary to develop a robust prognostic tool to guide clinical decision-making and resource allocation[1]. In the context of a severe shortage of ICU beds, accurate

prognostic models can facilitate the prioritization of patients with the highest likelihood of therapeutic benefit.

Previous studies have developed various scoring systems[3-10]. The Sequential Organ Failure Assessment (SOFA) score was developed to evaluate the degree of organ dysfunction, providing a comprehensive view of disease progression[3]. The Acute Physiology Score II and III (APS III) was developed to predict mortality risk in critically ill patients by incorporating physiological variables[4-7]. The Systemic Inflammatory Response Syndrome (SIRS) criteria was developed to identify systemic inflammation, caused by infection, trauma and burn, et al[8]. The Oxford Acute Severity of Illness Score (OASIS) was developed to provide a quick assessment of illness severity in acute care settings[9]. Finally, the Charlson Comorbidity Index (Charlson) was developed to quantify comorbidity burden[10].

These scoring systems have been used widely. For instance, studies have demonstrated the superior performance of the APS III and LODS in predicting 28-day mortality in sepsis-associated acute respiratory failure patients[11], whereas the SOFA score was used to predict the mortality in sepsis patients[12-14]. However, the application of these scoring systems in COVID-19 patients was few. The purpose of this study was to determine which scoring system was best to predict mortality in COVID-19 patients.

2. Methods

Data for this study were extracted from the MIMIC-IV version 3.0 database. This publicly available resource contains information of 546,028 patients admitted to the Beth Israel Deaconess Medical Center between 2008 and 2022, of whom 364,627 were admitted to the ICU.

Author Youfeng Zhu obtained access to the database (agreement date: January 4, 2021). This study was conducted in adherence to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines[15].

2.1 Patient selection

Adult COVID-19 patients were identified from the 364,267 admissions in MIMIC-IV v3.0 by ICD-10 codes. The following exclusion criteria were used: (1) age under 18 years, (2) ICU stay less than 24 hours, or (3) multiple ICU admissions (in which case only data from the first admission was retained).

2.2 Data extraction

We recorded baseline characteristics and clinical information within the first 24 hours of ICU admission.

Demographics: age, sex, height, weight.

Vital Signs: heart rate (HR), respiratory rate (RR), temperature, mean arterial pressure (MAP), peripheral blood oxygen saturation (SpO2).

Intervention: mechanical ventilation (MV) utilization.

We also extracted the following laboratory parameters within the first 24 hours of ICU admission: blood glucose level, hemoglobin level, white blood cell (WBC) count, platelet count, albumin level, blood urea nitrogen (BUN) level, sodium level, potassium level and serum creatinine level.

Comorbidities were assessed using the Charlson comorbidity score system based on the ICD-9 and ICD-10 codes. The evaluated comorbidities included chronic heart failure, myocardial infarction (MI), and chronic pulmonary disease, hypertension, chronic kidney disease, diabetes, cirrhosis, stroke, and malignant cancer. Additionally, we collected data with regards to 28-day mortality after ICU

admission, the use of continuous renal replacement therapy (CRRT) and mechanical ventilation (MV) during hospitalization, and the length of hospital stay and ICU stay.

Records of SIRS, SOFA, APS III, SAPS II, OASIS, and Charlson scores were also extracted.

2.3 Statistical analysis

Statistical analyses were carried out using SPSS (version 27) and STATA (version 18). Normally distributed data are described as the means ± standard deviations (SDs). In contrast, nonnormally distributed data are expressed as medians (interquartile ranges) [M (QL, QU)]. We employed Student's t-test or nonparametric tests for continuous variables, as appropriate. Fisher's exact test was used for analyzing of categorical variables, and the results are shown as counts and percentages. Logistic and Cox regression analyses were carried out to determine the independent risk factors associated with 28-day mortality. Variables with a P value < 0.05 from the univariate analysis were incorporated into the multivariate model. We employed the area under the receiver operating characteristic curve (AUROC) to assess and compare the discriminatory power of the models. Specifically, an AUROC ranging from 0.7 to 0.8 indicates a fair model, a range of 0.8 to 0.9 signifies a good model, and a value greater than 0.9 denotes an excellent model.

2.4 Subgroup analyses

To validate the reliability of the findings concerning the ability of the models to predict 28-day mortality in COVID-19 patients, subgroup analyses were conducted. In accordance with prior studies, the subgroups were categorized by sex (male vs. female) and age (⩾60 years vs. <60 years). A two-tailed P value of less than 0.05 was considered significant.

3. Results

3.1 Patient characteristics

Initially, 51,997 patients admitted to the ICU for the first time and for more than 24 h were screened. Patients with insufficient baseline records (defined as more than 30% missing data) were excluded from the analysis. Ultimately, 4,274 patients were involved. Based on 28-day survival status after ICU admission, we divided the patients into two groups: a nonsurvival group (3569 patients) and a survival group (705 patients). A flow chart of the data extraction process is shown in Figure 1. The baseline characteristics of the patients cohort are summarized in Table 1. Overall, the median age was 67 years (IQR 57-77), and 58.7% of the patients (n=2,507) were male.

Significant differences in baseline demographics were observed between both groups. Patients in the survival group were younger (65.27 ± 15.78 years) compared to those in the non-survival group (69.57 ± 15.23 years; P < 0.001). The proportion of male patients was also higher in the survival group (59.2% vs. 55.7%; P < 0.001). As shown in Table 1, several other baseline parameters differed significantly between the nonsurvival and survival groups. These parameters included vital signs (MAP, HR, RR, SpO2), laboratory values (WBC and platelet counts, hemoglobin, BUN, creatinine, PCO2, PO2, and glucose levels) (Table 1). A total of 705 patients (16.49%) died within 28 days of ICU admission. The nonsurvival group had a significantly more frequent use of both mechanical ventilation (58.9% vs. 37.8%; P < 0.001) and CRRT (21.6% vs. 4.5%; P < 0.001) compared to the survival group. The length of ICU stay [4.45 (1.99, 8.75) days vs. 3.00 (1.77, 6.15) days; P < 0.001) and hospital stay [9.12 (4.77, 15.925) days vs. 10.55 (6.63, 20.02) days; P < 0.001] differed significantly between both groups.

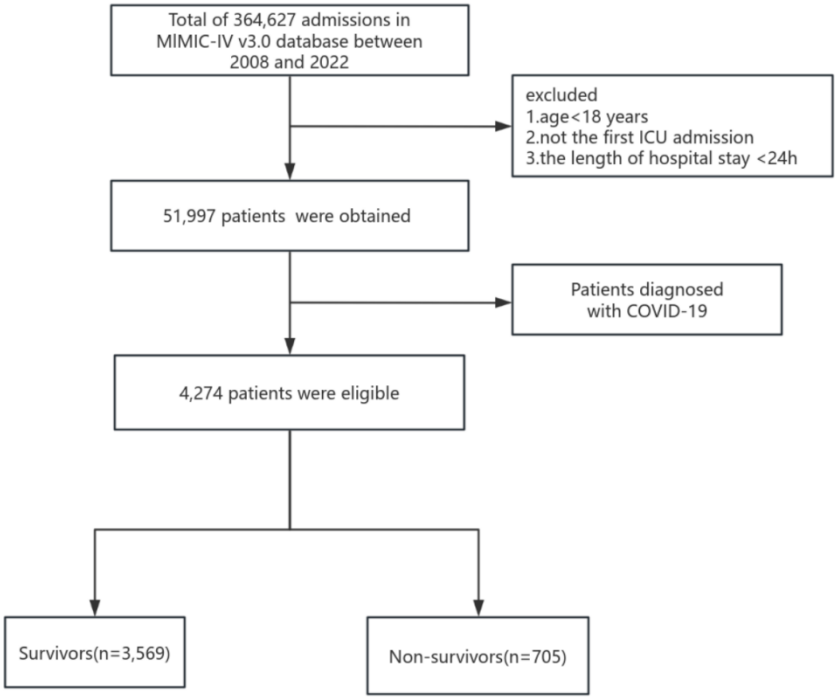

Figure 1. Flow chart of the study patients selection process

Table 1. Baseline characteristics between survivors and non-survivors

Variables	Total (n=2407)	Survivors(n=3569)	Non-survivors(n=705)	P - value
Demographics and characteristics				
Age,year,median (IQR)	67(57,77)	67 (55,76)	72 (63,82)	＜0.001
Gender,no.(%)				0.086
Male	2507(58.7%)	2114(59.2%)	393(55.7%)	
Female	1767(41.3%)	1455(40.8%)	312(44.3%)	
Weight,median (IQR)	80.00(67.10,95.80)	80.70(67.87,97.00)	77 (63.75,91.45)	＜0.001
Signs and symptoms				
Heart Rate,median (IQR)	85(75,100)	84 (74,98)	91 (79,108)	＜0.001
MAP,mmHg,median (IQR)	85(74,97)	85 (75,97)	84 (72,96)	0.009
Respiratory rate,median (IQR)	19(16,23)	18 (15,22)	21 (18,26)	＜0.001
SPO2,%,median (IQR)	97(95,100)	98 (95,100)	96 (93,99)	＜0.001

Temperature,℃,median (IQR)	36.78(36.56,37.06)	36.78(36.56,37.06)	36.78(36.50,37.80)	0.088
		Laboratory findings		
WBC count, ×109/L, median (IQR)	10.80(7.80,15.20)	10.60 (7.70,14.75)	11.80 (8.30,17.00)	＜0.001
Platelet count, ×109/L, median (IQR)	195(137,264)	197(142,264)	183(111,262)	＜0.001
Hemoglobin,g/dL,median (IQR)	10.90(9.10,12.70)	11.10(9.20,12.80)	10.30(8.30,12.30)	＜0.001
Blood glucose,mg/dL, median (IQR)	129(107,165)	128(106,160)	141(113,191)	＜0.001
Sodium,mEq/L,median (IQR)	138(135,140)	138(135,140)	138(134,142)	0.054
Potassium,mEq/L,median (IQR)	4.10(3.80,4.60)	4.10(3.80,4.50)	4.20(3.80,4.80)	＜0.001
PCO2,mmHg,median (IQR)	41(36,48)	41(36,47.70)	41(35,49)	0.549
PO2,,mmHg,median (IQR)	101(52,202.42)	112(56,218)	68(43.50,117)	＜0.001
PT,sec,median (IQR)	14.10(12.50,17.10)	13.90(12.40,16.70)	15.20(13.10,19.50)	＜0.001
Total bilirubin,mg/dL,median (IQR)	0.70(0.40,1.60)	0.70(0.40,1.66)	0.70(0.40,1.50)	0.728
ALT,IU/L,median (IQR)	36(16.90,196)	37(16,223)	33(17,86.50)	0.002
AST,IU/L,median (IQR)	49(24,270.40)	49(24,317.70)	51(26,141.50)	0.542
Blood urea nitrogen,mg/dL, median(IQR)	18(12,30)	17(12,26)	30(18,49.50)	＜0.001
Creatinine,mg/dL, median(IQR)	0.90(0.70,1.40)	0.90(0.70,1.30)	1.30(0.90,2.30)	＜0.001
		Comorbidities,no.(%)		

Hypertension, no.(%)	1678(39.3%)	1455(40.8%)	223(31.6%)	<0.001
Diabetes, no.(%)	1258(29.4%)	1024(28.7%)	234(33.2%)	0.017
Heart Failure,no.(%)	1056(24.7%)	824 (23.1%)	232 (32.9%)	<0.001
Myocardial infarct,no.(%)	572(13.4%)	464 (13.0%)	108 (15.3%)	0.099
Chronic kidney disease,no.(%)	767(17.9%)	581 (16.3%)	186 (26.4%)	<0.001
Chronic pulmonary diease,no.(%)	471(11.0%)	367 (10.3%)	104 (14.8%)	<0.001
Cirrhosis,no.(%)	292(6.8%)	201 (5.6%)	91 (12.9%)	<0.001
Stroke,no.(%)	318(7.4%)	259 (7.3%)	59 (8.4%)	0.034
Malignant cancer,no.(%)	621(14.5%)	471 (13.2%)	150 (21.3%)	<0.001
		Organ dysfunction		
SOFA,median (IQR)	4(2,7)	4(2,6)	7(4,10)	<0.001
APSIII,median (IQR)	39(29,55)	37(27,50)	59(44,79)	<0.001
SIRS,median (IQR)	3(2,3)	2(2,3)	3(2,3)	<0.001
SAPSII,median (IQR)	35(27,45)	34(26,42)	48(38,59)	<0.001
OASIS,median (IQR)	31(25,37)	30(24,35)	38(32,43)	<0.001
Charlson,median (IQR)	5(3,7)	5(3,7)	6(4,9)	<0.001
		Outcomes		

Ventilation,no.(%)	1764(41.3%)	1349 (37.8%)	415 (58.9%)	＜0.001
CRRT,no.(%)	313(7.3%)	161 (4.5%)	152 (21.6%)	＜0.001
ICU stay, days, median (IQR)	3.13(1.80,6.68)	3.00(1.77,6.15)	4.45(1.99,8.75)	＜0.001
Hospital stay, days, median (IQR)	10.19(6.25,19.13)	10.55(6.63,20.02)	9.12(4.77,15.93)	＜0.001

IQR, interquartile range; MAP, mean artery pressure; WBC, white blood vell count; SpO2, pulse oxygen saturation; PCO2, partial pressure of carbon dioxide; PO2 ,partial pressure of oxygen; PT, prothrombin time; ALT, alanine aminotransferase; AST, aspartate aminotransferase; SOFA, Sequential Organ Failure Assessment; APS III, Acute Physiology Score III; SIRS, systemic inflammatory response syndrome; SAPS II, Simplified Acute Physiology Score II; OASIS, Oxford Acute Severity of Illness Score; Charlson, Charlson Comorbidity Index; CRRT, Continuous Renal Replacement Therapy.

We assessed factors associated with 28-day mortality through the multivariate logistic regression model (Table 2). Among the variables, age (OR 1.012, 95% CI: 1.003 – 1.021, P=0.012), weight (OR 0.993, 95% CI: 0.990 – 0.997, P=<0.001), respiratory rate (OR 1.016, 95% CI: 1.001 – 1.032, P=0.039), PO2 (OR 0.996, 95% CI: 0.995 – 0.997, P=<0.001), total bilirubin (OR 1.030, 95% CI: 1.000 – 1.062, P=0.048), blood urea nitrogen (OR 1.011, 95% CI: 1.005 – 1.016, P=<0.001), creatinine (OR 0.861, 95% CI: 0.784 – 0.945, P=0.002), diabetes (OR 0.682, 95% CI: 0.540 – 0.861, P=0.001), chronic kidney disease (OR 0.719, 95% CI: 0.537 – 0.962, P=0.026, P=0.026), malignant cancer (OR 1.358, 95% CI: 1.023-1.765, P=0.033), MV use (OR 1.623, 95% CI: 1.339-1.968, P<0.001), and CRRT use (OR 1.51, 95% CI: 1.199-1.901, P=<0.001) were independent risk variables for 28-day mortality in COVID-19 patients.

Table 2. Multivariate logistic regression analysis of risk factors for the 28-day mortality in COVID-19 patients

Covariates	Univariate model		Multivariate model	
	Odds Ratio (95% CI)	P value	Odds Ratio (95% CI)	P value
Age	1.029(1.023,1.035)	<0.001	1.012(1.003,1.021)	0.012
Weight	0.991(0.988,0.995)	<0.001	0.993(0.990,0.997)	<0.001
Gender	1.153(0.980,1.358)	0.086	-	-
Respiratory rate	1.062(1.049,1.075)	<0.001	1.016(1.001,1.032)	0.039
Heart rate	1.017(1.013,1.020)	<0.001	0.999(0.994,1.004)	0.625
MAP	0.995(0.990,0.999)	0.024	1.004(0.998,1.009)	0.171
Spo2	0.936(0.918,0.954)	<0.001	0.977(0.955,1.001)	0.058

Temperature	0.939(0.809,1.090)	0.409	-	-
WBC count	1.023(1.014,1.033)	<0.001	1.003(0.995,1.010)	0.518
Platelet count	0.998(0.997,0.999)	<0.001	0.999(0.998,1.000)	0.060
Hemoglobin	0.920(0.890,0.951)	<0.001	1.006(0.964,1.051)	0.774
Blood glucose	1.003(1.002,1.004)	<0.001	1.001(1.000,1.002)	0.183
Sodium	1.020(1.005,1.036)	0.001	1.002(0.986,1.019)	0.796
Potassium	1.282(1.157,1.420)	<0.001	1.044(0.916,1.189)	0.523
PCO2	1.009(1.002,1.016)	0.012	1.004(0.995,1.013)	0.384
PO2	0.994(0.993,0.995)	<0.001	0.996(0.995,0.997)	<0.001
PT	1.040(1.030,1.050)	<0.001	1.020(1.009,1.031)	<0.001
Total bilirubin	1.071(1.048,1.094)	<0.001	1.030(1.000,1.062)	0.048
ALT	1.000(0.999,1.000)	0.007	1.000(0.999,1.000)	0.784
AST	1.000(1.000,1.000)	0.044	1.000(1.000,1.000)	0.299
Blood urea nitrogen	1.025(1.021,1.028)	<0.001	1.011(1.005,1.016)	<0.001
Creatinine	1.171(1.122,1.222)	<0.001	0.861(0.784,0.945)	0.002
Hypertension	0.672(0.566,0.799)	<0.001	0.899(0.705,1.148)	0.393
Diabetes	1.235(1.039,1.468)	0.017	0.682(0.540,0.861)	0.001
Heart Failure	1.634(1.371,1.947)	<0.001	0.917(0.716,1.175)	0.495
Myocardial infarct	1.211(0.965,1.519)	0.099	-	-
Chronic kidney disease	1.843(1.525,2.228)	<0.001	0.719(0.537,0.962)	0.026
Chronic pulmonary diease	1.510(1.194,1.909)	0.001	1.118(0.845,1.478)	0.435
Cirrhosis	2.483(1.911,3.228)	<0.001	0.863(0.570,1.306)	0.486
Stroke	1.167(0.869,1.568)	0.304	-	-
Malignant cancer	1.778(1.449,2.182)	<0.001	1.344(1.023,1.765)	0.033
Ventilation	0.425(0.360,0.501)	<0.001	1.623(1.339,1.968)	<0.001
CRRT	0.172(0.135,0.218)	<0.001	1.51(1.199,1.901)	<0.001
SOFA	1.284(1.252,1.316)	<0.001	1.074(1.024,1.127)	0.003
APSIII	1.044(1.040,1.048)	<0.001	1.013(1.004,1.021)	0.003
SIRS	1.654(1.504,1.819)	<0.001	1.229(1.083,1.395)	0.001
SAPSII	1.075(1.068,1.082)	<0.001	1.004(0.990,1.019)	0.549
OASIS	1.110(1.099,1.122)	<0.001	1.044(1.026,1.061)	<0.001
Charlson	1.213(1.181,1.245)	<0.001	1.179(1.122,1.239)	<0.001

MAP, mean artery pressure; SpO2, pulse oxygen saturation; PCO2,partial pressure of carbon dioxide; PO2 ,partial

pressure of oxygen; PT, prothrombin time ; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRRT, Continuous Renal Replacement Therapy; SOFA, Sequential Organ Failure Assessment; APS III, Acute Physiology Score III; SIRS, systemic inflammatory response syndrome; SAPS II, Simplified Acute Physiology Score II; OASIS, Oxford Acute Severity of Illness Score; Charlson, Charlson Comorbidity Index.

Table 3 shows the results of the Cox regression analysis of risk factors for 28-day mortality in COVID-19 patients. Age (HR 1.012, 95% CI: 1.005-1.020, P=0.001), weight (HR 0.993, 95% CI: 0.990-0.997, P<0.001), RR (HR 1.014, 95% CI: 1.002-1.026, P=0.021), platelet count (HR 0.999, 95% CI: 0.015-1.000, P=0.008), PO2 (HR 0.996, 95% CI: 0.995-0.997, P<0.001), PT (HR 1.014, 95% CI: 1.007-1.021, P<0.001), total bilirubin level (HR 1.033, 95% CI: 1.015-1.051, P<0.001), ALT level (HR 1.000, 95% CI: 1.000-1.000, P=0.021), blood urea nitrogen level (HR 1.006, 95% CI: 1.002-1.009, P=0.003), creatinine level (HR 0.884, 95% CI: 0.822-0.951, P=0.001), diabetes (HR 0.682, 95% CI: 0.540-0.861, P=0.001), chronic kidney disease (HR 0.806, 95% CI: 0.651-0.997, P=0.047), and malignant cancer (HR 1.356, 95% CI: 1.104-1.665, P=0.004) were independent risk variables for 28-day mortality in COVID-19 patients.

Table 3. Cox regression analysis of risk factors for 28-day mortality in COVID-19 patients

Covariates	Univariate model		Multivariate model	
	Hazard Ratio (95% CI)	P value	Hazard Ratio (95% CI)	P value
Age	1.026(1.021,1.032)	<0.001	1.012(1.005,1.020)	0.001
Weight	0.992(0.989,0.996)	<0.001	0.993(0.990,0.997)	<0.001
Gender	0.870(0.750,1.010)	0.067	-	-
Respiratory rate	1.053(1.043,1.064)	<0.001	1.014(1.002,1.026)	0.021
Heart rate	1.015(1.012,1.018)	<0.001	0.999(0.996,1.003)	0.786
MAP	0.995(0.990,0.999)	0.014	1.001(0.997,1.005)	0.544
Spo2	0.948(0.935,0.962)	<0.001	0.984(0.966,1.002)	0.074
Temperature	0.931(0.808,1.072)	0.320	-	-
WBC count	1.011(1.008,1.014)	<0.001	1.001(0.995,1.007)	0.726
Platelet count	0.998(0.998,0.999)	<0.001	0.999(0.998,1.000)	0.008
Hemoglobin	0.924(0.896,0.953)	<0.001	1.004(0.97,1.039)	0.824
Blood glucose	1.003(1.002,1.003)	<0.001	1.000(0.999,1.001)	0.417
Sodium	1.020(1.006,1.035)	0.006	1.000(0.987,1.012)	0.958
Potassium	1.251(1.143,1.369)	<0.001	1.025(0.923,1.138)	0.648
PCO2	1.008(1.002,1.015)	0.011	1.005(0.998,1.013)	0.125
PO2	0.995(0.994,0.996)	<0.001	0.996(0.995,0.997)	<0.001

PT	1.021(1.017,1.025)	<0.001	1.014(1.007,1.021)	<0.001
Total bilirubin	1.059(1.046,1.073)	<0.001	1.033(1.015,1.051)	<0.001
ALT	1.000(0.999,1.000)	0.008	1.000(1.000,1.000)	0.021
AST	1.000(1.000,1.000)	0.061	-	-
Bloodurea nitrogen	1.017(1.015,1.019)	<0.001	1.006(1.002,1.009)	0.003
Creatinine	1.088(1.067,1.111)	<0.001	0.884(0.822,0.951)	0.001
Heart Failure	1.555(1.329,1.820)	<0.001	1.028(0.862,1.226)	0.756
Myocardial infarct	1.202(0.979,1.476)	0.078	-	-
Chronic kidney disease	1.725(1.459,2.040)	<0.001	0.806(0.651,0.997)	0.047
Chronic pulmonary diease	1.442(1.171,1.776)	0.001	1.126(0.906,1.401)	0.285
Cirrhosis	2.265(1.818,2.823)	<0.001	0.900(0.666 ,1.217)	0.495
Stroke	1.150(0.881,1.501)	0.304	-	-
Malignant cancer	1.657(1.384,1.985)	<0.001	1.356(1.104,1.665)	0.004
Ventilation	2.192(1.887,2.547)	<0.001	1.623(1.339,1.968)	<0.001
CRRT	4.301(3.593,5.148)	<0.001	1.510(1.199,1.901)	<0.001
SOFA	1.238(1.216,1.261)	<0.001	1.031(0.994,1.070)	0.101
APSIII	1.034(1.032,1.037)	<0.001	1.011(1.005,1.018)	0.001
SIRS	1.601(1.469,1.745)	<0.001	1.191(1.075,1.319)	0.001
SAPSII	1.059(1.054,1.063)	<0.001	1.004(0.994,1.015)	0.399
OASIS	1.094(1.085,1.103)	<0.001	1.016(1.003,1.030)	0.020
Charlson	1.178(1.153,1.204)	<0.001	1.113(1.074,1.153)	<0.001

MAP, mean artery pressure; SpO2, pulse oxygen saturation; PCO2,partial pressure of carbon dioxide; PO2 ,partial pressure of oxygen; PT, prothrombin time ; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRRT, Continuous Renal Replacement Therapy; SOFA, Sequential Organ Failure Assessment; APS III, Acute Physiology Score III; SIRS, systemic inflammatory response syndrome; SAPS II, Simplified Acute Physiology Score II; OASIS, Oxford Acute Severity of Illness Score; Charlson, Charlson Comorbidity Index.

3.2 Comparison of the prediction models

The predictive values of the scoring systems for 28-day mortality were compared using the AUROC. The AUROC curves are shown in Figure 2. The specific values are shown in Table 4: SAPSII (AUROC: 0.774, 95% CI: 0.755-0.793), APSIII (AUROC: 0.767, 95% CI: 0.748-0.786), OASIS (AUROC: 0.740, 95% CI: 0.721-0.760), SOFA (AUROC: 0.727, 95% CI: 0.707-0.748), Charlson (AUROC: 0.666, 95% CI: 0.644-0.688), and SIRS (AUROC: 0.617, 95% CI: 0.595-0.640). The AUROCs of the SAPSII, APSIII, OASIS and SOFA scores were greater than 0.7. SAPSII had the highest AUROC. The threshold of each scoring system corresponding to Youden’s index was chosen as the best threshold for predicting 28-day mortality in COVID-19 patients. Among the scoring systems, SAPSII demonstrated the highest Youden's index (0.407) and sensitivity(75%), with

a specificity(65.7%) that was considered acceptable. In contrast, APSIII had the highest specificity (76.3%).

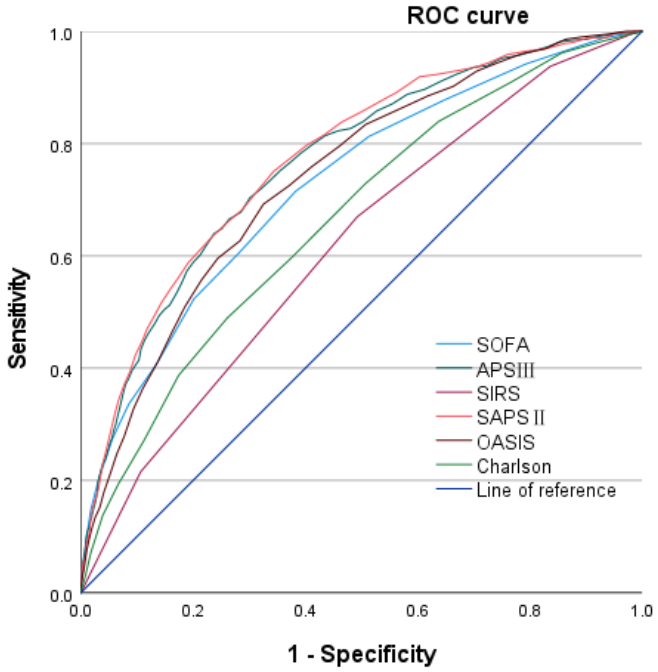

Figure 2. Comparison of receiver operating characteristic curves for predicting 28-day mortality in COVID-19 patients

Table 4. Comparison of different scoring systems for predicting 28-day mortality in COVID-19 patients

Covariates	AUROC	95% CI	P -value	Sensitivity	Specificity	Youden index
SOFA	0.727	0.707-0.748	<0.0001	0.715	0.618	0.333
APSIII	0.767	0.748-0.786	<0.0001	0.640	0.763	0.403
SIRS	0.617	0.595-0.640	<0.0001	0.670	0.508	0.178
SAPSII	0.774	0.755-0.793	<0.0001	0.750	0.657	0.407
OASIS	0.740	0.721-0.760	<0.0001	0.692	0.675	0.367
Charlson	0.666	0.644-0.688	<0.0001	0.489	0.739	0.228

AUROC, area under the receiver operating characteristic curve; CI, confidence interval; SOFA, Sequential Organ Failure Assessment;APS III, Acute Physiology Score III;SIRS, systemic inflammatory response syndrome; SAPS II, Simplified Acute Physiology Score II; OASIS, Oxford Acute Severity of Illness Score;Charlson, Charlson Comorbidity Index;

3.3 Subgroup analyses

To further evaluate the predictive credibility of the models, we performed subgroup analyses according to sex (male, female) and age (categorizing patients as < 60 or ≥60 years). In line with the overall analysis, the APSIII model's superior discriminatory performance for 28-day mortality was

consistently observed in all predefined subgroups (Table 5). The AUROCs of the APSIII model in each subgroup were 0.790 (Age<60 years, 95% CI: 0.749-0.831), 0.758 (Age ≥60 years, 95% CI: 0.735- 0.780), 0.774 (Male, 95% CI: 0.749-0.799), and 0.758 (Female, 95% CI: 0.728-0.788), respectively, and the Youden indices of the APSIII model in each subgroup were 0.430, 0.398, 0.420, and 0.397, respectively.

The SAPSII model performed second only to APSIII in discriminating 28-day mortality across all subgroups. The AUROCs of the SAPSII model in each subgroup were 0.786 (Age<60 years, 95% CI: 0.743-0.829), 0.749 (Age ≥60 years, 95% CI: 0.726-0.772), 0.778 (Male, 95% CI: 0.753-0.803), and 0.769 (Female, 95% CI: 0.740-0.798), respectively, and the Youden indices of the SAPSII model in each subgroup were 0.455, 0.375, 0.409, and 0.413, respectively (Table 5).

Table 5. Subgroup analysis of scoring models for predicting 28-day mortality in patients with COVID-19

Subgroup (n , %)	Covariates	AUROC	95% CI	P -value	Sensitivity	Specificity	Youden index
Age<60 (1386,32.4%)	SOFA	0.742	0.692-0.793	<0.0001	0.507	0.899	0.406
	APSIII	0.790	0.749-0.831	<0.0001	0.619	0.811	0.430
	SIRS	0.641	0.594-0.689	<0.0001	0.769	0.438	0.207
	SAPSII	0.786	0.743-0.829	<0.0001	0.642	0.813	0.455
	OASIS	0.737	0.695-0.779	<0.0001	0.776	0.565	0.341
	Charlson	0.638	0.590-0.686	<0.0001	0.642	0.567	0.209
Age>60 (2888,67.6%)	SOFA	0.724	0.700-0.747	<0.0001	0.713	0.614	0.327
	APSIII	0.758	0.735-0.780	<0.0001	0.636	0.762	0.398
	SIRS	0.626	0.601-0.651	<0.0001	0.646	0.546	0.192
	SAPSII	0.749	0.726-0.772	<0.0001	0.618	0.757	0.375
	OASIS	0.733	0.710-0.756	<0.0001	0.716	0.651	0.367
	Charlson	0.638	0.612-0.664	<0.0001	0.459	0.752	0.211
Male (2507,58.7%)	SOFA	0.737	0.710-0.764	<0.0001	0.756	0.595	0.351
	APSIII	0.774	0.749-0.799	<0.0001	0.751	0.669	0.420
	SIRS	0.615	0.585-0.645	<0.0001	0.654	0.501	0.155
	SAPSII	0.778	0.753-0.803	<0.0001	0.753	0.656	0.409
	OASIS	0.738	0.712-0.765	<0.0001	0.684	0.680	0.364
	Charlson	0.669	0.640-0.698	<0.0001	0.735	0.506	0.241
Female (1767,41.3%)	SOFA	0.719	0.687-0.751	<0.0001	0.663	0.651	0.314
	APSIII	0.758	0.728-0.788	<0.0001	0.567	0.830	0.397
	SIRS	0.621	0.588-0.654	<0.0001	0.689	0.518	0.207
	SAPSII	0.769	0.740-0.798	<0.0001	0.638	0.775	0.413
	OASIS	0.742	0.713-0.771	<0.0001	0.702	0.667	0.369
	Charlson	0.660	0.627-0.693	<0.0001	0.494	0.730	0.224

AUROC, area under the receiver operating characteristic curve; CI, confidence interval; SOFA, Sequential Organ Failure Assessment;APS III, Acute Physiology Score III;SIRS, systemic inflammatory response syndrome; SAPS

II, Simplified Acute Physiology Score II; OASIS, Oxford Acute Severity of Illness Score; Charlson, Charlson Comorbidity Index.

4. Discussion

COVID-19 is a disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which poses a great threat to human health, life and public safety. Although most patients have mild symptoms, 25 – 34% still experience respiratory failure, severe pneumonia, ARDS, and critical illnesses such as septic shock and multiple organ dysfunction, seriously endangering the lives of patients[1,2]. The COVID-19 pandemic has underscored a critical need for reliable prognostic tools to predict outcomes in critically ill patients, particularly those admitted to the ICU[16-19]. Previous studies have evaluated various scoring systems in a variety of disease, such as sepsis, acute respiratory failure, and other critical conditions[2-4]. However, the application of these scoring systems in COVID-19 patients was few. Our study showed that the SAPSII and APSIII models was superior for predicting 28-day mortality in COVID-19 patients.

The scoring systems are useful for COVID-19 management[16-19]. First, it can identify high-risk patients who may require intensive care early. Second, these tools can help to identify those COVID-19 patients with high risk of mortality, providing an early warning to clinical staffs. Third, these scoring system can be used for risk stratification of COVID-19 patients, and valuable in clinical trials and epidemiological studies.

The ICU scoring systems are widely used for predict the prognosis in a variety of patients[20-26]. Oliveira et al. perfromed a retrospective study in Brazil to evaluate the prognostic value of six widely used scoring systems in ICU patients with HIV/AIDS[27].They found that all models demonstrated good calibration. Ghanghoria et al. evaluated the scoring systems in critically ill patients with infectious disease. They found SOFA was more reliable than other scoring systems for assessing mortality in these patients[28]. Our previous study also evaluated the scoring systems in sepsis 2.0 patients and found that the SAPSII had a better prediction value than other scoring systems for assessing 28-day mortality in these patients[29]. In this study, we evaluated the performance of five widely used scoring systems (SOFA, APSIII, SIRS, SAPSII and OASIS) for predicting 28-day mortality in COVID-19 patients. Our findings reveal that the SAPSII demonstrates the best discrimination capacity for 28-day mortality. This finding is consistent with our previous research. SAPSII demonstrates superior performance in our study, which may be because SAPSII score system incorporates many important physiological variables. These variables, including respiratory failure, multiorgan dysfunction and systemic inflammation, are highly relevant to severe COVID-19 pathophysiology. In comparison, the Charlson Comorbidity Index was less effective in predicting mortality in COVID-19 patients. This suggests that acute physiological derangements play an important role in COVID-19 outcomes than pre-existing conditions.

The major strength of our study is that we involved a large cohort of COVID-19 patients in the ICU. This provides robust data for evaluating these scoring systems in this specific population. Furthermore, we used various comparison methods among the scoring systems, making our findings more reliable. Our results support using SAPSII in critical ill patients, even for a new disease, such as COVID-19. Our results also highlight that the prognostic models are needed to be reevaluated periodically. Their performance can change over time and be different across patient populations.

This study has several limitations. Firstly, this is a single-center study, and the generalizability of our findings is limited. Secondly, this is a retrospective study, because of the nature of the study, the potential of bias could not be avoided. Thirdly, the dynamic nature of COVID-19 and its evolving variants may affect the long-term performance of the scoring systems, which means it is necessary with regards to the ongoing evaluation and potential model recalibration.

5. Conclusion

The SAPSII and APSIII models was superior than other scoring systems with regards to the discrimination of 28-day mortality in COVID-19 patients. Compared with the other models, the SAPSII model showed the best discrimination capacity for 28-day mortality.

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