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Analysis of Risk Factors for Criticalization and Death in Hospitalized Patients with COVID-19 Infection in the Post Epidemic Era

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Abstract: *To study the risk factors of criticalization and death in hospitalized patients with COVID-19 infection. 320 patients with COVID-19 infection admitted to a tertiary hospital from January to October 2023 were collected, and unifactorial and multifactorial analyses were performed in terms of clinical features, laboratory test results, imaging examinations and treatment measures to find out the risk factors related to the critical deterioration and death of patients hospitalized with new crown among them. The results of multifactorial analysis showed that prolonged hospitalization days, decreased absolute serum lymphocyte values, decreased CD4+ T lymphocytes, increased IL6 counts, and chest CT inflammatory index classification grade IV were risk factors for the evolution of critical illness in patients with COVID-19 infection, whereas comorbid diabetes mellitus, decreased absolute serum lymphocyte values, decreased CD4+ T lymphocytes, and increased IL6 counts were risk factors for death in patients with COVID-19 infection critical illness. The combined test of blood IL-6 and lymphocyte levels and imaging chest CT inflammatory index grading can assess the inflammatory response, immune function, and lung lesion status in patients with COVID-19 infection pneumonia, thus making a prognosis of disease severity and prognosis. Patients with diabetes mellitus, progressive decrease in lymphocyte count, increase in IL-6 level, and grade IV of chest CT inflammation index should be alerted, to achieve early detection, early diagnosis, early intervention, early adjustment of the treatment plan, avoid progressive disease progression, and reduce the rate of criticalization and mortality.*

Keywords: *COVID-19, hospitalized patients, criticalization, death, risk factors*

1. Introduction

As of January 13, 2023, China has cumulatively reported 348,858,000 doses of COVID-19 vaccine, with the number of people covered by the vaccine accounting for 92.37% of the country's total population (especially the vaccination rate of elderly people aged 65 years or older exceeds 90%) [1], China's prevention and control of epidemics has entered a new stage of shifting from prevention and control of infections to medical care and treatment [2]. But the proportion of patients hospitalized with COVID-19 infection infections who were clinically classified as severe and critical remained high, and of the 320 patients hospitalized with COVID-19 infection infections included in this study, 31.6% were critically ill and 15% died. Critically ill patients with COVID-19 infection infections often have severe symptoms, recurrent disease, and even various complications leading to death, but the outcome of treatment could have been much different with earlier clinical intervention. In this study, we retrospectively analyzed the clinical data of 320 cases of COVID-19 infection in a tertiary hospital between January 1, 2023 and October 1, 2023 to explore the risk factors affecting the criticalization and death of patients hospitalized with COVID-19 infections, and to provide a basis for further guidance on the early detection, diagnosis, and treatment of critically ill patients with COVID-19 infections.

2. Methods

2.1. Study design and participants

A total of 333 patients with novel coronavirus infection admitted to a tertiary hospital from January 1, 2023 to October 1, 2023 were collected. Inclusion criteria: 333 patients were diagnosed according to

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the diagnostic criteria of "Guidelines on diagnostic and treatment of novel coronavirus pneumonia (Trial 10th edition)"[3] (hereinafter referred to as the "Guidelines") issued by the General Office of the National Health Commission, and were excluded from the study. 13 patients were classified into mild (8 cases), ordinary (160 cases), or chronic (3 cases) according to the clinical classification criteria. According to the clinical classification criteria, the patients were classified into light type (8 cases), ordinary type (160 cases), heavy type (57 cases) and critical type (95 cases). In order to initially and effectively count the relationship between the patients' laboratory indexes and the severity of the disease, 95 patients with critical type were categorized into the critical group, and the critical group was divided into the death group (41 cases) and the survival group (54 cases), while 225 patients with other types were categorized into the non-critical group, with the following exclusion criteria: (1) patients with non-confirmed diagnosis; and (2) patients with incomplete medical record data. The study was reviewed and approved by the Ethics Committee of the hospital, and the patients were exempted from informed consent.

2.2. Methodology

The age, gender, time from onset to admission, as well as the main symptoms, the results of the first examination of laboratory indexes (blood routine, blood biochemistry, inflammatory factors, lymphocyte subpopulations, D-dimer, troponin), imaging manifestations, underlying diseases, therapeutic measures were retrospectively analyzed and statistically analyzed. Among them, the imaging examination was expressed by chest CT inflammation index (pulmonary inflammation index (PII)), and the CT inflammation index[4,5] was the sum of the lesion volume score, lesion distribution score, and solid lesions divided by 40, which was expressed as a percentage, with 0 being classified as grade 0; 1% to 25% as grade I; 26% to 50% as grade II; 51% to 75% as grade III; and more than 75% as grade IV; in addition, if the lesion volume score, lesion distribution score and solid lesions were divided by 40, it was classified as grade IV; in addition, if the lesion volume score, lesion distribution score and solid lesions were divided by 40, it was classified as grade IV. In addition, if the lesions were lobulated, the grading was adjusted downward by one grade, and if pleural effusion was present, the grading was adjusted upward by one grade. Scoring of lesion distribution: according to the distribution of the lesion in lung segments, one lung segment was scored as 1 point, the apical posterior segment of the upper lobe of the left lung and the anterior medial basal segment of the lower lobe of the left lung were each scored as 2 points, and the left and right lungs were scored as a total of 20 points.

2.3. Data analysis

SPSS27.0 software was used for statistical analysis. Measurement information that conformed to normal distribution was expressed as $x \pm s$, and t test was used for comparison between the two groups; measurement information that did not conform to normal distribution was expressed as M (P_{25}, P_{75}), and Mann-Whitney U test was used for comparison between the groups. Count data were expressed as the number of cases (percentage), and comparisons were made using the $\chi 2$ test. Variables with statistically significant differences were included in binary logistic regression equations and multifactorial Cox regression models to exclude the effects of confounding factors and to screen for independent risk factors for the progression of patients with COVID-19 infection infections to critical illnesses and for death in critically ill patients. Differences were considered statistically significant at $P<0.05$.

3. Results

3.1. Epidemiological characteristics

Of the 320 patients hospitalized with COVID-19 infection, 225 were in the non-critical group and 95 were in the critical group, 4 died in the non-critical group and 41 died in the critical group, the average number of days of hospitalization for the non-critical patients was 11.0 (7.0, 15.0) days, the average age was 68.5 (57.8, 80) years old, and the average number of days of hospitalization for the critically ill patients was 15.0 (7.0, 26.0) days, and the average age was 75.0(66.0, 83.0) years old, the age and hospitalization days of the critical group were significantly higher than those of the non-critical group, and the difference was statistically significant ($P<0.05$); comparing other indicators, the difference was not statistically significant ($P>0.05$). See Table 1.

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

Table1: Comparison of clinical data between critically ill and noncritically ill patients with novel coronavirus infection.

sports event	Non-risk group (n=225)	Critical group(n=95)	statistical value	P
Age [years]	68.5 (57.8, 80)	75.0 (66.0, 83.0)	12993.500	<0.01
Length of hospitalization [days]	11.0 (7.0, 15.0)	15.0 (7.0, 26.0)	13020.000	<0.01
Onset [days]	8.0 (5.0, 10.0)	7.0 (4.0, 12.0)	4937.500	0.753
BMI [kg/m2]	23.3 (21.0, 25.5)	23.7 (21.3, 26.7)	4818.000	0.227
Sex [cases (%)]			0.806	0.369
male	152 (67.6)	69 (72.6)		
Female	73 (32.4)	26 (27.4)		
Hypertension [cases (%)]			0.271	0.603
not have	109 (48.4)	43 (45.3)		
have	116 (51.6)	52 (54.7)		
Diabetes mellitus [cases (%)]			0.138	0.710
not have	147 (65.3)	60 (63.2)		
have	78 (34.7)	35 (36.8)		
Heart disease [cases (%)]			0.150	0.903
not have	139 (61.8)	58 (61.1)		
have	86 (38.2)	37 (38.9)		
Lung diseases [cases (%)]			2.727	0.099
not have	163 (72.4)	60 (63.2)		
have	62 (27.6)	35 (36.8)		
Tumors [cases (%)]			0.800	0.371
not have	193 (85.8)	85 (89.5)		
have	32 (14.2)	10 (10.5)		

3.2. Laboratory and imaging tests

Table 2: Comparison of critical and non-critical group testing examinations in patients with COVID-19 infection.

sports event	Non-risk group (n=225)	Critical group (n=95)	statistical value	P
White blood cell count [$\times 10^9$ /L]	5.78 (4.23, 8.33)	7.6 (5.5, 10.88)	14440.5	<0.01
Absolute lymphocyte values [$\times 10^9$	0.83 (0.61,1.17)	0.55(0.34,0.75)	5953	<0.01

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

/L)]				
Absolute neutrophil value [×10^9 /L]	4.44 (2.88, 6.55)	6.64 (4.36, 9.41)	14852	<0.01
Platelet count [×10^9 /L]	175 (132,225)	166 (123.5, 224)	10355.5	0.694
D-2 polymers [mg/L]	0.64 (0.33,1.33)	2.59 (0.96,5.52)	17278	<0.01
Fibrin degradation products [mg/L]	1.9 (1,3.6)	6 (3.1, 17.35)	17283.5	<0.01
C-reactive protein [mg/L]	37.7 (13.8, 68.8)	65.2 (22.45, 117.5)	13856	<0.01
Procalcitonin [μg/L]	0.07(0.05,0.17)	0.22(0.1,0.67)	16384	<0.01
B-type natriuretic peptide precursor [ng/L]	751 (171,1548)	1723 (340, 2450)	14967.5	<0.01
Lactate dehydrogenase [U/L]	236 (189,317)	316 (259.5, 431.5)	16129	<0.01
Alanine aminotransferase [U/L]	30 (20, 47.26)	29(19,53)	11166.5	0.929
Aspartate aminotransferase [U/L]	37(24,50)	37 (27.5, 64.5)	12435.5	0.142
Creatinine [μmol/L]	75 (58,95)	87 (63.5, 135.27)	13571.5	0.008
Serum Troponin I [mg/L]	0.013(0.008,0.29)	0.033(0.013,0.104)	15708	<0.01
CD3+ T lymphocytes[/μL]	564.14 (505.48, 603.5)	424.53 (266.5, 442.72)	4219.5	<0.01
CD4+ T-lymphocytes[/μL]	301.76 (258.48, 329.5)	197.46 (137.5, 211.05)	4081	<0.01
CD8+ T-lymphocytes[/μL]	218.18 (201.18, 229.22)	176.24 (89,181.43)	4254.5	<0.01
B lymphocytes[/μL]	133.67 (123.14, 140.88)	109.13 (68, 112.53)	4198.5	<0.01
IL-6 [ng/L]	22.12 (5.6, 80.3)	58.1 (19.5, 314.85)	14692.5	<0.01
Chest CT PII classification [cases (%)]			52.77	<0.01
0 level	6	0		
Class I	34	2		
Class II	135	27		
Class III	42	35		
Level IV	8	31		

After the patients were admitted to the hospital, routine blood and blood biochemical indexes were checked, and serum leukocytes, absolute neutrophil value, absolute lymphocyte value, D-2 polymers, fibrinogen degradation products, B-type natriuretic peptide precursor, lactate dehydrogenase, creatinine, and serum troponin I counts in the critical group were all increased compared with that of the non-critical group, and the differences were all statistically significant (all $P<0.05$). While platelet, ESR, AST and ALT levels were compared between the two groups, the differences were not statistically significant (all $P>0.05$). In addition, inflammatory factors such as C-reactive protein, Procalcitonin, and IL6 were significantly higher in the critical group than in the non-critical group; CD3+ T-lymphocyte counts, CD4+ T-lymphocyte counts, CD8+ T-lymphocyte counts, and absolute values of B-lymphocytes of patients in the two groups were lower than the lower limit of the normal reference range, but the decline was more pronounced in the critical group; whereas in terms of imaging, CT PII of chests of patients with the critical group classification grade IV accounted for a significantly higher proportion (32.6%) than that in the non-critical group (3.6%), and the difference was statistically significant ($P<0.05$). See Table 2.

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

3.3. Therapeutic measures

In this study, the proportion of antimicrobials, hormones and other immuno-intervention therapeutic drugs used in the critical group was significantly higher than that in the non-critical group, with the proportion of hormone use in the deceased group further elevated compared with that in the survivor group, and the difference was statistically significant (both $P < 0.05$). The guideline [3] points out that antiviral treatment for COVID-19 infection infections is applicable to adult patients with mild or moderate disease with high risk factors for progression to severe disease within 5 days of the onset of the disease, and there is a temporal limitation on the use of COVID-19 infection potent antiviral drugs such as Paxlovid, Molnupiravir, etc. Therefore, the proportions of antiviral use in at-risk groups and non-at-risk groups in the present study are similar, and the differences between the groups are not statistically significant. See Table 3.

Table 3: Comparison of therapeutic measures for patients hospitalized with COVID-19 infections.

sports event	non-critical reorganization (n=225)	hazardous reorganization (n=95)	χ^2	P	survivor group (n=54)	death panels (n=41)	χ^2	P
Antiviral treatment [cases (%)]			0.567	0.452			0.404	0.525
not have	80 (35.6)	38 (40.0)			21 (38.9)	18 (43.9)		
have	145 (64.4)	57 (60.0)			33 (61.1)	23 (56.1)		
Hormone therapy [cases (%)]			8.566	0.003			4.095	0.043
not have	101 (44.9)	26 (27.4)			20 (37.0)	7 (17.1)		
have	124 (55.1)	69 (72.6)			34 (63.0)	34 (82.9)		
Antimicrobial treatment [cases (%)]			10.368	0.001			1.832	0.176
not have	40 (17.8)	4 (4.2)			1(1.9)	3 (7.3)		
have	185 (82.2)	91 (95.8)			53 (98.1)	38 (92.7)		
Immunomodulatory therapy [cases (%)]			8.349	0.004			0.332	0.565
not have	175 (77.8)	59 (62.1)			36 (66.7)	24 (58.5)		
have	50 (22.2)	36 (37.9)			18 (33.3)	17(41.5)		

3.4. Multifactor logistic analysis

Table 4: Logistic regression analysis of the progression to critical cases in patients with COVID-19 infections.

variant	beta value	standard error	Wald value	P	OR value	95% credible interval
Days of hospitalization	0.046	0.020	5.330	0.021	1.047	1.007~1.088
PII Classification IV	1.925	0.547	12.380	0.001	6.854	2.346~20.028

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

Absolute Lymphocytes	-1.751	0.616	8.076	0.004	0.174	0.052~0.581
CD4+ T-lymphocyte count	-0.010	0.004	7.302	0.007	0.990	0.982~0.997
IL6	0.001	0.001	4.333	0.037	1.001	1.000~1.003

To exclude confounding factors, binary logistic regression analysis was performed on the parameters for which the difference between critically ill and non-critically ill patients at the time of admission was statistically significant, and the final logistic regression model obtained was statistically significant. The model was able to correctly classify 83.4% of the study population. The predictive value of the model was good for non-critical patients, 92.9% (209/225) of the non-critical patients were predicted by the model to be non-critical, and 65 out of 95 critically ill patients were able to be recognized by the model. Among them, date of hospitalization, chest CT PII classification grade IV, absolute lymphocyte count, CD4+ T lymphocyte count, and IL-6 were correlated with the severity of patient's condition and were independent risk factors for progression to critical illness in patients with COVID-19 infection infections. See Table 4. while gender, age, absolute neutrophil count, platelet count, ALT, AST, lactate dehydrogenase, creatinine, CRP, procalcitonin, D-dimer, B-type natriuretic peptide precursor 、 serum troponin I ultimately failed to be included in the regression model. See Table 4.

3.5. Analysis of risk factors for death in critically ill patients with COVID-19 infection

Of the 95 critically ill patients with COVID-19 infection, 41 died. compared with the critically ill surviving patients, the differences between D-dimer, fibrinogen degradation products, PCT, IL6, LDH, creatinine, serum troponin I, CD4+ T lymphocyte count, and combined diabetes mellitus were statistically significant in the patients in the deceased group (all $P<0.05$) as shown in Table 5 and Table 6. as a confounding factor in the COX multifactorial regression analysis, the results showed that combined diabetes mellitus, absolute lymphocyte count, CD4+T lymphocyte count, and IL6 were statistically significant ($P<0.05$) as independent risk factors for death in patients with COVID-19 infection infections. See Table 7.

Table 5: Comparison of clinical data between deceased and surviving patients in the critical group of novel coronavirus infections.

sports event	Survival group (n=54)	Death group (n=41)	statistical value	P-value
Age [years]	73 (69,83)	74 (65,82)	1286.5	0.311
Length of hospitalization [days]	16 (11,29)	11 (4,17)	565.500	<0.01
Onset [days]	9.0 (5.0, 11.0)	7.0 (4.0, 10.0)	524.500	0.415
BMI [kg/m2]	24.0 (21.5, 26.7)	23.4 (20.6, 27.2)	249.000	0.651
Sex [cases (%)]			0.141	0.351
Male	42 (77.8)	28 (68.3)		
Female	12 (22.2)	13 (31.7)		
Hypertension [cases (%)]			0.236	0.627
not have	26 (48.1)	17 (41.5)		
have	28 (51.9)	24 (58.5)		
Diabetes mellitus [cases			4.142	0.042

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

(%)]				
not have	40 (74.1)	21 (51.2)		
have	14 (25.9)	20 (48.8)		
Heart disease [cases (%)]			2.750	0.097
not have	38 (70.4)	21 (51.2)		
have	16 (29.6)	20 (48.8)		
Lung diseases [cases (%)]			0.268	0.605
not have	34 (63.0)	27 (65.6)		
have	20 (37.0)	14 (34.4)		
Tumors [cases (%)]			0.024	0.878
not have	50 (92.6)	37 (90.2)		
have	4 (7.4)	4 (9.8)		

Table 6: Comparison of test examinations between deceased and surviving patients in the critical group of COVID-19 infections.

sports event	Survival group (n=54 cases)	Death group (n=41 cases)	statistical value	P-value
White blood cell count [$\times 10^9$ /L]	7.6 (5.9,10.8)	8.6 (4.5,12.1)	1395.5	0.071
Absolute lymphocyte values [$\times 10^9$ /L)]	0.516 (0.320,0.766)	0.600 (0.511,0.867)	1234.0	0.530
Absolute neutrophil value [$\times 10^9$ /L]	6.78 (4.67,9.37)	7.52 (3.51,10.80)	1366.5	0.111
Platelet count [$\times 10^9$ /L]	172 (146,253)	177 (99,219)	957.0	0.163
D-2 polymers [mg/L]	1.52 (0.59,3.97)	4.67 (1.46,17.40)	1643.5	<0.01
Fibrin degradation product [mg/L]	4.4 (2.0, 15.2)	15.2 (5.5, 41.8)	1638.0	<0.01
C-reactive protein [mg/L]	48.3 (10.6, 128.0)	80.7 (24.6, 134.4)	1394.5	0.072
Procalcitonin [μg/L]	0.22(0.09,0.46)	0.39 (0.16,4.61)	1502.5	0.010
B-type natriuretic peptide precursor [ng/L]	1140.0 (268.0, 2653.3)	2296.3 (379.0, 3371.3)	1359.0	0.123
Lactate dehydrogenase [U/L]	310.0 (222.0, 331.0)	368.0 (381.8.0, 516.5)	1558.0	0.003
Alanine aminotransferase [U/L]	24.0 (17.0, 60.0)	20.5 (17.0, 44.8)	1075.0	0.594
Aspartate aminotransferase [U/L]	35.0 (25.0, 51.0)	35.0 (26.0, 42.8)	1201.0	0.699
Creatinine [μmol/L]	84.0 (59.0, 131.0)	98.5 (73.8,205.0)	1425.5	0.043

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

Serum Troponin I [mg/L]	0.020 (0.013,0.055)	0.032 (0.012,0.120)	1521.5	0.006
CD3+ T lymphocytes[/μL]	427 (413,440)	419 (171,443)	762.5	0.005
CD4+ T-lymphocytes[/μL]	201 (192,225)	193 (89,211)	758.5	0.004
CD8+ T-lymphocytes[/μL]	178 (174,181)	169 (48,181)	839.5	0.024
B lymphocytes[/μL]	110 (108,113)	106 (30,112)	899.0	0.069
IL-6 [ng/L]	50.8 (7.2, 203.0)	241.8 (36.4, 733.6)	1554.0	0.003
Chest CT PII grade IV [cases (%)]			2.950	0.086
clogged	42 (77.8)	24 (58.5)		
be	12 (22.2)	17 (41.5)		

Table 7: Multifactorial COX regression analysis of death in critically ill patients with COVID-19 infection.

	beta value	standard error	Wald value	significance	OR value	95% credible interval
diabetes	-0.864	0.342	6.388	0.011	0.422	0.216~0.824
Absolute Lymphocytes	0.003	0.001	14.495	0.000	1.003	1.002~1.005
CD4+T-lymphocyte count	-0.006	0.003	4.715	0.030	0.994	0.989~0.999
IL6	0.000	0.000	9.263	0.002	1.000	1~1.001

4. Conclusion

The average hospitalization date of patients in the critical group in this study was 15.0 (7.0, 26.0) days, which was significantly higher than that of non-critical group, which was 11.0 (7.0, 15.0) days. The New Crown Guidelines [3] pointed out that critically ill patients with COVID-19 infections tend to develop respiratory distress and/or hypoxemia after 5-7 days of the onset of the disease, and even progressed to acute respiratory distress syndrome, septic shock, difficult-to-correct metabolic acidosis and coagulation dysfunction and multi-organ failure. Critically ill patients with poor baseline conditions, complex conditions, and poor therapeutic effects often require prolonged hospitalization [6]. On the other hand, prolonged hospitalization is more prone to secondary infections, venous thrombosis and other complications, further affecting the prognosis[7,8] . The length of hospitalization is an independent risk factor for the criticalization of patients hospitalized with new coronary infections. For patients hospitalized for a long period of time, on the one hand, it is necessary to increase vigilance to prevent the emergence of various complications, and on the other hand, it is also necessary to consider the use of hormones, monoclonal antibodies, and other immune-modulating drugs in an early stage, to prevent the condition of critically ill patients from deteriorating and to promote clinical regression.

It has been shown that COVID-19 infection can invade and proliferate immune organs such as lymph nodes and spleen, which in turn infects more lymphocytes and monocytes, and causes apoptosis of many lymphocytes [9] Therefore, patients with COVID-19 infection infections often have lymphopenia and T-cell damage, and the degree of lymphopenia correlates with the severity of the disease [10]. In this study, lymphocyte depletion was seen in both normal, heavy, and critical forms, and 76.9% of hospitalized patients with COVID-19 infection infections showed absolute lymphocyte reduction. Absolute lymphocyte counts of 0.55 (0.34, 0.75) × 10^9 /L were significantly lower in critically reconstituted

ISSN 2616-5791 Vol.5, Issue 2: 30-40, DOI: 10.25236/AJMHS.2024.050205

patients than in non-critically reconstituted 0.83 (0.61, 1.17) × 10^9 /L. Among them, the counts of CD3+ T-lymphocytes, CD4+ T-lymphocytes, and CD8+ T-lymphocytes were lower in critically reconstituted than in non-critically reconstituted, which showed that the cellular immune functions of critically reconstituted patients were significantly suppressed. In contrast, the absolute lymphocyte values and CD4+ T lymphocyte counts of the 41 patients who died in the critically reconstituted group were lower than those of the surviving group. Low levels of lymphocytes and CD4+ T cell counts are positively correlated with disease progression and even death.

The most common causes of death in patients with COVID-19 infection infections are respiratory failure due to acute respiratory distress syndrome and infectious shock due to multiple infections [11], In this study, 29 (64.4%) of the 45 patients who died presented with acute respiratory distress syndrome, 19 (42.2%) with sepsis, and 15 (33.3%) with infectious shock. Interleukin-6 (IL-6) is rapidly released into the circulation after infection and is one of the commonly used indicators of inflammation in infectious diseases [12]. After neo-crown infection, the virus rapidly activates pathogenic T-cells to produce factors such as granulocyte-macrophage colony-stimulating factor (GM-CSF) and IL-6, and GM-CSF further activates CD14+CD16+ inflammatory monocytes to produce even larger quantities of IL-6 and other inflammatory factors, resulting in the formation of an inflammatory storm[13] , which leads to severe immune damage to the lungs and other organs, causing acute respiratory distress syndrome, multi-organ failure and other complications. On the other hand, it has been found that IL-6>56.12ng/L is an independent predictor of poor outcome in patients with sepsis [14]. Elevated IL-6 means that the probability of complications such as acute respiratory distress syndrome and septic shock in patients with COVID-19 infection infections is greatly increased, and in the present study, the IL-6 level in the critical group was significantly higher than that in the non-critical group, in which the IL6 level in the patients in the fatal group was further elevated. It is one of the risk factors for COVID-19 infection infections to become severe and death.

The use of glucocorticoids was higher in the critically ill group and the death group than in the control group in this study. Glucocorticoids can reduce the inflammatory response, attenuate the damaging effects of tissues, and reduce the exudation, injury, and late pulmonary fibrosis of the lungs [15], which can be used in the treatment of ARDS [16], therefore, glucocorticoids are used in short-term and small doses in critically ill patients to inhibit excessive inflammatory response of the body, reduce the incidence of complications, and effectively reduce the mortality rate. However, glucocorticosteroids can also inhibit the immune response and clearance of pathogens, thus increasing the rate of mortality and secondary infections [17]. The use of glucocorticoids needs to be weighed against the advantages and disadvantages, and their combined use is not recommended for patients with mild, common types of disease, as well as caution in patients with concomitant bacterial and fungal infections, and the presence of suppression, so glucocorticoids were not ultimately included in the regression model of factors influencing the critically-illuminated group. In addition, in this study, the use rate of new coronary effect antiviral drugs in the survival group of patients in the critical group is slightly higher than that in the death group. Antiviral drugs play an antiviral role by blocking the process of viral polyprotein hydrolysis after viral invasion into the human cells, to inhibit the replication of the virus, but it cannot prevent the new coronary viruses from invading the healthy cells, and there is no direct viral killing [18], and have certain conditions of restriction on the use of the virus, therefore, it was not included in the regression model. It was not included in the regression model.

The chest CT of patients with COVID-19 infection mostly showed subpleural ground glass density shadow, with the change of patient's condition, there is a tendency of increasing or decreasing lung foci, before and after the review of the lung inflammation index score is very close, but there is a difference in the volume of the foci in individual lung segments, according to the foci with or without lobular distribution of the lung inflammation index grading to adjust, which can more realistically reflect the severity of the lung injury of the patients with COVID-19 infection. [19]. In this study 6 patients with lung inflammation index grade 0 were light patients, 31 of 36 grade I were normal, 135 of 162 grade II were normal, 29 of 77 grade III were heavy patients, 35 were critically ill, and 31 of 39 grade IV were critically ill , and the statistical results showed that there was a good correlation between CT lung inflammation index grading IV and the critical group of COVID-19 infection ($P < 0.05$), which is consistent with the clinical assessment as a risk factor for critical grading of COVID-19 infection. The CT lung inflammation index system can assist physicians to classify patients quickly and conveniently clinically with COVID-19 infections, and at the same time can be used as an auxiliary means of evaluating the severity of the disease and the progression or not of patients with COVID-19 infections [20].

It has been noted that new coronary patients with concomitant hypertension, diabetes, and coronary artery disease have a greater proportion of critical illness and a higher risk of death [21]. Although diabetes

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mellitus was a risk factor for death in the critically ill group of patients in this study. In addition, a review of cases in the mortality group that presented with acute heart failure or even cardiogenic shock revealed that most of them had underlying cardiac diseases. On the one hand, the control of the underlying disease needs to be taken into account, such as diabetic patients with uncontrolled blood glucose have a heavier inflammatory response and are more prone to inflammatory storms; on the other hand, the underlying disease can affect other aspects of the patient such as immune function, such as patients with concomitant diabetes often have poorer cellular often immune function[22] , and in the present study, diabetic patients with CD3+ T-cells, CD4+ T-cells, and CD8+ T-cells counts were lower than those of non-diabetic patients. The presence of underlying diseases such as diabetes mellitus has an important impact on the prognosis of patients with new crowns in critical condition.

Although the COVID-19 infection has been prevented and controlled infection transferred to a new stage of medical treatment, the number of hospitalized patients has been greatly reduced, but the proportion of their critical illnesses and deaths are still high, and the factors affecting the criticality and deaths of the new crown hospitalized patients are still worthy of attention. Patients with COVID-19 infections are often combined with bacterial and fungal infections, and IL-6 levels are also elevated. In addition, non-infectious factors such as trauma and surgery can also cause elevation of IL-6, and it is not absolute to use one clinical indicator alone to judge the progression of the disease. Therefore, the combination of IL-6 and lymphocyte levels in blood and the inflammatory index on imaging chest CT [23] can assess the inflammatory response and immune function and the status of lung foci in patients with COVID-19 inertinites, and thus make a prognosis for the severity and prognosis of the disease. In addition, patients with diabetes mellitus and other underlying diseases, progressive decrease in lymphocyte counts, and increase in IL-6 levels should be alerted, and the treatment plan should be adjusted as early as possible to avoid progressive disease progression, to achieve early detection, early diagnosis, and early intervention in critically ill patients, and to reduce the rate of weight transfer and mortality.

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