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Predictive factors for disease progression in hospitalized patients with coronavirus disease 2019 in Wuhan, China

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ABSTRACT

Background: A few studies have revealed the clinical characteristics of hospitalized patients with COVID-19. However, predictive factors for the outcomes remain unclear.
Objective: Attempted to determine the predictive factors for the poor outcomes of patients with COVID-19.
Study design: This is a single-center, retrospective study. Clinical, laboratory, and treatment data were collected and analyzed from 111 hospitalized patients with laboratory-confirmed COVID-19 in Union Hospital. The gathered data of discharged and deteriorated patients were compared.
Results: Among these 111 patients, 93 patients were discharged and 18 patients were deteriorated. The lymphocyte count (0.56 G/L [0.47 – 0.63] vs 1.30 G/L [0.95 – 1.65]) was lower in the deteriorated group than those in the discharged group. The numbers of pulmonary lobe involved (5.00 [5.00–5.00] vs 4.00 [2.00 – 5.00]), serum C-reactive protein (CRP, 79.52 mg/L [61.25 – 102.98] vs 7.93 mg/L [3.14 – 22.50]), IL-6 (35.72 pg/mL [9.24 – 85.19] vs 5.09 pg/mL [3.16 – 9.72]), and IL-10 (5.35 pg/mL [4.48 – 7.84] vs 3.97 pg/mL [3.34 – 4.79]) concentrations in deteriorated patients were elevated compared with discharged patients. Multivariate logistic regression analysis showed that male gender (OR, 24.8 [1.8 – 342.1]), comorbidity (OR, 52.6 [3.6 – 776.4]), lymphopenia (OR, 17.3 [1.1 – 261.8]), and elevated CRP (OR, 96.5 [4.6 – 2017.6]) were the independent risk factors for the poor prognosis in COVID-19 patients.
Conclusions: This finding would facilitate the early identification of high-risk COVID-19 patients.

1. Background

Coronavirus disease 2019 (COVID-19) is an emerging lethal respiratory disease from December 2019 [1]. Full-genome sequencing analysis has indicated that the pathogen is a novel enveloped RNA betacoronavirus currently named as severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) [2]. Since first identified, the epidemic scale of the recently emerged COVID-19 has increased rapidly, with cases arising across China and other countries [3,4].

Recently, a few studies have revealed the clinical characteristics of hospitalized patients with COVID-19 [1,5]. Huang et al. indicated that 32 % of patients were admitted to an ICU and 15 % of patients died among the 41 hospitalized patients, and the ICU patients had higher plasma levels of proinflammatory cytokines [1]. Wang et al. proved that patients treated in the ICU were older men with comorbidities, dyspnea, and anorexia compared with those not treated in the ICU among 138 hospitalized patients with COVID-19 [6]. Nevertheless, the predictive risk factors for the poor outcomes of COVID-19 patients remain unclear.

2. Objectives

We, therefore, collected the data of clinical manifestations together with detailed laboratory examination and attempted to determine the predictive factors for the poor outcomes of patients with COVID-19.

3. Study design

The laboratory-confirmed patients with COVID-19 admitted to Union Hospital, Tongji Medical College, Huazhong University of Science and Technology from January 13 to February 16 in 2020 were

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enrolled. All patients were diagnosed based on the WHO guidance [6]. We excluded the patients who were prescribed corticosteroids or immunosuppressant within 14 days before admission, procalcitonin level more than 0.5 ug/L, and influenza, bacteria, or fungi infection revealed by nasal and pharyngeal swab cultures on admission. This study was approved by the ethics committee of Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, and complied with the principles expressed in the Declaration of Helsinki. Written informed consent was waived because of the urgent situation and the retrospective nature by the ethics commission.

A total of 111 patients were included. The medical history, clinical manifestation, comorbidities, radiologic assessments, laboratory findings on admission, and treatment strategies were extracted and cross-checked from electronic medical records. Comorbidities included hypertension, cardiovascular disease, diabetes, chronic obstructive pulmonary disease, chronic liver disease, and malignancy. Numbers of pulmonary lobe involved were evaluated by chest computed tomography on admission. Laboratory tests on admission comprised complete blood count, liver and renal function, C-reactive protein (CRP), interleukin (IL)-2, IL-4, IL-6, IL-10, TNF-α, and IFN-γ. Laboratory confirmation of SARS-CoV-2 was achieved by the RT-PCR assay conducted in accordance with the protocol established by the WHO [7]. All laboratory tests were performed using commercial kits in the department of clinical laboratory of Union Hospital. The preliminary assessment of disease severity was developed by 6-category ordinal scale of clinical status on admission as follows: category 6, death; 5, intensive care unit (ICU) hospitalization, requiring extracorporeal membrane oxygenation (ECMO) and/or invasive mechanical ventilation; 4, ICU hospitalization, not requiring ECMO and/or invasive mechanical ventilation; 3, non-ICU hospitalization, requiring supplemental oxygen; 2, non-ICU hospitalization, not requiring supplemental oxygen; 1, hospital discharge [8].

The primary outcome was the disease deterioration, including the transfer from isolation ward to ICU and all-cause death. The included patients were divided into two groups according to their clinical outcomes: group with favorable prognosis (discharge after recovery) and group with poor prognosis (disease deterioration).

Continuous variables were expressed as median (interquartile range, IQR) and compared with the Mann-Whitney U test; categorical variables were expressed as number (%) and compared with χ^2 test or Fisher's exact test between discharged and deteriorated group. A two-sided α of less than 0.05 was considered statistically significant. Odds ratio (OR) for poor prognosis in COVID-19 patients was analyzed with multivariate logistic regression adjusted for selected confounders: age, gender, comorbidity, body temperature, number of pulmonary lobe involved, leukocyte count, neutrophil count, lymphocyte count, monocyte count, alanine aminotransferase, aspartate aminotransferase, C-reactive protein level, IL-6 level, and IL-10 level on admission. For this analysis, the upper limit of IQRs of this cohort was used as the cut-off values for age (57 years), CRP (39.0 mg/L), IL-6 (15.7 pg/mL), and IL-10 (5.1 pg/mL), respectively. A two-tailed p-value of < 0.05 was considered statistically significant. All analyses were carried out with SPSS version 13.0 (SPSS, Chicago, IL, USA).

4. Results

The clinical characteristics are shown in Table 1. The median age was 38.0 years (IQR, 32.0–57.0), and 46 (41.4 %) patients were males. 33.3 % of patients had at least one comorbidity (hypertension, diabetes, chronic obstructive pulmonary disease, malignancy, and chronic liver disease). The most common symptoms on admission were fever (71.2 %), cough (37.8 %), fatigue (18.0 %), and dyspnea (16.2 %). Symptoms including diarrhea (9.0 %), pharyngalgia (6.3 %), myalgia (6.3 %), and headache (3.6 %) were rare. The median duration from illness onset to admission was 7 days (IQR, 5.0–10.0). Among these 111 patients, 93 patients were discharged, and 18 patients were deteriorated, although there was no significant difference in scale of clinical status on admission between these two groups. Of the 18 deteriorated patients, 15 patients had died and 3 patients remained hospitalized in ICU up to Feb 26th, 2020.

Compared with the discharged patients, the deteriorated patients were significantly older (median age, 36.0 years [IQR, 31.0 − 47.5]) vs 60.0 years [IQR, 48.5 − 81.5], have more underlying comorbidities (22 [23.7 %] vs 15 [83.3 %]), and were more likely to report dyspnea (9 [9.7 %] vs 9 [50.0 %]). Days from illness onset to admission were not different between discharged and deteriorated patients. Increased proportions of elevated body temperature, respiratory frequency, and systolic pressure were higher in the deteriorated group compared with the discharged group.

Table 2 shows the laboratory findings on admission. White blood cell counts (6.51 G/L [4.03 − 10.10] vs 3.97 G/L [3.14 − 5.72]) and neutrophil counts (5.68 G/L [3.10 − 9.37] vs 2.34 G/L [1.82 − 3.51]) were higher, whereas lymphocyte count (0.56 G/L [0.47 − 0.63] vs 1.30 G/L [0.95 − 1.65]) were lower in the deteriorated group than those in the discharged group. The proportions of liver dysfunction (12 [66.67 %] vs 21 [22.58 %]) were increased in the deteriorated patients compared with the discharged patients. The numbers of pulmonary lobe involved (5.00 [5.00 − 5.00] vs 4.00 [2.00 − 5.00]), CRP (79.52 mg/L [61.25 − 102.98] vs 7.93 mg/L [3.14 − 22.50]), IL-6 pg/mL (35.72 [9.24 − 85.19] vs 5.09 pg/mL [3.16 − 9.72]), and IL-10 (5.35 pg/mL [4.48 − 7.84] vs 3.97 pg/mL [3.34 − 4.79]) concentrations in deteriorated patients were elevated compared with the discharged patients.

During hospitalization, the treatments of these patients were adjusted according to the patient's condition (Table 3). All patients received antiviral therapy, mostly antibacterial therapy. Corticosteroids were given to 27.0 % of cases, and more in dead cases than discharged cases (88.9 % vs 15.1 %). All the deteriorated patients required mechanical ventilation, and ECMO was employed in one severe case.

The main baseline clinical and laboratory characteristics by CRP quartiles are shown in Table 4. The patients with elevated CRP levels had a greater proportion of comorbidity and dyspnea. Age, lymphopenia, IL-6/IL-10 concentrations, and the numbers of pulmonary lobe involved were increased with the rising CRP level. And most of the deteriorated patients (88.9 %) were divided into the last quartile of CRP levels (> 39.00 mg/L).

The results of multivariate logistic regression analysis are shown in Table 5. The 14 significant variables were included. After adjusted, male gender (OR, 24.8 [1.8 − 342.1]), comorbidity (OR, 52.6 [3.6 − 776.4]), lymphopenia (OR, 17.3 [1.1 − 261.8]), and elevated CRP (OR, 96.5 [4.6 − 2017.6]) were found as the significant risk factors for the poor prognosis in COVID-19 patients.

5. Discussion

We report here a cohort of 111 laboratory-confirmed hospitalized patients with COVID-19. In this cohort, most patients presented with fever, cough, and dyspnea. However, upper respiratory tract signs and gastrointestinal symptoms were rare, suggesting different viral tropism as compared with severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) [9,10].

Among these 111 patients in isolation ward at baseline, 93 (83.8 %) patients were discharged, and 18 (16.2 %) were deteriorated. Those patients with poor prognosis were older male patients with more comorbidities, dyspnea, higher neutrophil count, lower lymphocyte count, more liver dysfunction, and increased numbers of pulmonary lobe involved from chest CT images. Moreover, we noted that patients with poor prognosis also had high amounts of CRP, IL-6, and IL-10 on admission. For further multivariate analysis of these risk factors, male, comorbidity, lymphopenia, and obviously elevated CRP were the significant predictors of poor prognosis in patients with COVID-19.

Hypertension was the dominant comorbidity in this study. The

Table 1
Baseline clinical characteristics of patients with COVID-19.

	ALL (n = 111)	Discharge (n = 93)	Deterioration (n = 18)	p value
Age (years)	38.0 (32.0 – 57.0)	36.0 (31.0 – 47.5)	60.0 (48.5 – 81.5)	< 0.001
Sex (male/female)	46/65	32/61	14/4	0.001
Comorbidity	37 (33.3 %)	22 (23.7 %)	15 (83.3 %)	< 0.001
Hypertension	15 (13.5 %)	5 (5.4 %)	10 (55.6 %)	< 0.001
Cardiovascular disease	3 (2.7 %)	1 (1.1 %)	2 (11.1 %)	0.068
Chronic obstructive pulmonary disease	3 (2.7 %)	2 (2.2 %)	1 (5.6 %)	0.415
Diabetes	14 (12.6 %)	5 (5.4 %)	9 (50.0 %)	< 0.001
Malignancy	8 (7.2 %)	8 (8.6 %)	0	0.350
Chronic liver disease	1 (0.9 %)	0	1 (5.6 %)	0.162
Signs and symptoms				
Fever	79 (71.2 %)	63 (67.7 %)	16 (88.9 %)	0.090
Cough	42 (37.8 %)	39 (41.9 %)	3 (16.7 %)	0.062
Dyspnea	18 (16.2 %)	9 (9.7 %)	9 (50.0 %)	< 0.001
Pharyngalgia	7 (6.3 %)	7 (7.5 %)	0	0.362
Fatigue	20 (18.0 %)	18 (19.4 %)	2 (11.1 %)	0.520
Myalgia	7 (6.3 %)	5 (5.4 %)	2 (11.1 %)	0.596
Headache	4 (3.6 %)	4 (4.3 %)	0	0.610
Diarrhea	10 (9.0 %)	9 (9.7 %)	1 (5.6 %)	0.698
Chest pain	12 (10.8 %)	9 (9.7 %)	3 (16.7 %)	0.408
Temperature > 37.3℃	39 (35.1 %)	26 (28.0 %)	13 (72.2 %)	0.001
Respiratory rate > 24 breaths per min	14 (12.6 %)	6 (6.5 %)	8 (44.4 %)	< 0.001
Systolic pressure (mmHg)	124.0 (115.0 – 134.0)	122.0 (115.0 – 130.0)	135.5 (111.0 – 153.0)	0.034
Diastolic pressure (mmHg)	79.0 (74.0 – 87.0)	80.0 (74.0 – 88.0)	78.0 (74.5 – 85.5)	0.703
Heart rate (bpm)	84.0 (78.0 – 98.0)	83.0 (78.0 – 97.0)	90.0 (78.8 – 100.5)	0.069
Days from illness onset to admission	7.0 (5.0 – 10.0)	7.0 (5.0 – 10.0)	8.0 (4.0 – 13.3)	0.917
6-category ordinal scale of clinical status				0.303
2	63	55	8	
3	48	38	10	

P values indicate differences between discharge and dead patients. P < 0.05 was considered statistically significant.

Table 2
Laboratory findings of COVID-19 patients on admission to hospital.

	ALL (n = 111)	Discharge (n = 93)	Deterioration (n = 18)	p value
White blood cell count (G/L)	4.30 (3.21 – 6.36)	3.97 (3.14 – 5.72)	6.51 (4.03 – 10.10)	0.002
Neutrophil count (G/L)	2.52 (1.85 – 4.30)	2.34 (1.82 – 3.51)	5.68 (3.10 – 9.37)	< 0.001
Lymphocyte count (G/L)	1.20 (0.83 – 1.62)	1.30 (0.95 – 1.65)	0.56 (0.47 – 0.63)	< 0.001
Monocyte count (G/L)	0.32 (0.22 – 0.43)	0.33 (0.23 – 0.46)	0.28 (0.19 – 0.37)	0.103
Red blood cell count (T/L)	4.19 (3.92 – 4.55)	4.17 (3.91 – 4.53)	4.21 (3.95 – 4.56)	0.517
Platelet count (G/L)	182.00 (139.00 – 237.00)	190.00 (144.50 – 238.00)	144.50 (122.25 – 212.75)	0.381
Liver dysfunction	33 (29.73 %)	21 (22.58 %)	12 (66.67 %)	< 0.001
Alanine aminotransferase (U/L)	23.00 (16.00 – 36.00)	22.00 (15.00 – 33.00)	29.50 (24.50 – 51.00)	0.004
Aspartate aminotransferase (U/L)	24.00 (19.00 – 39.00)	23.00 (18.00 – 32.50)	45.00 (32.75 – 60.75)	< 0.001
kidney dysfunction	2 (1.80 %)	0	2 (1.80 %)	0.162
Blood urea nitrogen (mmol/L)	3.93 (2.99 – 5.10)	3.69 (2.89 – 4.40)	6.30 (4.94 – 9.39)	< 0.001
Creatinine (μmol/L)	69.50 (57.80 – 82.70)	66.90 (57.30 – 77.90)	83.95 (69.90 – 109.93)	0.001
C-reactive protein (mg/L)	11.30 (3.14 – 39.00)	7.93 (3.14 – 22.50)	79.52 (61.25 – 102.98)	< 0.001
Cytokines				
IL-6 (pg/mL)	6.37 (3.61 – 13.73)	5.09 (3.16 – 9.72)	35.72 (9.24 – 85.19)	< 0.001
IL-2 (pg/mL)	2.56 (2.33 – 2.72)	2.56 (2.34 – 2.74)	2.34 (2.32 – 2.72)	0.127
IL-4 (pg/mL)	1.95 (1.62 – 2.31)	1.95 (1.59 – 2.31)	1.98 (1.65 – 2.27)	0.800
IL-10 (pg/mL)	4.23 (3.49 – 5.10)	3.97 (3.34 – 4.79)	5.35 (4.48 – 7.84)	< 0.001
TNF-α (pg/mL)	2.12 (1.81 – 2.31)	2.09 (1.80 – 2.36)	2.16 (1.81 – 2.25)	0.391
IFN-γ (pg/mL)	2.12 (1.82 – 2.50)	2.09 (1.80 – 2.53)	2.17 (1.83 – 2.28)	0.746
Numbers of pulmonary lobe involved	4.00 (2.00 – 5.00)	4.00 (2.00 – 5.00)	5.00 (5.00 – 5.00)	< 0.001

P values indicate differences between discharge and dead patients. P < 0.05 was considered statistically significant.

Table 3
Treatment of patients with COVID-19 during hospitalization.

	ALL (n = 111)	Discharge (n = 93)	Deterioration (n = 18)	p value
Antiviral therapy	111 (100.0 %)	93 (100.0 %)	18 (100.0 %)	NA
Antibiotic therapy	107 (96.4 %)	89 (95.7 %)	18 (100.0 %)	0.610
Use of corticosteroid	30 (27.0 %)	14 (15.1 %)	16 (88.9 %)	< 0.001
Use of intravenous immunoglobulin	39 (35.1 %)	30 (32.3 %)	9 (50.0 %)	0.181
mechanical ventilation	18 (16.2 %)	0	18 (100 %)	< 0.001
extracorporeal membrane oxygenation	1 (0.9 %)	0	1 (5.6 %)	0.011

P values indicate differences between discharge and dead patients. P < 0.05 was considered statistically significant.

Table 4
Clinical and laboratory characteristics by CRP quartiles.

		Quartiles for CRP (mg/L)				
	ALL (n = 111)	< 3.14 (n = 28)	3.14 – 11.30 (n = 28)	11.30 – 39.00 (n = 28)	> 39.00 (n = 27)	p value
Age (years)	38.00 (32.00 – 57.00)	32.00 (29.25 – 37.00)	37.00 (31.00 – 49.25)	39.50 (33.25 – 60.25)	57.00 (44.00 – 66.00)	< 0.001
Sex (male/female)	46/65	6/22	12/16	12/16	11/16	0.275
Comorbidity						
Hypertension	15 (13.51 %)	0	4 (14.29 %)	2 (7.14 %)	9 (33.33 %)	0.002
Diabetes	14 (12.61 %)	0	2 (7.14 %)	4 (14.29 %)	8 (29.63 %)	0.006
symptoms						
Dyspnoea	18 (16.22 %)	3 (10.71 %)	4 (14.29 %)	0	11 (40.74 %)	< 0.001
Laboratory Findings						
Lymphocyte count (G/L)	1.20 (0.83 – 1.62)	1.53 (1.24 – 1.86)	1.27 (0.92 – 1.66)	1.08 (0.88 – 1.38)	0.60 (0.50 – 0.96)	< 0.001
Liver dysfunction	33 (29.73 %)	5 (17.86)	7 (25.00 %)	8 (28.57 %)	13 (48.15 %)	0.088
IL-6 (pg/mL)	6.37 (3.61 – 13.73)	3.06 (2.64 – 3.78)	4.70 (3.33 – 7.63)	7.91 (4.95 – 13.78)	25.82 (9.72 – 61.09)	< 0.001
IL-10 (pg/mL)	4.23 (3.49 – 5.10)	3.61 (3.12 – 4.11)	3.93 (2.89 – 4.61)	4.64 (3.56 – 6.46)	4.83 (4.27 – 6.92)	0.015
Numbers of pulmonary lobe involved	4.00 (2.00 – 5.00)	2.00 (1.00 – 3.75)	4.00 (2.00 – 5.00)	4.00 (2.00 – 5.00)	5.00 (5.00 – 5.00)	< 0.001
Prognosis						< 0.001
Discharge	93 (83.78 %)	28 (100 %)	27 (96.43 %)	27 (96.43 %)	11 (40.74 %)	
Deterioration	18 (16.22 %)	0	1 (3.57 %)	1 (3.57 %)	16 (59.26 %)	

proportion of COVID-19 patients with hypertension was significantly increased in those with poor prognosis. Molecular modeling revealed that the receptor binding domain (RBD) of SARS-CoV-2 has a stronger interaction with angiotensin converting enzyme 2 (ACE2) [11]. ACE2 could be up-regulated by ACE inhibitors (ACEI) or blockade of Angiotensin II Receptors (ARB) in liver and heart [12,13]. Thus, an increased entry of coronaviruses into host cells might be found in COVID-19 patients complicated with hypertension taking ACEI or ARB, resulting in the poor prognosis.

Lymphopenia is a common feature of coronavirus infection [5,9,10]. Lymphocyte apoptosis directly induced by coronaviruses might be the major cause of lymphopenia [14,15]. Yang et al. observed that as the SARS patients improved, T lymphocyte counts gradually returned to the normal ranges [16]. Thus, lymphopenia is temporally associated with disease severity [17].

Besides the direct attack from virus, progressive inflammatory injury has been suggested as the possible mechanism in COVID-19 [1]. CRP is a downstream acute phase protein in the innate immune response [18]. It is produced because of the increased synthesis of pro-inflammatory cytokines to activate the immune response [19]. Therefore, serum CRP level has been often used as a laboratory marker of inflammation [18,19]. A few studies indicated that CRP is a predictive factor for disease progression in MERS-CoV- and H1N1- infected patients [20,21]. In this study, we first reported that CRP could also be the predictor for the progression of COVID-19.

In view of the excessive inflammation induced by SARS-CoV-2 infections, corticosteroids are used for the treatment of patients with severe illness to reduce inflammatory-induced lung injury. However, current evidence in patients with SARS and MERS suggests the significant effect of corticosteroids on mortality [22,23]. As different from the extensive anti-inflammatory effect of corticosteroids, the drugs specific for inflammasome/IL-1β/IL-6/CRP axis might show their advantages [24,25]. Thus, CRP might not only be the predictor for the poor prognosis but also an indicator for anti-inflammatory therapy.

There are some limitations in this study. First, this is a single center study with a small sample size. The 95 % CI of OR is relatively large. Second, it is a retrospective study, and the results need to be further verified by prospective studies. Third, we aimed to study the risk factors of prognosis. But the sample size in the poor prognosis group is small. Moreover, we were unable to analyze the differences in clinical characteristics of patients in the poor prognosis group due to the small sample size. Fourth, we missed asymptomatic and mild cases managed at home, and hence our cohort might represent the more severe population of COVID-19. Fifth, a few risk factors such as viral load, viral antibody titers, and cause of death were not available in this study. Sixth, the treatment of these patients was clinically driven and not

Table 5
Risk factors for poor prognosis in COVID-19 patients.

Risk factors		Deterioration (n = 18)	Discharge (n = 93)	Crude OR (95 %CI)	P value for crude OR	Adjusted OR (95 %CI)	P value for adjusted OR
Male sex	No	4	61	1		1	
	Yes	14	32	6.7 (2.1 – 21.9)	0.002	24.8 (1.8 – 342.1)	0.016
Comorbidity	No	3	75	1		1	
	Yes	15	18	20.8 (5.4 – 79.7)	$3..5 \times 10^{-4}$	52.6 (3.6 – 776.4)	0.004
Lymphopenia	No	2	63	1		1	
	Yes	16	30	16.8 (3.6 – 77.8)	9.2×10^{-4}	17.3 (1.1 – 261.8)	0.039
Elevated CRP	No	1	80	1		1	
	Yes	17	13	104.6 (12.8 – 854.5)	4.1×10^{-4}	96.5 (4.6 – 2017.6)	0.003

COVID-19: coronavirus disease 2019; Comorbidity: hypertension, coronary heart disease, diabetes, cerebrovascular disease, chronic obstructive pulmonary disease, chronic hepatitis, and cancer; Lymphopenia: leukocyte count less than 1.1 G/L; Elevated CRP: C-reactive protein more than 39.00 mg/L; CI: confidence interval; OR: odds ratio; CRP: C-reactive protein; Data were calculated by logistic regression adjusted for age, gender, comorbidity, and body temperature, number of pulmonary lobe involved, leukocyte count, neutrophil count, lymphocyte count, monocyte count, alanine aminotransferase, aspartate aminotransferase, C-reactive protein, IL-6, and IL-10 level on admission.

unified standard.

In conclusions, male gender, comorbidity, lymphopenia, and elevated CRP are the risk factors for the poor prognosis in COVID-19 patients. Our findings would facilitate the early identification of high-risk COVID-19 patients, especially in primary hospitals.

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CRediT authorship contribution statement

Jun Zhang: Data curation, Investigation, Methodology, Project administration, Supervision, Writing - review & editing, Validation. **Miao Yu:** Conceptualization, Data curation, Formal analysis, Writing - original draft, Validation. **Song Tong:** Writing - review & editing, Validation. **Lu-Yu Liu:** Writing - review & editing, Validation. **Liang-V. Tang:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Supervision, Writing - original draft, Validation.

Declaration of Competing Interest

None.

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