Research Square

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Death Risk Analysis for Patients With Severe COVID-19 Pneumonia

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Abstract

Background: Coronavirus Disease 2019 (COVID-19) is currently a global pandemic. Information about the death predicting of severe COVID-19 is not clear.

Methods: 151 in-patients from January 23th to March 8th 2020 were divided into severe and critically severe group, as well as survival and death group. The analysis of differences of clinical and imaging data were performed between groups. The logistic regression analysis of factors associated with death in COVID-19 were conducted, and the prediction model of death risk was developed.

Results: Many clinical and imaging indices were significantly different between groups, including the age, the epidemic history, the past medical history, the duration of symptoms prior to admission, blood routine, inflammatory related factors, Na^+, myocardial zymogram, liver and renal function, coagulation function, fraction of inspired oxygen and complications. The proportion of patients in imaging stage III and comprehensive CT scores was increased significantly in death group. The area under receiver operating characteristic curve of the prediction model was 0.9593.

Conclusions: The clinical and imaging data reflect the severity of COVID-19 pneumonia. The prediction model of death risk might be a promising method to help clinicians to quickly identify and screen potential individuals who had a high-risk of death.

Introduction

Coronavirus disease 2019 (COVID-19), pathogenic agent of which is severe acute respiratory syndrome corona virus 2 (SARS-COV-2), is currently a global pandemic. SARS-COV-2 is a novel betacoronavirus belonging to the sarbecovirus subgenus of Coronaviridae family, which is closely related to severe acute respiratory syndrome coronavirus (SARS-CoV) and middle east respiratory syndrome coronavirus (MERS-CoV). It can lead to respiratory symptoms or severe pneumonia symptoms [1]. According to the estimation of the World Health Organization, 14% patients with SARS-COV-2 infection are severe type, requiring hospitalization and 5% are critical severe, requiring intensive care [2, 3]. The mortality rate of SARS-COV-2 infected patients could be as high as 4% [2], which is much greater than that of seasonal influenza.

A study on the epidemiological characteristics of 72314 cases in China pointed out that SARS-COV-2 was highly infectious, but most patients were with mild clinical performances [4]. The death cases were often more than 60 years old and suffering from some basic diseases such as hypertension, cardiovascular disease and diabetes. Furthermore, a few severe patients rapidly developed to acute respiratory distress syndrome (ARDS) and died from multiple organs failure [5]. The latest biopsy samples from autopsy of a patient with severe illness demonstrated diffuse alveolar damage [6]. Additionally, the inconsistence existed in clinical and imaging performances of patients with COVID pneumonia and diversity imaging features might exist in a certain clinical stage of the disease [7–9]. A few studies[10–14] summarized the comprehensive clinical, laboratory and / or imaging findings of severe and critically severe patients,

which is of great importance for clinicians to adjust the treatment plan and afford clues to predict the death. Therefore, clinical and imaging evidence of severe and clinical severe COVID-19 patients need to be further explored. And it is also urgent to explore the risk factor of death for the severe and critically severe patients, in the international environment of many countries still in, or entering, the pandemic.

The purpose of this study was to conclude the clinical and imaging characteristics and to develop a model for predicting the risk of death in patients with severe or critically severe COVID-19 pneumonia.

Methods

Patients enrollment

This was a multicenter, retrospective clinical study that was performed at 6 hospitals in Jiangsu and 1 hospital in Wuhan, China. 151 in-patients (104 severe and 47 critical severe) with COVID-19 pneumonia were included from January 23th to March 8th 2020. All the cases were confirmed by reverse transcription-polymerase chain reaction (RT-PCR), and conformed with following diagnosis criteria: Severe type, fulfill any one of the following conditions 1) respiratory distress, respiratory rate (RR) ≥ 30 times per minute, 2) resting state oxygen saturation (SaO2) ≤ 93%, or 3) oxygenation index (calculated by partial pressure of oxygen /fraction of inspired oxygen (FiO2)) ≤ 300mmHg (1mmHg = 0.133kPa); Critically severe type, fulfill any one of the following conditions 1) respiratory failure and mechanical ventilation needed, 2) shock, 3) concomitant failure of other organs. There were respectively 104 patients diagnosed as severe type and 47 as critical severe type COVID-19 pneumonia. In addition, 114 patients were divided into survival group and 37 into death group according to their clinical outcome. This multicenter research was approved by the institutional review board at each study center, and informed consent was obtained from the patients or their surrogates.

Clinical Data

Epidemic history, past medical history, symptoms and signs, as well as age and gender, were recorded. The detailed outcomes of initial laboratory examinations during the severe course were also recorded, containing blood routine, infection related factors, serum ion concentration, myocardial zymogram, liver and kidney function test, coagulation function test, RR, blood gas analysis and complications. Diagnostic criteria of cardiac injury: the serum troponin is the most important index, and its confidence interval greater than 95% of the normal value indicates myocardial damage, and the increase of other indexes can also indicate myocardial damage, in the order of importance: creatine kinase isoenzyme, creatine kinase, lactic dehydrogenase (LDH) [15]. Diagnostic criteria for renal injury: estimated glomerular filtration rate (eGFR) was calculated based on serum creatinine, and it was defined as impaired renal function when eGFR□60 ml/min [16]. The score of the past medical history was determined by additions of these items if any (3 for malignant tumor, 2 for benign tumor, renal or liver malfunction, 1 for Chronic obstructive pulmonary disease, hypertension, diabetes or others).

Imaging Data

At the beginning of severe course, the initial imaging (138 patients underwent chest CT and 13 underwent chest radiograph) was analyzed, among which 76 patients were with follow-up CT examination and 31 patients were with follow-up chest radiograph examination. The scanning parameters for CT were as following: tube voltage 120kV, tube current 110mA, pitch 1.0, rotation time ranging from 0.5s to 0.75s, slice thickness 5mm, with 1mm or 1.5mm section thickness for axial, coronal and sagittal reconstructions. The parameters for chest radiograph were as following: the flat panel detector was attached to the patients' chest, and the voltage and current were 120kV and 200mA, respectively. The chest imaging of 151 patients were analyzed by two experienced attending radiologists, who were blinded to the clinical information, and separately evaluated the imaging and recorded the severity. The chest CT imaging and chest radiograph were classified into mild (stage I), progressed (stage II) and severe stage (stage III), according to the scope of lung field involved, with mild stage less than 25%, progressed stage 26–50% and severe stage more than 50%. The CT score of ground-glass opacity (GGO), consolidation, and the comprehensive score of inflammatory pulmonary infiltration were analyzed quantitatively using a radiologic scoring system ranging from 0–25 points, which was an adaptation of the method previously used to describe idiopathic pulmonary fibrosis and SARS [17]. Each lung lobe was evaluated by 0–5 points, on the basis of the area involved, with score 0 for normal performance, 1 for less than 5% of lung lobe areas involved, 2 for 6–25%, 3 for 26–50%, 4 for 51–75%, and 5 for more than 75%. A total score was eventually calculated via the addition of the score of each lobe.

Statistical analysis

Mann-Whitney U test and two-sample T test were used respectively for non-normal distributed and normal distributed data to compare the continuous variables and Pearson Chi-square test was used to compare the categorical variables, between severe and critically severe group, and between survival and death group, by statistical analysis system software (SAS ver. 9.4, SAS Institute Inc., Cary, NC). Then, univariate and multivariable logistic regression analysis were conducted, and the prediction model for mortality in patients with severe COVID-19 pneumonia was developed. Finally, the model was tested by the receiver operating characteristic (ROC) curve. A P value less than 0.05 was considered statistically significant. The mean value of the continuous variables in normal distribution was recorded as Mean (SD) and the mean value of non-normal distributed data was recorded as Median (IQR). The categorical variables were recorded as count and percentage.

Results

Clinical Features

The clinical data of 151 patients with severe COVID-19 pneumonia and results of group comparison were shown in Table 1. There were 91 (60.26%) men and 60 (39.74%) women included, with mean age 62.47 (13.39) years old (range 26–92 years). The average age of patients in survival group 58.52 (13.99) years was significantly lower than that of patients in death group 68.54 (10.83) years ($P < 0.001$). The epidemic

history, past medical history, score of past medical history and the duration of symptoms prior to admission were different between survival and death group.

Table 1
Clinical data of 151 patients with severe COVID-19 pneumonia and group comparison

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
Sex		0.120	2.413	0.374	0.789
Male	91 (60.3%)				
Female	60 (39.7%)				
Age (years)	60.97 (13.94)	0.130	-1.523	< 0.001	-3.984
Epidemic history	125 (82.8%)	0.418	1.744	0.007	9.823
Past medical history	85 (56.3%)	0.368	0.812	0.049	3.892
Score of past medical history	1.00 (0, 2.00)	0.561	0.581	0.014	2.457
Time interval[a] (days)	10.00 (5.00, 15.00)	0.185	1.324	0.026	2.223
Time interval[b] (days)	10.00 (7.00, 15.00)	0.400	0.841	0.105	1.621
Time interval[c] (days)	3.00 (0, 9.00)	0.697	0.389	0.136	-1.492
Symptoms					
Fever		0.495	1.408	0.036	6.644
< 37	51 (33.8%)				
37.0–38.0	48 (31.8%)				
> 38.0	52 (34.4%)				
Cough	112 (74.2%)	0.575	1.107	0.596	1.036

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
Dyspnea	76 (50.3%)	0.026	4.974	0.098	2.744
Fatigue	70 (46.4%)	0.436	0.608	0.066	3.383
Throat irritation	19 (12.6%)	0.964	0.002	0.929	0.008
Diarrhea	37 (24.5%)	0.304	1.058	0.977	0.001
Headache	13 (8.6%)	0.732	0.117	0.644	0.214
Muscle soreness	19 (12.6%)	0.102	2.675	1.000	< 0.001
Nausea and vomiting	11 (7.3%)	0.959	0.003	0.887	0.020
Blood routine					
WBC (×10^9 /L)	6.40 (4.38, 9.26)	< 0.001	3.888	0.001	3.250
Neutrophil (×10^9 /L)	4.86 (3.12, 8.84)	< 0.001	3.658	0.003	3.007
Neutrophil ratio (%)	76.45 (11.64)	0.361	0.923	None	None
Lymphocyte (×10^9 /L)	0.86 (0.56, 1.12)	< 0.001	-4.327	< 0.001	-4.299
Lymphocyte ratio (%)	16.34 (9.84)	0.358	-0.928	None	None
RBC (×10^{12}/L)	4.21 (3.98, 4.77)	0.532	-0.629	0.904	0.891
Platelet (×10 □/L)	198.50 (133.00, 246.50)	0.091	-1.690	0.071	-1.802
Infection related factors					
CRP (mg/L)	61.50 (22.35, 104.15)	< 0.001	4.230	0.001	3.398

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
ESR (mm/H)	28.0 (10.00, 53.00)	0.022	2.288	0.013	2.485
Procalcitonin (ng/ml)	0.10 (0.08, 0.32)	< 0.001	3.635	0.008	2.648
IL-6 (pg/ml)	25.43 (6.83, 52.55)	0.009	2.620	0.003	2.949
IL-8 (pg/ml)	19.75 (10.58, 30.63)	0.016	2.411	0.029	2.186
IL-10 (pg/ml)	5.75 (4.00, 10.98)	0.001	3.358	< 0.001	3.816
Serum ion concentration					
Na^+ (mmol/L)	138.20 (134.60, 141.30)	0.009	2.630	0.021	2.311
K^+ (mmol/L)	4.26 (3.68, 4.56)	0.580	-0.554	0.282	1.075
Cl^- (mmol/L)	100.25 (96.95, 103.63)	0.055	1.919	0.059	1.891
Ca^{2+} (mmol/L)	2.06 (0.15)	0.481	0.711	0.626	-0.492
Myocardial zymogram					
CK (U/L)	108.00 (65.25, 147.75)	0.266	-1.111	0.647	0.458
CK-MB (ng/ml)	3.00 (1.05, 12.00)	0.379	-0.880	0.483	-0.702
Myoglobin (ng/ml)	50.00 (45.00, 125.90)	0.003	3.020	< 0.001	5.758
Troponin (ng/ml)	4.45 (0.05, 23.25)	< 0.001	4.305	< 0.001	7.119

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
LDH (U/L)	307.00 (195.00, 509.00)	< 0.001	4.204	< 0.001	5.020
Liver function					
Total bilirubin (umol/L)	11.10 (7.80, 14.70)	0.003	2.985	0.055	1.917
Albumin (g/L)	33.90 (30.80, 36.80)	< 0.001	-4.752	< 0.001	-3.787
AST (U/L)	31.50 (22.00, 45.93)	0.035	2.105	0.009	2.613
ALT (U/L)	27.00 (18.00, 46.00)	0.766	0.298	0.209	1.257
Renal function					
eGFR (ml/min)	84.23 (27.83)	0.640	0.470	0.008	2.722
Serum creatinine (umol/L)	73.50 (57.00, 89.23)	0.233	1.192	0.006	2.725
Serum urea nitrogen (mmol/L)	5.40 (3.90, 9.05)	0.006	2.724	< 0.001	4.839
Coagulation function					
PT (sec.)	14.00 (12.75, 15.00)	< 0.001	4.523	< 0.001	5.117
APTT (sec)	38.75 (34.48, 43.78)	0.034	2.122	0.001	3.362
Fibrinogen (g/L)	4.63 (3.00, 6.04)	0.015	2.434	0.356	0.922
D-dimer (ng/mL)	2.50 (0.87, 25.00)	0.081	1.744	0.309	1.018

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
INR	1.11 (1.05, 1.20)	< 0.001	3.900	0.003	3.019
Respiratory rate (times/min)	21.00 (20.00, 26.00)	0.017	2.391	0.065	1.848
Blood gas analysis					
PaO_2 (mmHg)	76.85 (62.85, 95.63)	0.199	1.309	0.104	-1.673
PCO_2 (mmHg)	37.94 (6.70)	0.493	-0.698	None	None
SaO_2 (%)	96.00 (91.00, 98.00)	0.023	-2.280	0.099	-1.651
Oxygenation index (mmHg)	286.38 (71.41)	1.000	< 0.001	None	None
FiO_2	0.41 (0.30, 0.81)	< 0.001	4.246	< 0.001	4.321
Complications					
Cardiac injury	55 (36.4%)	0.293	1.107	< 0.001	24.245
Liver injury	20 (13.2%)	0.050	3.831	0.010	6.590
Kidney injury	26 (17.2%)	0.375	0.788	0.188	1.736
ARDS	43 (28.5%)	< 0.001	31.976	< 0.001	62.369
Septic shock	31 (20.5%)	< 0.001	33.751	< 0.001	66.463

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

Items	Mean (SD) Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*T/Z/x^2 value*	*P value*	*T/Z/x^2 value*
DIC	21 (13.9%)	< 0.001	23.107	< 0.001	57.389
AKI	31 (20.5%)	< 0.001	13.205	< 0.001	33.760

a: The duration of symptoms prior to admission; b: The duration of symptoms prior to severe phase; c: The duration of severe illness prior to initial CT scan; WBC: white blood cell; RBC, red blood cell; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IL-6: interleukin-6; IL-8: interleukin-8; IL-10: interleukin-10; CK: creatine kinase; CK-MB: creatine kinase isoenzyme; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alamine aminotransferase; eGFR: estimated glomerular filtration rate; PT: prothrombin time; APTT: activated partial thromboplastin time; INR: international normalized ratio; PaO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon-dioxide; SaO_2: oxygen saturation; FiO_2: fraction of inspiration O_2: ARDS, acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury. T/Z/x^2 value corresponding to Mean (SD), Median (IQR) and n (%) were respectively from two-sample T test, Mann-Whitney U test and Pearson Chi-square test. “None” means that the *P* value cannot be calculated due to too many missing values of the variate.

The count of white blood cell and neutrophil, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), procalcitoni, interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-10 (IL-10), Na^+, myoglobin, troponin, LDH, aspartate aminotransferase, serum urea nitrogen, prothrombin time (PT), activated partial thromboplastin time (APTT), international normalized ratio (INR), FiO2, the occurrence rate of ARDS, septic shock, disseminated intravascular coagulation (DIC) and acute kidney injury (AKI) were lower, while the count of lymphocytes and albumin were higher in severe and survival group than those in critically severe and death group ($P < 0.05$). The percentage of patients with dyspnea, total bilirubin, fibrinogen and RR were lower, while the SaO2 was higher in the severe group than those in critically severe group ($P < 0.05$). The serum creatinine, the occurrence rate of cardiac injury and liver injury were lower, while the proportion of patients with moderate and high fever and estimated glomerular filtration rate were higher in survival group than those in the death group ($P < 0.05$).

Imaging findings

As showed in Table 2, among the 151 severe and critically severe patients, 6 (3.97%) patients were diagnosed as stage I (Fig. 1), 68 (45.03%) were stage II (Fig. 2), and 77 (50.99%) were stage III (Fig. 3) on chest CT or chest radiograph images. On CT images, 116 (84.06%) patients were with the whole lung involved. The lesions of 83 (60.14%) patients on chest CT were mainly peripherally distributed. The proportion of patients with stage III in the death group was significantly higher than that in the survival group (73.0% vs. 43.9%, $P < 0.05$). There was significant difference in comprehensive score not only between severe and critically ill group, but also between survival and death group ($P < 0.05$).

Table 2
Imaging data of 151 patients with severe COVID-19 pneumonia and group comparison

Items	Median (IQR) n (%)	Severe vs. critically severe group		Survival vs. death group	
		P value	*Z/x^2 value*	*P value*	*Z/x^2 value*
Imaging stage		0.185	3.379	0.007	9.828
I	6 (4.0%)				
II	68 (45.0%)				
III	77 (51.0%)				
Number of lung lobes involved		0.162	6.538	0.701	2.190
One lobe	2 (1.4%)				
Two lobes	5 (3.6%)				
Three lobes	7 (5.1%)				
Four lobes	8 (5.8%)				
Five lobes	116 (84.1%)				
Distribution of pulmonary lesions		0.202	1.631	0.777	0.080
Peripheral	83 (60.1%)				
Non-peripheral	55 (39.9%)				
Comprehensive score	17.00 (11.00, 20.00)	0.035	2.103	0.021	2.303
GGO score	14.00 (8.75, 17.25)	0.054	1.930	0.054	1.924
Consolidation score	5.00 (3.00, 9.00)	0.680	0.412	0.851	0.188
Follow-up		0.309	2.347	0.735	0.615
No obvious change	8 (8.5%)				
Absorption	66 (70.2%)				
Progress	20 (21.3%)				

GGO: ground-glass opacity. *Z/χ^2* value corresponding to Median (IQR) and n (%) were respectively from Mann-Whitney U test and Pearson Chi-square test.

94 patients had chest CT and/or chest radiograph follow-up, of which 8 patients showed no obvious change, 66 patients showed absorption and 20 patients showed progress of pulmonary lesions. The proportion of patients with absorption during imaging follow-up was higher (71.8% vs. 63.6%), while the proportion of patients with progress was lower (19.7% vs. 27.3%) in the survival group than that in the death group. However, there were no significant differences between the two groups ($P > 0.05$).

Logistic regression analysis and prediction model

The univariate logistic regression analysis of factors associated with death in COVID-19 was shown in Table 3. The value of odds ratio estimates in patients with DIC was the highest (59.105), followed by septic shock (37.500) and myocardial injury (34.500). Multivariate logistic regression analysis of factors associated with death in COVID-19 were shown in Table 3. The death prediction model of risk factors for a severe patient was written as:

$$\ln\frac{p}{1-p} = -10.6756 + 0.0898age + 2.5089\text{cardiac\_injury} + 1.4740\text{AKI} + 3.8355\text{ARDS}$$

$$\text{or } p = \frac{e^{-10.6756+0.0898\,age+2.5089\,\text{cardiac\_injury}+1.4740\text{AKI}+3.8355\text{ARDS}}}{1+e^{-10.6756+0.0898\,age+2.5089\,\text{cardiac\_injury}+1.4740\text{AKI}+3.8355\text{ARDS}}}$$

Table 3
Logistic regression analysis of risk factors for death prediction

Items	Odds Ratio Estimates	95% Wald Confidence Limits	*P*
Univariate analysis			
Sex (Male/ Female)	1.404	0.663–2.969	0.375
Age (years)	1.061	1.028–1.095	0.001
Past medical history	2.203	0.995–4.879	0.051
Score of past medical history	1.363	1.040–1.788	0.025
Duration of symptoms prior to admission (days)	1.052	1.001–1.106	0.044
Duration of symptoms prior to severe illness (days)	2.132	0.945–4.807	0.768
Fever	0.328	0.369–0.833	0.005
Cough	0.922	0.577–1.474	0.735
Dyspnea	1.336	0.885–2.015	0.168
Diarrhoea	0.942	0.398–2.229	0.892
Headache	0.535	0.113–2.533	0.431
Fatigue	2.091	0.983–4.448	0.056
Muscle soreness	1.116	0.373–3.339	0.844
WBC ($\times 10^9$ /L)	3.728	1.595–8.714	0.002
Neutrophil ($\times 10^9$ /L)	3.927	1.809–8.525	<0.001
Lymphocyte ($\times 10^9$ /L)	0.152	0.051–0.450	<0.001
Cardiac injury	34.500	11.065-107.568	<0.001
Live injury	2.103	0.983–4.498	0.055
Kidney injury	4.208	1.731–10.230	0.002
PT (sec.)	5.893	1.702–20.411	0.005
APTT (sec.)	3.040	1.280–7.222	0.012
Fibrinogen (g/L)	2.407	0.972–5.964	0.058

WBC: white blood cell; PT: prothrombin time; APTT: activated partial thromboplastin time; CRP: C-reactive protein; ARDS: acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury.

Items	Odds Ratio Estimates	95% Wald Confidence Limits	*P*
CRP (mg/L)	4.617	1.326–16.082	0.016
ARDS	29.592	10.919–80.199	<0.001
Septic shock	37.500	12.835-109.566	<0.001
DIC	59.105	12.676-275.596	<0.001
AKI	11.013	4.491–27.006	<0.001
Comprehensive CT score	1.079	1.007–1.156	0.031
Consolidation score	1.030	0.933–1.138	0.558
Imaging stage	2.912	1.366–6.206	0.006
Number of lung lobes involved	1.772	0.755–4.157	0.189
Multivariate analysis			
Age (years)	1.094	1.024–1.168	0.008
Cardiac injury	12.291	2.872–52.607	<0.001
AKI	4.367	1.055–18.081	0.042
ARDS	46.319	8.959–239.471	<0.001

WBC: white blood cell; PT: prothrombin time; APTT: activated partial thromboplastin time; CRP: C-reactive protein; ARDS: acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; AKI: acute kidney injury.

The percent concordant of the prediction model was 96.1%. The ROC curve of the prediction model was shown in Fig. 4 and the area under curve of the ROC curve was 0.9593.

Discussion

COVID-19 is a novel infectious disease, characterized by high transmissibility and serious harmfulness. A few patients with severe course of disease tend to have severe clinical symptoms, who may rapidly progress into ARDS and need the aids of intensive care unit [18]. Hence, it is essential to closely monitor the condition of patients, by dynamically monitoring the alteration of symptoms and laboratory examinations, the change of the chest imaging performances, which are helpful for the evaluation of the disease severity and to adjust treatment plan timely.

There were some characteristic clinical features pertaining to the severe disease course of SARS-COV-2 infected. The past medical history had an effect on disease mortality, which confirmed by the reports from Sohrabi et al [19], Guan et al [20] and Jordan et al [3]. In present study, the mean age of death cases was approximately 10 years older than that of survivors, which was similar to the previous study [21]. The

gender prevailing of patients with severe COVID-19 was obvious, almost 3: 2 for male-female ratio in present study. This was in consistence with Chen's study, suggested that older men were more likely to be infected with SARS-COV-2, resulting in severe and even fatal respiratory diseases such as ARDS [5]. In the death group, the duration of symptoms prior to admission was longer than that of survival group, reflecting that the prolonged duration of symptom onset to hospitalization tended to poorer outcomes, which was in consistence with Liang's study [22].

In present study, the main initial symptoms of the severe patients were fever and/or cough. The dyspnea was frequently seen in the severe course of the patients with COVID-19 pneumonia, especially in critically severe patients, due to the severe lung lesions of the pneumonia. The incidence of ARDS in critically severe and death group was significantly higher than that in severe and survival group, respectively. The RR in critically severe patients was significantly higher than that in severe patients, as well as the SaO2 and FiO_2, which may be due to mechanical ventilation. As to the blood routine, increased leukocyte and neutrophil counts, decreased lymphocytes count and ratio were remarkable features, especially for critically severe group and death group. Wang et al firstly uncovered the continuous increase of neutrophil counts in dead cases [23]. It may be related to cytokine storm induced by virus invasion. And lymphopenia suggested SARS-COV-2 might mainly target at lymphocytes and lead to the progression of the disease [5].

The infection related factors, including CRP, ESR, procalcitonin, IL-6, IL-8 and IL-10, were increased in the severe patients, especially in critically severe patients and death cases. The study from Ulhaq et al suggested that continuous measurement of circulating IL-6 levels may be of great significance in identifying disease progression in patients infected with COVID-19 [24]. A retrospective study suggested that elevated levels of IL-6 was related to the high mortality of COVID-19 infection [25]. A significantly higher incidence of septic shock and DIC was seen in critically severe and death group. This may be due to the imbalance of thrombin production caused by the activation of vascular endothelium, platelets and white blood cells, which occurred locally and systematically in the lung system of patients with severe pneumonia, resulting in fibrin deposition, tissue damage and microangiopathy [26]. It could be aggravated by the occurrence of septic shock [27, 28]. It was reported that most of death cases and very few survivors have evidence of DIC, which occurred frequently in the deterioration of COVID-19 pneumonia and was often associated with mortality [29]. It also suggested that clinicians needed to be vigilant to identify the presence of DIC, especially in patients who had already experienced septic shock.

There was some significant relationship between multiple organs injury and mortality. In critically severe and death patients, myoglobin, troponin, LDH and the incidence of cardiac injury were more higher than those in non-death patients, which was similar to the results of some previous studies on the relationship between the severity of illness and myocardial injury in patients with COVID-19, and was consistent with the correlation study between heart injury and death after SARS-CoV-2 infection [30, 31]. Recent studies on COVID-19 had shown that the incidence of liver injury ranges from 14.8–53%, with the decreased albumin level in critically ill patients, and the incidence of liver injury might reach as high as 78.0% in the death cases of COVID-19 [32]. In this study, the incidence of liver injury in critically severe and death

group was significantly increased compared to severe and survival group. This demonstrated that liver injury was related to the severity of the disease and mortality, which may be due to the cytokine storm, or the drug-induced liver damage [32, 33]. In the present study, the eGFR, serum creatinine and serum urea nitrogen levels in the death group were significantly higher than those in the survival group, and there was a significant prevalence with AKI of patients in both the critically severe group and the death group. It was consistent with the study of Cheng et al, which showed that the development of AKI during hospitalization in patients with COVID-19 was related to in-hospital mortality [34].

The coagulation function and the serum Na^+ concentration changed in the severe course of COVID-19 pneumonia. Recently, the coagulation function was concerned and some related indices were studied between severe and non-severe patients [18, 35]. In this study, these indices were further compared between severe and critically severe patients, and between survival and death patients. PT, APTT, INR and fibrinogen level were related to the severity of the disease, and the former three might be related to the mortality. According to previous study [36], hypernatremia was a common electrolyte disorder, which was related to long-term hospitalization and death, and was more common in critically ill patients. Abnormal changes in the central nervous system and mental state may be the causes of hypernatremia, while the digestive tract or urinary system disorder cannot be ruled out [32]. In addition, it may also be related to a large number of intravenous supplements of sodium-containing fluids.

As to the imaging performances, multiple lung lobes were involved in 98.6% patients, and whole lung lobes were involved in 84.06% patients. The proportion of patients in stage III increased significantly in death group, as well as comprehensive CT imaging scores in critically severe and death group. Our results showed that the severity of CT findings was consistent with the severity of clinical course of the disease, as suggested by previous study [37]. Li et al [38] found that the development pattern of COVID-19 on CT images was similar to that of SARS or MERS. There were some common imaging features, so the final diagnosis had to be combined with the clinical manifestation, epidemic history and laboratory examination. However, the advantage of convenient and rapid CT examination was irreplaceable. A study about critically ill patients with SARS-CoV-2 pneumonia demonstrated that early or repeated radiological examination is helpful to screen patients with SARS-CoV-2 pneumonia [39].

The previous studies referred to the mortality risk, calculated overall probability based on the infection and confirmed population [40, 41]. However, the individual rough risk of death was important, especially in severe and critically severe patients, which might influence the treatment plan and the response of clinicians or medical institutions. In the univariate logistic regression analysis, DIC showed as the best predictor (nearly 59 times of the death risk for the patients without DIC), followed by septic shock and cardiac injury. The prediction model included evidence of patient's age, cardiac injury, AKI and ARDS, among which the evidence of ARDS was the most powerful predictor. In the current COVID-19 epidemic, this prediction model might be a promising method to help clinicians to quickly identify and screen potential individuals who had a high-risk of death.

There were several limitations of this study. First, the clinical and imaging data of patients were from multiple centers, hence the data were heterogeneous which might affect the statistical results. Additionally, some indices were missing too many values, which lead to that the *P* value could not be calculated in the test of group differences. Second, the initial imaging and follow-up imaging of the patients were lack of uniform standard. Some patients were only with chest X-ray because of the disease severity, and the follow-up interval was not identical. Finally, although both of the percent concordant and the area under curve of the prediction model were in a high level, a larger cohort study might be warranted to validate the accuracy and application value of the prediction model.

Conclusion

The clinical and imaging data reflect the severity of COVID-19 pneumonia and part of them were related to mortality. The prediction model of death risk might be a promising method to help clinicians to quickly identify and screen potential individuals who had a high-risk of death.

Abbreviations

AKI
Acute kidney injury; APTT:Activated partial thromboplastin time; ARDS:Acute respiratory distress syndrome; COVID-19:Coronavirus disease 2019; CRP:C-reactive protein; CT:Computed tomography; DIC:Disseminated intravascular coagulation; ESR:Erythrocyte sedimentation rate; FiO_2:Fraction of inspired oxygen; GGO:Ground-glass opacity; HRCT:High-resolution CT; INR:International normalized ratio; MERS-CoV Middle East respiratory syndrome coronavirus; PT:Prothrombin time; ROC:Receiver operating characteristic; RR:Respiratory rate; RT-PCR:Reverse transcription-polymerase chain reaction; SaO_2:Oxygen saturation; SARS-CoV:Severe acute respiratory syndrome coronavirus; SARS-CoV-2:Severe acute respiratory syndrome coronavirus 2

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the amended Declaration of Helsinki. Independent ethics committees approved this retrospective study [see Additional file 1], and written informed consent was obtained from all patients or their surrogates.

Consent for publication

The consent for publication of chest CT and radiograph images in this study had been obtained from relevant patients.

Availability of data and materials

The datasets used during the current study are available from Yonggang Li on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

YL is the chief investigator of the study. HD and RH made substantial contributions to the study design, data collection and analysis, interpretation of the data, and drafting of the manuscript. MM, YS, JH and NS made substantial contributions to the data collection and analysis, interpretation of the data. DZ and HL made substantial contributions to the conception, study designs, data collection and analysis. All authors read and approved the final manuscript.

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References

1. Tian H Y. 2019-nCoV: new challenges from coronavirus. Zhonghua Yu Fang Yi Xue Za Zhi. 2020;54:235–238.
2. Grech Victor,Unknown unknowns - COVID-19 and potential global mortality. Early Hum. Dev. 2020; 144: 105026.
3. Jordan Rachel E., Adab Peymane., Cheng K K.2020. Covid-19: risk factors for severe disease and death. BMJ. 2020;368:m1198.
4. Zhonghua Liu Xing Bing Xue Za Zhi. Novel Coronavirus Pneumonia Emergency Response Epidemiology Team. The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19) in China. 2020; 41:145–151.

5. Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020; 395:507–513.
6. Xu Z, Shi L, Wang Y, Zhang J, Huang L, Zhang C, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med. 2020; 8:420–422.
7. Yang W, Cao Q, Qin L, Wang X, Cheng Z, Pan A, et al. Clinical characteristics and imaging manifestations of the 2019 novel coronavirus disease (COVID-19):A multi-center study in Wenzhou city, Zhejiang, China. J. Infect. 2020; 80:388–393.
8. Lin C, Ding Y, Xie B, Sun Z, Li X, Chen Z, et al. Asymptomatic novel coronavirus pneumonia patient outside Wuhan: The value of CT images in the course of the disease. Clin Imaging. 2020; 63:7–9.
9. Xu X, Yu C, Qu J, Zhang L, Jiang S, Huang D, et al. Imaging and clinical features of patients with 2019 novel coronavirus SARS-CoV-2. Eur J Nucl Med Mol Imaging. 2020; 47:1275–1280.
10. Tian R, Wu W, Wang C, Pang H, Zhang Z, Xu H, et al. Clinical characteristics and survival analysis in critical and non-critical patients with COVID-19 in Wuhan, China: a single-center retrospective case control study.Sci Rep. 2020;10:17524.
11. Cao Y, Han X, Gu J, Li Y, Liu J, Alwalid O, et al. Prognostic value of baseline clinical and HRCT findings in 101 patients with severe COVID-19 in Wuhan, China.Sci Rep. 2020;10:17543.
12. Li X, Xu S, Yu M, Wang K, Tao Y, Zhou Y, et al. Risk factors for severity and mortality in adult COVID-19 inpatients in Wuhan. J Allergy Clin Immunol. 2020; 146: 110–118.
13. Dellière S, Dudoignon E, Fodil S, Voicu S, Collet M, Oillic PA, et al. Risk factors associated with Covid-19-associated pulmonary aspergillosis in ICU patients: a French multicentric retrospective cohort.Clin Microbiol Infect. 2020; 13:S1198-743X(20)30756-20.
14. Wang JM, Liu W, Chen X, McRae MP, McDevitt JT, Fenyö D. Predictive modeling of morbidity and mortality in COVID-19 hospitalized patients and its clinical implications. medRxiv. 2020.
15. Chinese Society of Cardiology of Chinese Medical Association, Editorial Board of Chinese Journal of Cardiology. 2019 Chinese Society of Cardiology (CSC) guidelines for the diagnosis and management of patients with ST□segment elevation myocardial infarction (in chinese). Chin J Cardiol. 2019; 47(10): 766–783.
16. Kidney disease: improving global outcomes (KDIGO) living kidney donor work group. KDIGO clinical practice guideline on the evaluation and care of living kidney donors. Transplantation. 2017; 101(Suppl 8S): S1-S109.
17. Chang YC, Yu CJ, Chang SC, Galvin JR, Liu HM, Hsiao CH, et al. Pulmonary sequelae in convalescent patients after severe acute respiratory syndrome: evaluation with thin-section CT. Radiology. 2005;236:1067–75.
18. Zhang JJ, Dong X, Cao YY, Yuan YD, Yang YB, Yan YQ, et al. Clinical characteristics of 140 patients infected with SARS-CoV-2 in Wuhan, China. Allergy. 2020; 75: 1730–1741.
19. Sohrabi Catrin., Alsafi Zaid., O'Neill Niamh., Khan Mehdi., Kerwan Ahmed., Al-Jabir Ahmed.,et al. World Health Organization declares global emergency: A review of the 2019 novel coronavirus

(COVID-19). Int J Surg. 2020;76:71–76.

20. Guan Wei-Jie,Liang Wen-Hua,Zhao Yi et al. Comorbidity and its impact on 1590 patients with Covid-19 in China: A Nationwide Analysis.Eur. Respir. J. 2020;55: 2000547.
21. Chen Tao., Wu Di., Chen Huilong., Yan Weiming., Yang Danlei., Chen Guang., et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. BMJ, 2020;368:m1091.
22. Liang WH, Guan WJ, Li CC, Li YM, Liang HR, Zhao Y, Liu XQ, Sang L, Chen RC, Tang CL et al: Clinical characteristics and outcomes of hospitalised patients with COVID-19 treated in Hubei (epicenter) and outside Hubei (non-epicenter): A Nationwide Analysis of China. The European respiratory journal. 2020;55:2000562.
23. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. JAMA. 2020;323:1061–1069.
24. Ulhaq ZS, Soraya GV, Interleukin-6 as a potential biomarker of COVID-19 progression, Medecine et Maladies Infectieuses. 2020;50:382–383.
25. Chen X, Zhao B, Qu Y, Chen Y, Xiong J, Feng J, et al. Detectable serum SARS-CoV-2 viral load (RNAaemia) is closely associated with drastically elevated interleukin 6 (IL-6) level in critically ill COVID-19 patients. bioRxiv. 2020; 71: 1937–1942.
26. Lillicrap David. Disseminated intravascular coagulation in patients with 2019-nCoV pneumonia. J. Thromb. Haemost. 2020;18:786–787.
27. Schmitt Felix Carl Fabian., Manolov Vasil., Morgenstern Jakob., Fleming Thomas., Heitmeier Stefan.,et al. Acute fibrinolysis shutdown occurs early in septic shock and is associated with increased morbidity and mortality: results of an observational pilot study. Ann Intensive Care. 2019; 9:19.
28. Chang Jae C. Sepsis and septic shock: endothelial molecular pathogenesis associated with vascular microthrombotic disease. Thromb J. 2019;17:10.
29. Taylor FB, Toh CH, Hoots WK, et al. Towards definition, clinical and laboratory criteria, and a scoring system for disseminated intravascular coagulation. Thromb Haemost. 2001;86:1327–1330.
30. Mishra Ajay Kumar., Sahu Kamal Kant., Lal Amos., Sargent Jennifer.(2020). Patterns of heart Injury in COVID – 19 and relation to outcome. J. Med. Virol. 2020; 92:1747.
31. Han Huan., Xie Linlin., Liu Rui., Yang Jie., Liu Fang., Wu Kailang., et al. Analysis of heart injury laboratory parameters in 273 COVID-19 patients in one hospital in Wuhan, China. J. Med. Virol. 2020; 92:819–823.
32. Xu Ling., Liu Jia., Lu Mengji., Yang Dongliang., Zheng Xin. Liver injury during highly pathogenic human coronavirus infections. Liver Int. 2020;40:998–1004.
33. Liu C., Jiang Z C., Shao C X., Zhang H G., Yue H M., Chen Z H., et al. [Preliminary study of the relationship between novel coronavirus pneumonia and liver function damage: a multicenter study]. Zhonghua Gan Zang Bing Za Zhi. 2020;28:148–152.

34. Cheng Yichun., Luo Ran., Wang Kun., Zhang Meng., Wang Zhixiang., Dong Lei., et al. Kidney disease is associated with in-hospital death of patients with COVID-19. Kidney Int. 2020;97:829–838.
35. Xiong M, Liang X, Wei YD. Changes in Blood Coagulation in Patients with Severe Coronavirus Disease 2019 (COVID-19): a Meta-Analysis. Br J Haematol. 2020;189: 1050–1052.
36. Ates Ihsan., Özkayar Nihal., Toprak Güvenç., Yılmaz Nisbet., Dede Fatih. Factors associated with mortality in patients presenting to the emergency department with severe hypernatremia. Intern Emerg Med. 2016;11:451–459.
37. Shi H, Han X, Jiang N, Cao Y, Alwalid O, Gu J, et al. Radiological findings from 81 patients with COVID-19 pneumonia in Wuhan, China: a descriptive study. Lancet Infect Dis, 2020;20:425–434
38. Li Y, Xia L. Coronavirus Disease 2019 (COVID-19): Role of Chest CT in Diagnosis and Management. AJR. 2020;215:1–7.
39. Yang X, Yu Y, Xu J, Shu H, Xia J, Liu H, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med. 2020;8: 475–481.
40. Kobayashi Tetsuro., Jung Sung-Mok., Linton Natalie M., Kinoshita Ryo., Hayashi Katsuma., Miyama Takeshi., et al. Communicating the Risk of Death from Novel Coronavirus Disease (COVID-19). J Clin Med. 2020;9:580.
41. Jung Sung-Mok., Akhmetzhanov Andrei R., Hayashi Katsuma., Linton Natalie M., Yang Yichi., Yuan Baoyin.,et al. Real-Time Estimation of the Risk of Death from Novel Coronavirus (COVID-19) Infection: Inference Using Exported Cases. J Clin Med. 2020;9:523.

Figures

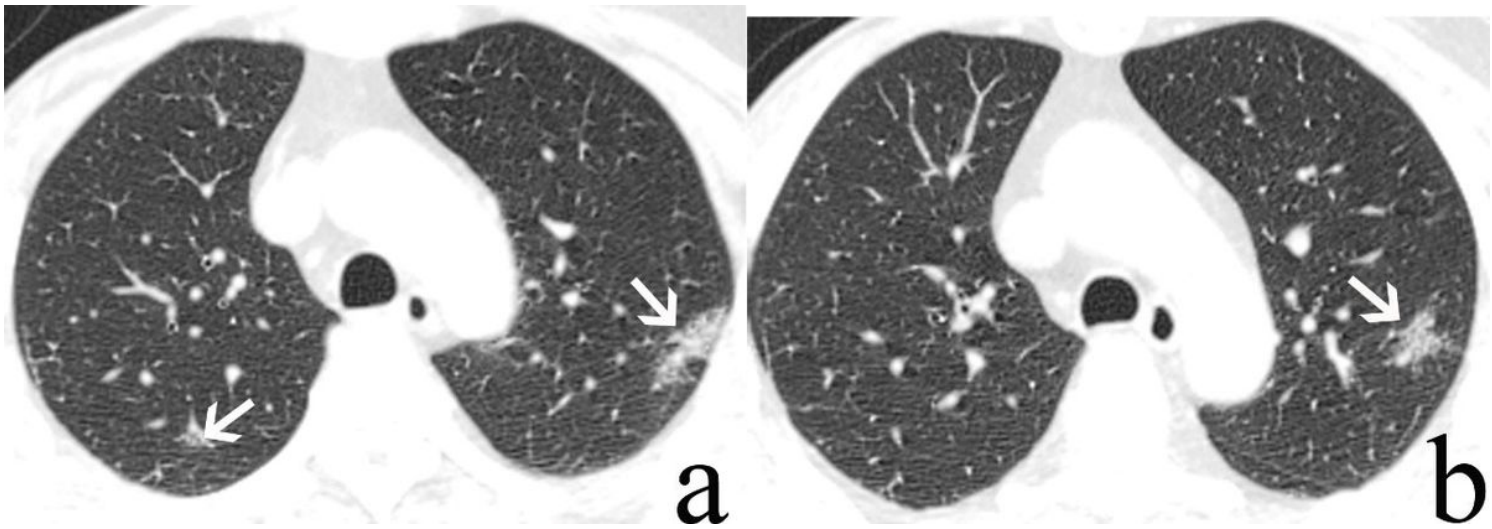

Figure 1

A 25-year-old woman diagnosed with severe COVID-19 on January 30th with diarrhea for 2 days. a-b. Axial chest CT on January 30th showed mild changes (stage I) with irregular high-density lesion in the

left upper lung lobe surrounded by ground-glass opacity (a, b, arrows), and patchy ground-glass opacity in the right upper lung lobe (a, arrow).

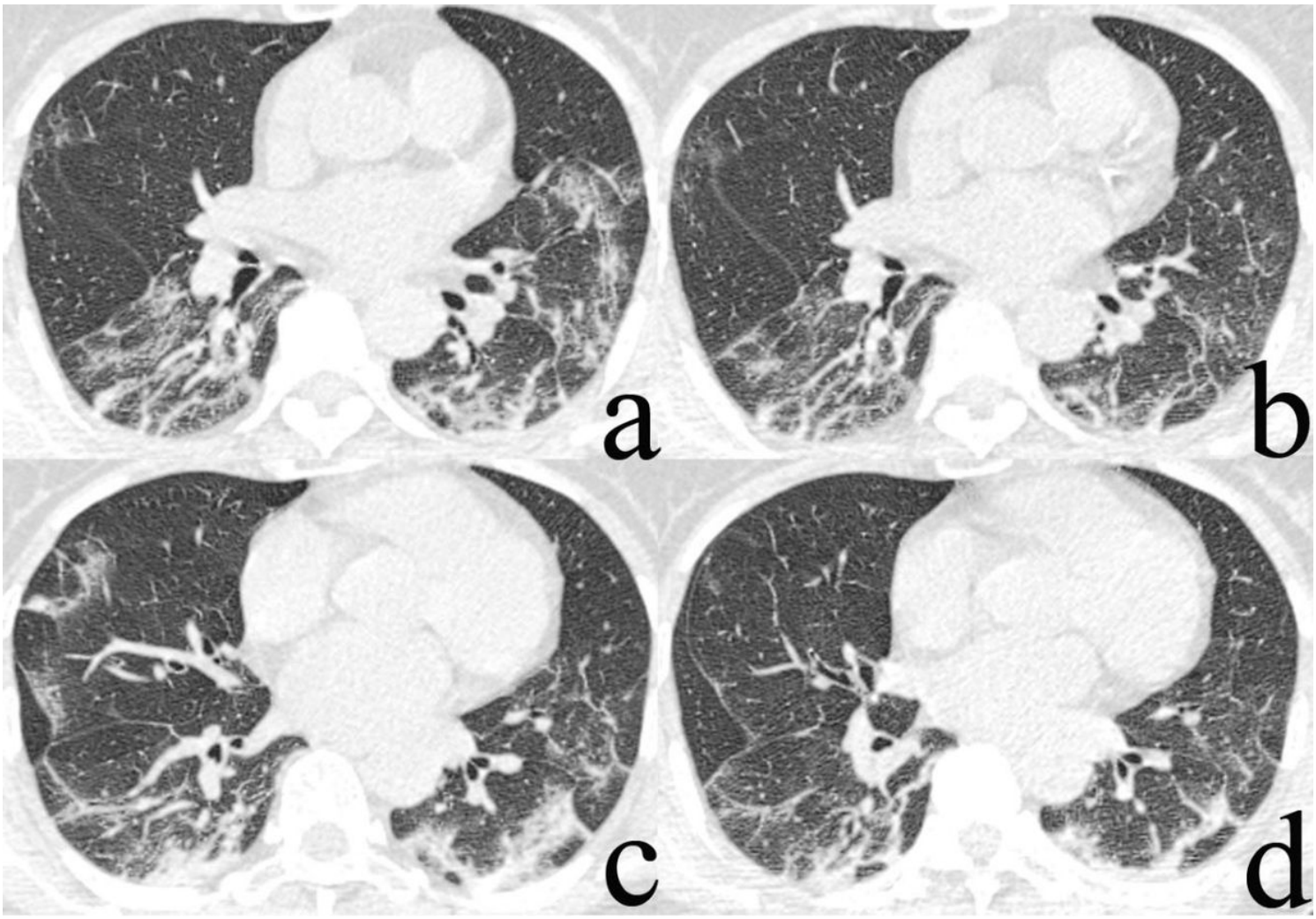

Figure 2

A 64-year-old woman diagnosed with severe COVID-19 on January 31th with vomiting and anorexia. a, c. Axial chest CT on February 1st showed progressed performances (stage II) with multiple lesions, including ground-glass opacity, consolidation and fibrosis, mainly distributed in the lower lung lobes. b, d. Axial chest CT on February 4th showed mild absorption of ground-glass opacity and consolidation.

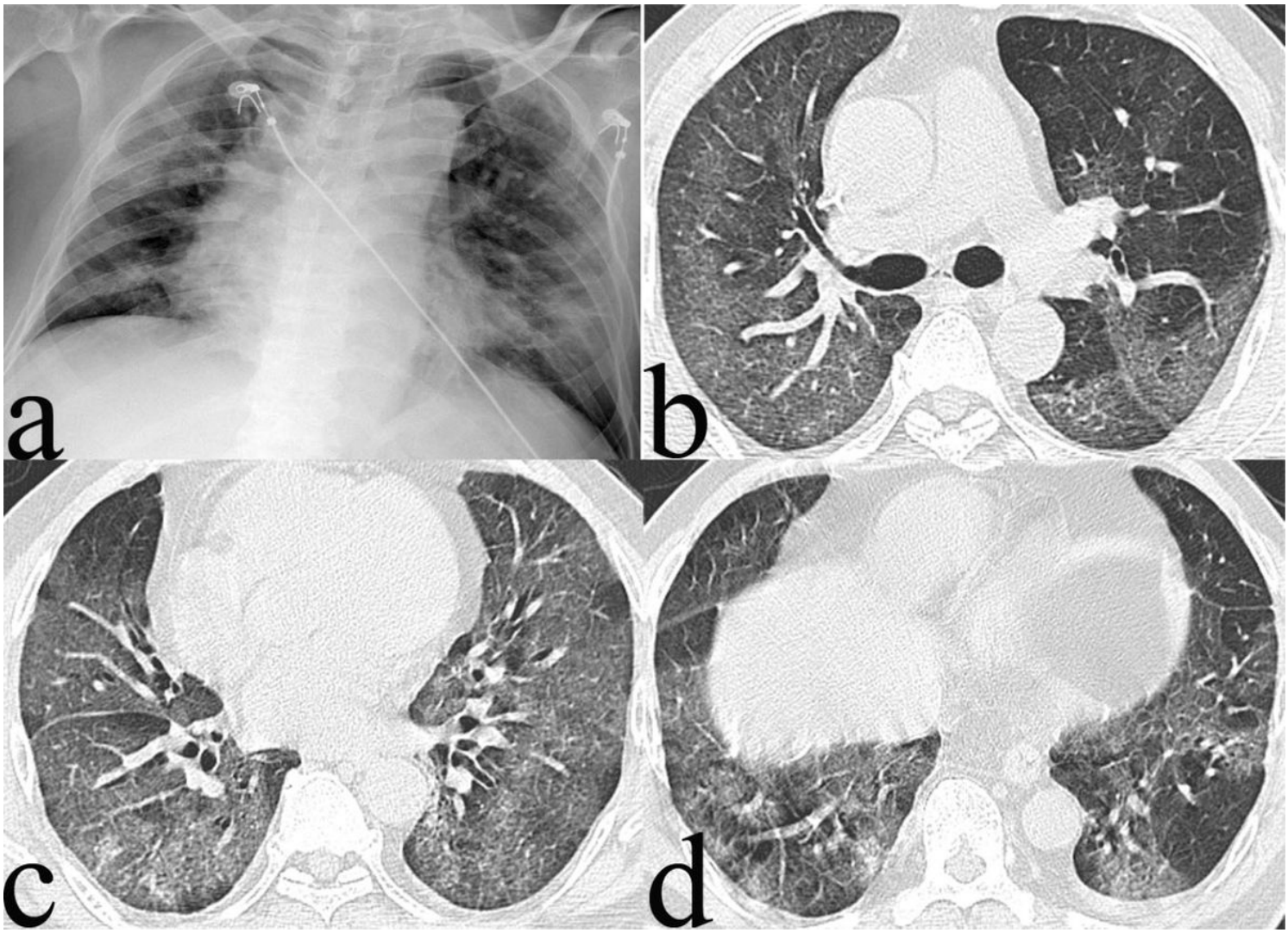

Figure 3

A 58-year-old man diagnosed with severe COVID-19 on January 30th with asthma. Imaging showed severe performances (stage III). a. Chest radiograph (a) on January 31th showed multiple high-density lesions with peripheral distribution and blurred boundary. b-d. Axial chest CT on February 4th showed diffusely distributed ground-glass opacity in bilateral lungs involving the whole lung lobes, with mild consolidation.

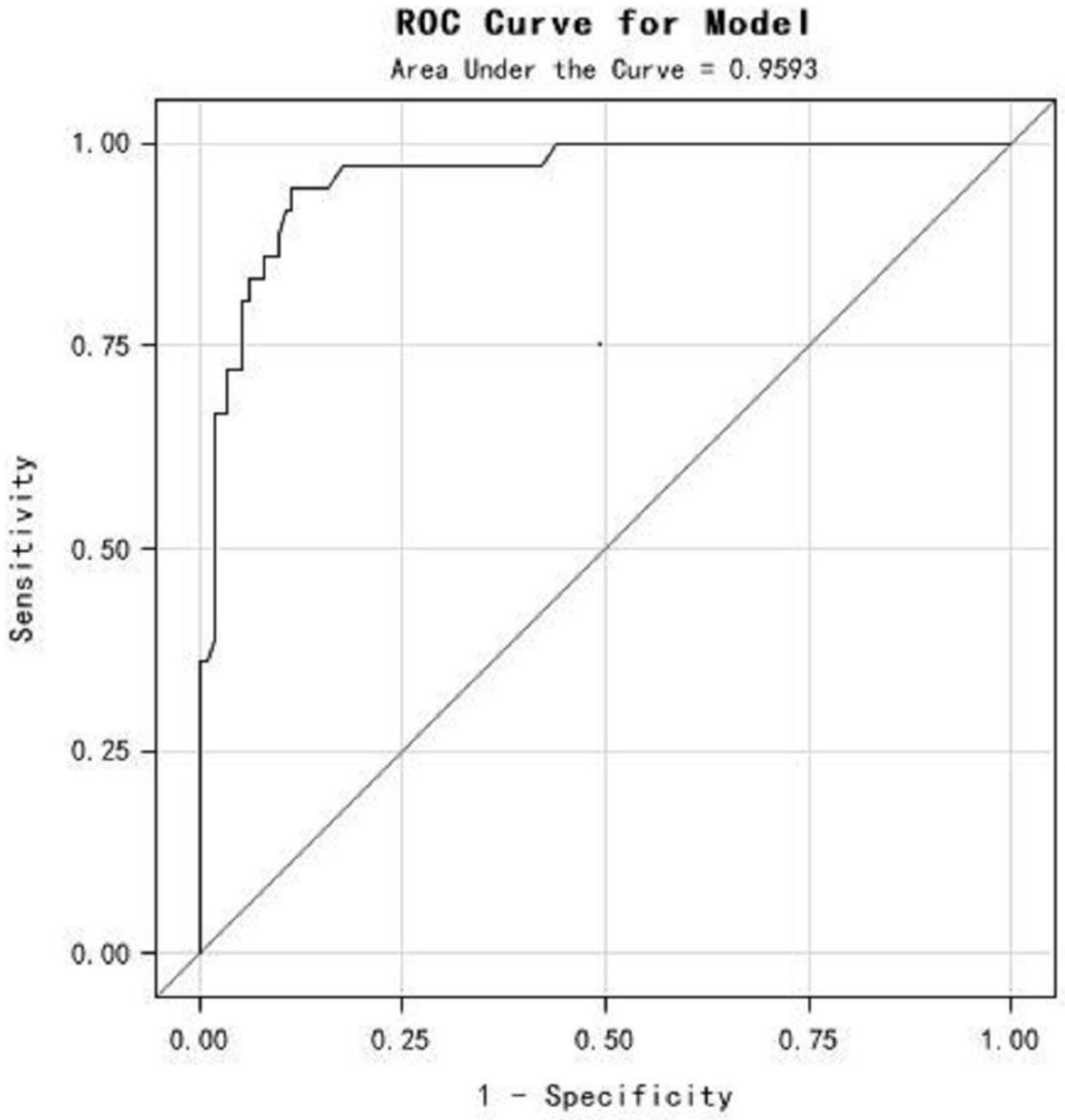

Figure 4

Receiver operating characteristic curve of death prediction model. With the area under curve of 0.9593, the multivariate logistic regression model is reliable.

Supplementary Files

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