Clinical characteristics of fatal and recovered cases of coronavirus disease 2019 in Wuhan, China: a retrospective study

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Abstract

Background: The 2019 novel coronavirus has caused the outbreak of the acute respiratory disease in Wuhan, Hubei Province of China since December 2019. This study was performed to analyze the clinical characteristics of patients who succumbed to and who recovered from 2019 novel coronavirus disease (COVID-19).

Methods: Clinical data were collected from two tertiary hospitals in Wuhan. A retrospective investigation was conducted to analyze the clinical characteristics of fatal cases of COVID-19 (death group) and we compare them with recovered patients (recovered group). Continuous variables were analyzed using the Mann-Whitney *U* test. Categorical variables were analyzed by χ^2 test or Fisher exact test as appropriate.

Results: Our study enrolled 109 COVID-19 patients who died during hospitalization and 116 recovered patients. The median age of the death group was older than the recovered group (69 [62, 74] *vs.* 40 [33, 57] years, $Z = 9.738$, $P < 0.001$). More patients in the death group had underlying diseases (72.5% *vs.* 41.4%, $\chi^2 = 22.105$, $P < 0.001$). Patients in the death group had a significantly longer time of illness onset to hospitalization (10.0 [6.5, 12.0] *vs.* 7.0 [5.0, 10.0] days, $Z = 3.216$, $P = 0.001$). On admission, the proportions of patients with symptoms of dyspnea (70.6% *vs.* 19.0%, $\chi^2 = 60.905$, $P < 0.001$) and expectoration (32.1% *vs.* 12.1%, $\chi^2 = 13.250$, $P < 0.001$) were significantly higher in the death group. The blood oxygen saturation was significantly lower in the death group (85 [77, 91]% *vs.* 97 [95, 98]%, $Z = 10.625$, $P < 0.001$). The white blood cell (WBC) in death group was significantly higher on admission (7.23 [4.87, 11.17] *vs.* 4.52 [3.62, 5.88] $\times 10^9$/L, $Z = 7.618$, $P < 0.001$). Patients in the death group exhibited significantly lower lymphocyte count (0.63 [0.40, 0.79] *vs.* 1.00 [0.72, 1.27] $\times 10^9$/L, $Z = 8.037$, $P < 0.001$) and lymphocyte percentage (7.10 [4.45, 12.73]% *vs.* 23.50 [15.27, 31.25]%, $Z = 10.315$, $P < 0.001$) on admission, and the lymphocyte percentage continued to decrease during hospitalization (7.10 [4.45, 12.73]% *vs.* 2.91 [1.79, 6.13]%, $Z = 5.242$, $P < 0.001$). Alanine transaminase (22.00 [15.00, 34.00] *vs.* 18.70 [13.00, 30.38] U/L, $Z = 2.592$, $P = 0.010$), aspartate transaminase (34.00 [27.00, 47.00] *vs.* 22.00 [17.65, 31.75] U/L, $Z = 7.308$, $P < 0.001$), and creatinine levels (89.00 [72.00, 133.50] *vs.* 65.00 [54.60, 78.75] μmol/L, $Z = 6.478$, $P < 0.001$) were significantly higher in the death group than those in the recovered group. C-reactive protein (CRP) levels were also significantly higher in the death group on admission (109.25 [35.00, 170.28] *vs.* 3.22 [1.04, 21.80] mg/L, $Z = 10.206$, $P < 0.001$) and showed no significant improvement after treatment (109.25 [35.00, 170.28] *vs.* 81.60 [27.23, 179.08] mg/L, $Z = 1.219$, $P = 0.233$). The patients in the death group had more complications such as acute respiratory distress syndrome (ARDS) (89.9% *vs.* 8.6%, $\chi^2 = 148.105$, $P < 0.001$), acute cardiac injury (59.6% *vs.* 0.9%, $\chi^2 = 93.222$, $P < 0.001$), acute kidney injury (18.3% *vs.* 0%, $\chi^2 = 23.257$, $P < 0.001$), shock (11.9% *vs.* 0%, $\chi^2 = 14.618$, $P < 0.001$), and disseminated intravascular coagulation (DIC) (6.4% *vs.* 0%, $\chi^2 = 7.655$, $P = 0.006$).

Conclusions: Compared to the recovered group, more patients in the death group exhibited characteristics of advanced age, pre-existing comorbidities, dyspnea, oxygen saturation decrease, increased WBC count, decreased lymphocytes, and elevated CRP levels. More patients in the death group had complications such as ARDS, acute cardiac injury, acute kidney injury, shock, and DIC.

Keywords: Coronavirus disease 2019; Fatality; Recovery; Clinical characteristics; Lymphocyte; C-reactive protein

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Introduction

The 2019 novel coronavirus (2019-nCoV) is the pathogen responsible for the outbreak of the acute respiratory disease in Wuhan, Hubei Province of China in December 2019.[1] Although a high proportion of infected individuals only develop mild symptoms, some cases can progress to pneumonia, multi-organ failure or even death. In this study, we performed a retrospective research focusing on clinical characteristics, laboratory findings, and the treatment regimens of the fatal and recovered coronavirus disease 2019 (COVID-19) cases with an aim to investigate the characteristics of dead patients and thereby provide some insights into the treatment of this disease.

Methods

Ethical approval

This study was conducted in accordance with the *Declaration of Helsinki* and was approved by the medical ethics committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (No. TJ-IRB20200330). Written informed consent was waived due to the rapid emergence of this infectious disease.

Patients and study design

A retrospective study focusing on patients who died due to confirmed COVID-19 during hospitalization (death group) was conducted in two tertiary hospitals in Wuhan (Hankou and Caidian branch of Tongji Hospital, Tongji Medical College, Huazhong University of Science & Technology, and Hankou branch of The Central Hospital of Wuhan) from January 1, 2020 to February 21, 2020. Recovered patients who were discharged from the same inpatient ward during the same period were also enrolled. Patients were diagnosed according to the Diagnosis and Treatment Protocol for Novel Coronavirus Pneumonia (Trial Version 6), National Health Commission of the People's Republic of China.[2] The severe cases are defined as patients with one of the following symptoms: respiratory rate ≥30 breaths/min, finger oxygen saturation ≤93% at rest, and arterial partial pressure of oxygen/fraction of inspired oxygen ≤300 mmHg.[2] The criteria for discharge are: (1) throat swab specimens collected 24 h apart were negative for tests of 2019-nCoV; (2) body temperature was normal for three consecutive days; (3) symptoms of COVID-19 were resolved; (4) the chest computed tomography manifestations of COVID-19 significantly improved.[2]

Laboratory confirmation

Throat swab specimens were tested by real-time reverse transcription polymerase chain reactions for laboratory confirmation of 2019-nCoV infection as recently reported.[3] The following primers and probe were used. The forward primer was 5′-TCAGAATGCCAATCTCCCCAAC-3′, and the reverse primer was 5′-AAAGGTCCACCCGATACATTGA-3′, while the probe was 5′ CY5-CTAGTTACACTAGCCATCCTTACTGC-3′ BHQ1. The amplification conditions were 50°C for 15 min and 95°C for 3 min, followed by 45 cycles of 95°C for 15 s and 60°C for 30 s.

Data collection

Basic information such as age, gender, underlying diseases, clinical presentations, and complications was collected from clinical charts and nursing records of each patient. Laboratory tests were conducted at admission and after treatment, including blood cell count, alanine transaminase (ALT), aspartate transaminase (AST), creatinine, and C-reactive protein (CRP). The treatment regimens including intravenous corticosteroids, intravenous gammaglobulin, anti-viral drugs, antibiotics, anti-fungal drugs, and respiratory supports were also collected from medical records.

Statistical analysis

The SPSS Statistics 23.0 software (SPSS Inc., Chicago, IL, USA) was used to perform statistical analysis of the data. Continuous variables were presented as median (interquartile range) (distribution of normality was checked by Kolmogorov-Smirnov test) and analyzed using the Mann-Whitney *U* test. Categorical variables were presented as counts and percentages, and analyzed by χ^2 test or Fisher exact test as appropriate. A value of $P < 0.05$ was considered to be statistically significant.

Results

General characteristics

One hundred and nine fatal and 116 recovered cases out of 964 COVID-19 patients admitted to two tertiary hospitals in Wuhan were enrolled in our study. The general information of the two groups was shown in the Table 1. The age ranges of the death and recovered group were 33 to 94 and 22 to 81 years, respectively. The median age of the death group was significantly older than the recovered group (69 [62, 74] *vs.* 40 [33, 57] years, $Z = 9.738$, $P<0.001$). There are more male patients in the death group (67.0% *vs.* 44.0%, $\chi^2 = 12.024$, $P < 0.001$) than the recovered group. The fatal cases had more underlying diseases (72.5% *vs.* 41.4%, $\chi^2 = 22.105$, $P < 0.001$), mainly including hypertension (36.7% *vs.* 15.5%, $\chi^2 = 14.184$, $P < 0.001$), lung disease (20.2% *vs.* 2.6%, $\chi^2 = 17.619$, $P < 0.001$), and heart disease (11.9% *vs.* 3.4%, $\chi^2 = 5.783$, $P = 0.031$) [Table 1]. Figure 1 showed the distribution of hypertension, lung disease, diabetes, heart disease, and malignancy in the two groups of COVID-19 patients [Figure 1]. More patients in the death group had more than one comorbidity.

Clinical manifestations

The baseline signs and symptoms of COVID-19 patients in the death group and the recovered group were shown in Table 2. There was no significant difference in the proportion of patients with fever, myalgia or fatigue, headache, cough, hemoptysis, diarrhea, and heart palpitations at the time of admission between two groups (all $P > 0.05$). However, the proportions of patients with dyspnea (70.6% *vs.* 19.0%, $\chi^2 = 60.905$, $P < 0.001$) and expectoration (32.1% *vs.* 12.1%, $\chi^2 = 13.250$, $P < 0.001$) were significantly higher in the death group as compared with the recovered group. The blood oxygen saturation when the patients were admitted to the hospital was

Table 1: General characteristics of the death and recovered groups with COVID-19.

Characteristics	Death group ($n = 109$)	Recovered group ($n = 116$)	Statistics	*P*-values
Age, years	69 (62, 74)	40 (33, 57)	9.738*	<0.001
Male	73 (67.0)	51 (44.0)	12.024†	<0.001
Comorbidity	79 (72.5)	48 (41.4)	22.105†	<0.001
Hypertension	40 (36.7)	18 (15.5)	14.184†	<0.001
Lung disease	22 (20.2)	3 (2.6)	17.619†	<0.001
Diabetes	17 (15.6)	9 (7.8)	3.378†	0.066
Heart disease	13 (11.9)	4 (3.4)	5.783†	0.031
Malignancy	6 (5.5)	2 (1.7)	1.369†	0.242
Others	31 (28.4)	15 (12.9)	8.311†	0.004

Data were shown as median (Q_1, Q_3) or *n* (%). * *Z* value. † χ^2 value. COVID-19: Coronavirus disease 2019.

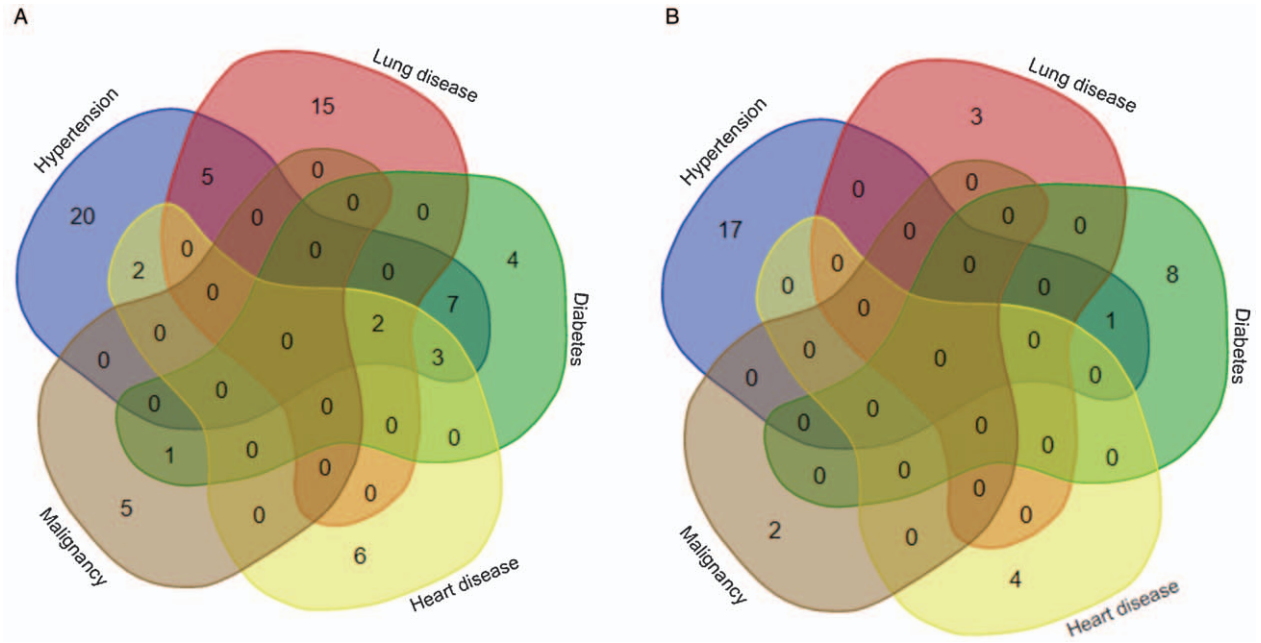

Figure 1: Venn diagrams showing the distribution of underlying diseases in the death group (A) and the recovered group (B) of COVID-19 patients. COVID-19: Coronavirus disease 2019.

significantly lower in the death group as compared with the recovered group (85 [77, 91]% *vs.* 97 [95, 98]%, $Z = 10.625$, $P < 0.001$). At the time of admission, 95 patients in the death group and nine patients in the recovered group were considered as severely ill patients. The proportion of severely ill patients was significantly higher in the death group than in the recovered group (87.2% *vs.* 7.8%, $\chi^2 = 142.515$, $P < 0.001$). The median time from illness onset to hospitalization was 10.0 (6.5, 12.0) days in the death group as compared with 7.0 (5.0, 10.0) days in the recovered group ($Z = 3.216$, $P = 0.001$) [Table 2].

Laboratory findings

Patients in the death group exhibited significantly higher white blood cell (WBC) count (7.23 [4.87, 11.17] *vs.* 4.52 [3.62, 5.88] $\times 10^9$/L, $Z = 7.618$, $P < 0.001$) which was even higher after treatment, but lower lymphocyte count (0.63 [0.40, 0.79] *vs.* 1.00 [0.72, 1.27] $\times 10^9$/L, $Z = 8.037$, $P < 0.001$), and lower lymphocyte percentage (7.10 [4.45, 12.73]% *vs.* 23.50 [15.27, 31.25]%, $Z = 10.315$, $P < 0.001$) at admission as compared with the recovered group. Notably, lymphocyte percentage (7.10 [4.45, 12.73]% *vs.* 2.91 [1.79, 6.13]%, $Z = 5.242$, $P < 0.001$) decreased after treatment during hospitalization in fatal cases. Lymphocyte count increased after treatment in the recovered group (1.00 [0.72, 1.27] *vs.* 1.53 [1.14, 2.05] $\times 10^9$/L, $Z = 5.427$, $P < 0.001$), which was not the case in the death group [Table 3].

ALT (22.00 [15.00, 34.00] *vs.* 18.70 [13.00, 30.38] U/L, $Z = 2.592$, $P = 0.010$), AST (34.00 [27.00, 47.00] *vs.* 22.00 [17.65, 31.75] U/L, $Z = 7.308$, $P < 0.001$), and creatinine levels (89.00 [72.00, 133.50] *vs.* 65.00 [54.60, 78.75] μmol/L, $Z = 6.478$, $P < 0.001$) differed between groups. Though statistically significant, most of these parameters were within the normal range or slightly higher. At the time of admission, a significant difference in the CRP level was noted between the death and the recovered group (109.25 [35.00, 170.28] *vs.* 3.22 [1.04,

Table 2: Clinical symptoms and signs of the death and recovered groups with COVID-19.

Symptoms and signs	Death group ($n=109$)	Recovered group ($n=116$)	Statistics	*P*-value
Fever before admission	95 (87.2)	94 (81.0)	1.567*	0.211
Myalgia or fatigue	30 (27.5)	27 (23.3)	0.536*	0.464
Headache	6 (5.5)	7 (6.0)	0.029*	0.865
Cough	47 (43.1)	38 (32.8)	2.566*	0.109
Expectoration	35 (32.1)	14 (12.1)	13.250*	<0.001
Hemoptysis	5 (4.6)	2 (1.7)	1.528*	0.394
Dyspnea	77 (70.6)	22 (19.0)	60.905*	<0.001
Diarrhea	19 (17.4)	14 (12.1)	0.256*	0.252
Palpitations	11 (10.1)	13 (11.2)	0.073*	0.787
Oxygen saturation, %	85 (77, 91)	97 (95, 98)	10.625†	<0.001
Severe illness	95 (87.2)	9 (7.8)	142.515*	<0.001
Time from symptom onset to admission, days	10.0 (6.5, 12.0)	7.0 (5.0, 10.0)	3.216†	0.001

Data were shown as median (Q_1, Q_3) or *n* (%). * χ^2 value. † *Z* value. COVID-19: Coronavirus disease 2019.

Table 3: Laboratory findings of the death and recovered groups with COVID-19.

	Death group ($n=109$)		Recovered group ($n=116$)	
Parameters	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment
WBC, $\times10^9$/L	7.23 (4.87, 11.17)*	12.26 (7.92, 17.51)†	4.52 (3.62, 5.88)	6.65 (4.82, 9.52)*
Lymphocyte, $\times10^9$/L	0.63 (0.40, 0.79)*	0.39 (0.24, 0.79)	1.00 (0.72, 1.27)	1.53 (1.14, 2.05)*
Lymphocyte percentage, %	7.10 (4.45, 12.73)*	2.91 (1.79, 6.13)†	23.50 (15.27, 31.25)	23.42 (16.94, 31.35)
ALT, U/L	22.00 (15.00, 34.00)*	27.00 (20.00, 47.00)‡	18.70 (13.00, 30.38)	23.20 (15.15, 38.93)§
AST, U/L	34.00 (27.00, 47.00)*	36.00 (24.00, 47.50)	22.00 (17.65, 31.75)	18.90 (15.65, 26.50)
Creatinine, μmol/L	89.00 (72.00, 133.50)*	87.00 (61.50, 181.50)	65.00 (54.60, 78.75)	60.00 (52.40, 71.50)
CRP, mg/L	109.25 (35.00, 170.28)*	81.60 (27.23, 179.08)	3.22 (1.04, 21.80)	0.50 (0.11, 2.08)*

Data were shown as median (Q_1, Q_3). The pre-treatment parameters were tested on the day of admission, and the post-treatment parameters were tested within 3 days before discharge or death. * $P<0.001$, compared with the recovered group before treatment. † $P<0.001$, compared with the death group before treatment. ‡ $P<0.05$, compared with the death group before treatment. § $P<0.05$, compared with the recovered group before treatment. COVID-19: Coronavirus disease 2019; WBC: White blood cell; ALT: Alanine transaminase; AST: Aspartate transaminase; CRP: C-reactive protein.

21.80] mg/L, $Z=10.206$, $P<0.001$). Moreover, CRP levels remained high after treatment in the death group (109.25 [35.00, 170.28] *vs.* 81.60 [27.23, 179.08] mg/L, $Z=1.219$, $P=0.233$), whereas CRP levels significantly decreased in patients in the recovered group (3.22 [1.04, 21.80] *vs.* 0.50 [0.11, 2.08] mg/L, $Z=4.980$, $P<0.001$) [Table 3].

Treatment regimen

All patients had pneumonia. The patients in the death group had more complications such as acute respiratory distress syndrome (ARDS) (89.9% *vs.* 8.6%, $\chi^2=148.105$, $P<0.001$), acute cardiac injury (59.6% *vs.* 0.9%, $\chi^2=93.222$, $P<0.001$), acute kidney injury (18.3% *vs.* 0%, $\chi^2=23.257$, $P<0.001$), shock (11.9% *vs.* 0%, $\chi^2=14.618$, $P<0.001$), and disseminated intravascular coagulation (DIC) (6.4% *vs.* 0%, $\chi^2=7.655$, $P=0.006$). More patients in the death group received high-grade antibiotics (carbapenem and/or linezolid) (35.8% *vs.* 18.1%, $\chi^2=8.979$, $P=0.003$), anti-fungal drugs (11.0% *vs.* 2.6%, $\chi^2=5.125$, $P=0.015$), and intravenous corticosteroids therapy (80.7% *vs.* 55.2%, $\chi^2=16.752$, $P<0.001$). More patients in the death group were treated with higher grade of respiratory support ($\chi^2=132.240$, $P<0.001$) such as non-invasive ventilation, invasive ventilation, and extracorporeal membrane oxygenation. The recovered patients had a longer length of hospital stay compared to death group (16 [12, 20] *vs.* 8 [4, 13] days, $Z=7.858$, $P<0.001$) [Table 4].

Discussion

Coronaviruses are enveloped RNA viruses that could affect birds, humans, and other mammals, leading to respiratory, digestive, hepatic, and nervous system disorders.[4,5] Among the six coronaviruses known to infect humans, two of them can cause ARDS, which are the severe acute respiratory syndrome coronavirus (SARS-CoV) that caused an outbreak in 2002 in China, and the Middle East respiratory syndrome coronavirus (MERS-CoV) that caused an outbreak in the Middle East in 2012.[6] 2019-nCoV is also a beta coronavirus that causes ARDS and can be transmitted between humans.[7] Similar to SARS-CoV, the 2019-nCoV is speculated to use the angiotensin-converting enzyme (ACE) 2 as a receptor for cell invasion.[8] Similar to the patients infected with SARS-CoV, some of the 2019-nCoV patients showed rapid progression of lung lesions, which might lead to death. In

Table 4: Complications and treatment of the death and recovered groups with COVID-19.

Complications and treatment	Death group ($n = 109$)	Recovered group ($n = 116$)	Statistics	*P*-value
Complications				
Acute respiratory distress syndrome	98 (89.9)	10 (8.6)	148.105*	<0.001
Acute cardiac injury	65 (59.6)	1 (0.9)	93.222*	<0.001
Acute kidney injury	20 (18.3)	0	23.257*	<0.001
Shock	13 (11.9)	0	14.618*	<0.001
Disseminated intravascular coagulation	7 (6.4)	0	7.655*	0.006
Anti-viral therapy	90 (82.6)	95 (81.9)	0.017*	0.895
Antibiotics	91 (83.5)	100 (86.2)	0.324*	0.569
High-grade antibiotics	39 (35.8)	21 (18.1)	8.979*	0.003
Anti-fungal therapy	12 (11.0)	3 (2.6)	5.125*	0.015
Intravenous corticosteroids	88 (80.7)	64 (55.2)	16.752*	<0.001
IVIG	44 (40.4)	44 (37.9)	0.140*	0.708
Respiratory support			132.240*	<0.001
Nasal cannula/mask only	12 (11.0)	103 (88.8)		
Non-invasive ventilation	58 (53.2)	10 (8.6)		
Transnasal high-flow oxygen	16 (14.7)	3 (2.6)		
Invasive ventilation	21 (19.3)	0		
Extracorporeal membrane oxygenation	2 (1.8)	0		
Length of hospital stay, days	8 (4, 13)	16 (12, 20)	7.858†	<0.001

Data were presented as *n* (%) or median (Q_1, Q_3). High-grade antibiotics refers to carbapenem and/or linezolid. Acute heart injury refers to blood levels of hypersensitive troponin I above the 99th percentile upper reference limit (>28 pg/mL) or new abnormalities shown on electrocardiography and echocardiography. * χ^2 value. † *Z* value. COVID-19: Coronavirus disease 2019; IVIG: Intravenous gammaglobulin.

this study, we analyzed the clinical characteristics of the fatal cases and recovered cases collected from two tertiary hospitals in Wuhan while most patients were still hospitalized. Therefore, it is possible that more patients in the death group and fewer patients in the recovered group were severely ill patients at admission. Differences may exist in the initial conditions of the two groups in our study.

A recent study showed that 2019-nCoV mainly infects middle-aged and elderly people.[3] Similar to that study, most of the patients in the current study were middle-aged and elderly people. The median age of the deceased patients was 69 (62, 74) years in our study. A previous study which enrolled 199 patients reported the median age of SARS non-survivors was 52 (25, 78) years and age (per 1-year increase) is a risk factor for death.[9] Another study showed that the median age of MERS non-survivors was 62 (53, 73) years, older than the survivors [46 (35, 57) years].[10] One of the possible reasons for this phenomenon might be that the lung aging is associated with an inability of lung cells and multiple structural and functional changes in the respiratory tract, giving rise to decreased lung function, altered pulmonary remodeling, diminished regeneration, and enhanced susceptibility to pulmonary disease.[11] It is also reported that the older patients have a higher risk of ARDS development.[12]

The comorbidities, particularly the cardiovascular diseases and chronic pulmonary diseases, were reported to be important to predict the in-hospital mortality in critically ill patients.[13] In our study, more patients in the death group had underlying diseases, especially hypertension, lung disease, and heart disease. Besides, more patients in the death group had more than one comorbidity. It is thought that diabetes may increase the risk of infection and can delay the recovery of the infectious illnesses. In our study, we found no significant difference between the death and recovered group in the percentage of patients complicated with diabetes. Recent studies also showed that diabetes has no significant correlation with the initiation, progression, and prognosis of ARDS.[14,15] A hypothesis is that there are more hypertensive patients who developed the 2019-nCoV infection, which is related to the ACE inhibitors used in these patients. ACE inhibitors could indirectly increase the cellular ACE2 receptors, which may be the receptors for 2019-nCoV. We found more patients in the death group had hypertension. However, the exact roles that age and underlying diseases played in the development and progression of novel coronavirus pneumonia require further investigation. Furthermore, time from illness onset to hospitalization was longer in the death group. The patients in the death group tended to have a delayed medical care compared with the recovered group.

Our study found that the proportion of patients with dyspnea was significantly higher in the death group compared to the recovered group, and initial blood oxygen saturation was lower in the death group compared to the recovered group. It is easy to understand that the progressive hypoxemia often suggests poor prognosis in pulmonary diseases and the indicators for hypoxemia are already used to evaluate the severity of the COVID-19.[3] Our study found that the WBC count increased in the death group during hospitalization, suggesting that comorbid bacterial or fungal infection might have occurred in these deceased patients. However, most of the patients in this study received broad-spectrum antimicrobial drug treatment, and no etiological evidence of infection was obtained from them. Previous studies found

that during the acute phase of SARS-CoV infection in humans, the blood lymphocyte counts, particularly CD_4^+ and CD_8^+ T cell counts, were decreased.[16-18] Another study suggested that lymphocyte cells, especially CD_4^+ and CD_8^+ T cells may play protective roles in coronavirus infection.[19] Similar to previous studies, our study found that patients in the death group had lower lymphocyte count and lymphocyte percentage. During hospitalization, the lymphocyte count and lymphocyte percentage decreased in the deceased patients. This may be because the viral infection causes persistent consumption and/or insufficient regeneration of lymphocytes.

ALT, AST, and creatinine levels were higher in the death group as compared to the recovered group. Previous studies also showed that the transaminases were elevated in the MERS and SARS patients.[20-23] A recent study showed that elderly patients infected with 2019-nCoV have higher CRP levels.[7] Moreover, CRP was considered to be a significant predictor for disease severity in SARS.[22,24] Similar to previous studies, our study found that the CRP levels were higher in the death group compared to the recovered group at the time of admission, and the CRP levels remained high during the progression of the disease.

Patients in the death group had more complications such as ARDS, acute cardiac injury, acute kidney injury, shock, and DIC, which was consistent with the previous study of COVID-19.[25] It is noteworthy that patients in the death group had a shorter length of hospital stay compared with the recovered group. This difference probably caused by the rapid progression of COVID-19 in the death group. The patients in the death group were empirically given more corticosteroids, more anti-fungal drugs, and better antibiotics without solid evidence. A limitation of our study is that the initial conditions of the two groups differed. Therefore, these results do not provide conclusive data on the effects of different treatments. More researches are required on the necessity of prophylactically using antibiotics and the time to use them in viral pneumonia patients. Previous studies in corticosteroid therapy suggest that high doses of corticosteroids do not diminish the mortality rate for SARS but tend to result in severe adverse reactions.[26,27] Further research is required to investigate the necessity, dose, and timing of corticosteroid therapy in 2019-nCoV infection. Furthermore, it is found that 45% of patients showed signs of pulmonary fibrosis within one month after being infected with SARS-CoV.[28] Another study found lung fibrosis in 33% of patients who have recovered from MERS-CoV.[29] It is possible that pulmonary fibrosis will become one of the serious complications in patients with 2019-nCoV infection.[30] Drugs for the treatment of idiopathic pulmonary fibrosis such as pirfenidone and nintedanib might be useful in preventing and treating pulmonary fibrosis in patients with 2019-nCoV infection.

In summary, we studied the clinical data of 109 patients who died from 2019-nCoV infection and 116 patients who recovered. Patients in the death group exhibited characteristics of advanced age, more pre-existing comorbidities, dyspnea, oxygen saturation decrease, increased WBC count, decreased lymphocytes, and elevated CRP levels. We hope our study could offer some suggestions to the understanding of this disease.

Conflicts of interest

None.

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