

Clinical Features and Short-term Outcomes of 102 Patients with Coronavirus Disease 2019 in Wuhan, China

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Background. In December 2019, a series of pneumonia cases of unknown cause emerged in Wuhan, Hubei, China. In this study, we investigate the clinical and laboratory features and short-term outcomes of patients with coronavirus disease 2019 (COVID-19).

Methods. All patients with COVID-19 admitted to Wuhan University Zhongnan Hospital in Wuhan, China, between 3 January and 1 February 2020 were included. All those patients were with laboratory-confirmed infections. Epidemiological, clinical, and radiological characteristics; underlying diseases; laboratory tests; treatments; complications; and outcomes data were collected. Outcomes were followed up at discharge until 15 February 2020.

Results. The study cohort included 102 adult patients. The median age was 54 years (interquartile range, 37–67 years), and 48.0% were female. A total of 34 patients (33.3%) were exposed to a source of transmission in the hospital setting (as health-care workers, patients, or visitors) and 10 patients (9.8%) had a familial cluster. There were 18 patients (17.6%) who were admitted to the intensive care unit (ICU), and 17 patients died (mortality, 16.7%; 95% confidence interval, 9.4–23.9%). Those patients who survived were younger, were more likely to be health-care workers, and were less likely to suffer from comorbidities. They were also less likely to suffer from complications. There was no difference in drug treatment rates between the survival and nonsurvival groups. Those patients who survived were less likely to require admission to the ICU (14.1% vs 35.3% of those admitted). Chest imaging examinations showed that patients who died were more likely to have ground-glass opacity (41.2% vs 12.9% in survivors).

Conclusions. The mortality rate was high among the COVID-19 patients described in our cohort who met our criteria for inclusion in this analysis. The patient characteristics seen more frequently in those who died were the development of systemic complications following onset of the illness and a severity of disease requiring admission to the ICU. Our data support those described by others indicating that COVID-19 infection results from human-to-human transmission, including familial clustering of cases, and from nosocomial transmission. There were no differences in mortality among those who did or did not receive antimicrobial or glucocorticoid drug treatments.

Keywords. COVID-19; human-to-human transmission; nosocomial infections; outcome SARS-CoV-2.

In December 2019, a cluster of patients with pneumonia of undetermined etiology was recognized in Wuhan, Hubei, China [1]; subsequently, a novel coronavirus (severe acute respiratory syndrome coronavirus [SARS-CoV]; SARS-CoV-2) was identified from lower respiratory tract samples obtained from affected patients [2]. The virus and its associated disease were given the designation of coronavirus disease 2019 (COVID-19) in February 2020, distinguishing this syndrome from the acute

respiratory syndromes associated with 2 other betacoronaviruses (SARS-CoV and Middle East respiratory syndrome coronavirus) that caused earlier outbreaks of severe disease in humans [3, 4]. A structural analysis suggests that SARS-CoV-2 might be able to bind to the angiotensin-converting enzyme 2 (ACE2) receptor as SARS-CoV in humans [5].

Yang et al [6] declared that the mortality of critically ill patients with SARS-CoV-2 pneumonia was considerable and that older patients (>65 years) with comorbidities and acute respiratory distress syndrome (ARDS) were at an increased risk of death, while another study indicated that as of early February 2020, compared with patients initially infected with SARS-CoV-2 in Wuhan, the symptoms of patients in Zhejiang province were relatively mild [7]. We speculated that the virus can also cause great harm to humans. However, data on the clinical features and short-term outcomes of patients with COVID-19 are still limited. In this study, we investigate the clinical and laboratory features and short-term outcomes of patients with COVID-19.

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METHODS

Patients and Data Collection

All patients with COVID-19 admitted to Wuhan University Zhongnan Hospital in Wuhan, China, between 3 January and 1 February 2020 were included [8]. All those patients had a laboratory-confirmed SARS-CoV-2 infection [9]. It should be noted that our hospital, located in the center of the epidemic area, is 1 of the major tertiary university hospitals and is responsible for the treatments for patients with severe COVID-19. The patients admitted to our hospital had SARS-CoV-2 pneumonia and/or were infected cases with a chronic illness. COVID-19 with minimally symptomatic or asymptomatic SARS-CoV-2 infections were admitted to the cabin hospital. The study was approved by Zhongnan Hospital Ethics Committee, and oral consent was obtained from either patients or their relatives.

Epidemiological, clinical, and radiological characteristics; underlying diseases; laboratory tests at admission and during hospitalization; treatments; complications; and outcomes data were collected [8–10]. Patient outcomes (discharge or death) were followed up from discharge until 15 February 2020 and admission to the intensive care unit (ICU; yes or no) was documented [8]. Throat swab samples were collected for extracting SARS-CoV-2 RNA from patients by real-time reverse transcription polymerase chain reaction (RT-PCR) [9]. A cycle threshold value (Ct value) less than 37 was defined as a positive test result, and a Ct value of 40 or more was defined as a negative test result. A medium load, defined as a Ct value of 37 to less than 40, required confirmation by retesting [9]. The other blood biomarkers were also tested in our hospital laboratory by conventional methods.

Epidemiological information was collected from patients, such as age, sex, body mass index, exposure to a source of transmission within 14 days (yes or no), the incubation period (defined as the time from exposure to the source of transmission to the onset of symptoms), inclusion in a familial cluster (yes or no), whether the patient was a health-care worker (yes or no), and whether the person was a hospitalized patient, outpatient, or visitor (yes or no). Clinical symptoms (fever, dry cough, fatigue, shortness of breath, diarrhea, headache, sore throat, nausea, and vomiting) and comorbidities (hypertension, diabetes, cerebrovascular and cardiovascular disease, respiratory diseases, malignancy, chronic kidney disease, and chronic liver disease) were also obtained. Clinical treatment options were collected and assessed. Drug treatments mainly included antiviral treatment, antibiotic treatment, glucocorticoid treatment, intravenous immunoglobulin therapy, and Chinese medicine treatments. Other treatment options were also recorded, such as oxygen inhalation, noninvasive ventilation, invasive mechanical ventilation, extracorporeal membrane oxygenation, and continuous renal replacement therapy. Clinical complications (lymphopenia, hypoxemia, shock, ARDS, acute infection,

arrhythmia, acute kidney injury, acute liver injury, and acute cardiac injury) during hospitalization were recorded and analyzed. The acute infection was defined by the serum level of procalcitonin (≥ 0.5 ng/ml).

Statistical Analysis

The results were presented as medians (interquartile ranges [IQR]) for continuous variables and numbers (percentages) for categorical variables. The different characteristics between death and survival groups were tested by either a Mann-Whitney U test (continuous variables) or Chi-square test (categorical variables). All statistical analyses were tested in SPSS 22.0 (IBM). A 2-sided α of less than 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

The initial study cohort included 104 adult patients. There were 2 patients excluded because of a transfer during hospitalization, leaving 102 patients for analysis. Demographic details are shown in Table 1. The median age was 54 years (IQR, 37–67 years), and 48.0% were female. A total of 34 patients (33.3%) were exposed to SARS-CoV-2 in the hospital setting (health-care workers [23.5%] and patients and/or visitors [9.8%]) and 10 patients (9.8%) had a familial cluster. The signs and symptoms most commonly seen at admission were a self-reported fever (81.4%), fatigue (54.9%), and a dry cough (49.0%). The timeline of SARS-CoV-2 onset in included patients is shown in Figure 1.

Chest imaging examinations showed that 18 patients (17.6%) had ground-glass opacity. Figure 2 shows chest computed tomographic images of a 42-year-old patient with COVID-19 (a surgeon in our hospital). Common laboratory features at admission included lymphopenia (63.7%), elevated procalcitonin (42.7%), cystatin-C (19.8%), alanine aminotransferase (24.8%), and N-terminal pro-brain natriuretic peptide (37.5%). During hospitalization, the results of those biomarkers had increased to 76.5%, 62.7%, 34.7%, 47.5%, and 62.5%, respectively (Table 2). All patients were treated in isolation. Patients were treated with antiviral, antibiotic, glucocorticoid, and mechanical ventilation at rates of 98.0%, 99.0%, 50.0%, and 19.6%, respectively. The median timing of the initiation of antiviral therapy, relative to the onset of symptoms, was 6 days (IQR, 3–7). As shown in Table 3, the most commonly used antibiotics included Arbidol (34.3%), oseltamivir (64.7%), and lopinavir (27.5%) and the most commonly used antiviral drugs included quinolones (85.3%), cephalosporins (33.3%), carbapenems (24.5%), and linezolid (4.9%). In addition, the most commonly used immunity and glucocorticoid therapies were immunoglobulin (10.8%) and methylprednisolone sodium succinate (50.0%).

During hospitalization, 19.6%, 16.7%, and 14.7% of patients had ARDS, an acute infection, and an acute cardiac injury,

Table 1. Baseline Characteristics, Complications, and Outcomes of Patients With Coronavirus Disease 2019

	All	Non-survivors	Survivors	P
n	102	17	85	
Age, years	54 (37–67)	72 (63–81)	53 (47–66)	<.001
Sex, female	49 (48.0)	4 (23.5)	45 (52.9)	.0512
BMI, kg/m ²	24.4 (21.8–26.0)	26.0 (23.4–28.7)	24.3 (21.8–25.7)	.088
Exposure to source of transmission within 14 days	47 (46.1)	10 (58.8)	37 (43.5)	.374
Familial cluster	10 (9.8)	1 (5.9)	9 (10.6)	.882
Infection				
Health-care workers	24 (23.5)	0	24 (28.2)	.0284
Hospitalized patients and/or outpatients in past 14 days	10 (9.8)	2 (11.8)	8 (9.4)	.882
Signs and symptoms				
Fever	83 (81.4)	12 (70.6)	61 (71.8)	.844
Fatigue	56 (54.9)	9 (52.9)	47 (55.3)	.859
Dry cough	50 (49.0)	8 (47.1)	42 (49.4)	.863
Muscle ache	35 (34.3)	5 (29.4)	30 (34.3)	.641
Diarrhea	11 (10.8)	3 (17.6)	8 (9.4)	.568
More than 1 sign or symptom	92 (90.2)	16 (94.1)	76 (89.4)	.882
Comorbidities				
Any	47 (46.1)	13 (76.5)	34 (40.0)	.006
Hypertension	28 (27.5)	11 (64.7)	17 (20.0)	<.001
Diabetes	11 (10.8)	6 (35.3)	5 (5.9)	<.001
Cerebrovascular disease	6 (5.9)	3 (17.6)	3 (3.5)	.090
Cardiovascular disease	5 (4.9)	3 (17.6)	2 (2.4)	.040
Respiratory diseases	10 (9.8)	4 (23.5)	6 (7.1)	.101
Malignancy	4 (3.9)	1 (5.9)	3 (3.5)	.819
Chronic kidney disease	4 (3.9)	3 (17.6)	1 (1.2)	.012
Chronic liver disease	2 (2.0)	1 (5.9)	2 (2.4)	.462
Incubation period, days, n = 47	3 (2–6)	3 (2–4)	3 (2–6)	.563
Onset of symptom to ..., days				
Hospital admission	6 (3–7)	6 (3–8)	6 (3–7)	.690
Confirmed diagnosis	8 (5–14)	9 (5–16)	8 (5–13)	.577
Transfer to ICU	18 (17.6)	6 (35.3)	12 (14.1)	.082
Length of hospitalization, days	11 (7–15)	9 (6–17)	11 (7–14)	.719
Cost of hospitalization, CNY	18 138 (8436–42 450)	50 779 (30 134–116 821)	14 464 (8707–28 605)	<.001
Treatments				
Oxygen inhalation	76 (74.5)	15 (88.2)	61 (71.8)	.264
Noninvasive ventilation	5 (4.9)	3 (17.6)	2 (2.4)	.040
Invasive mechanical ventilation	14 (13.7)	12 (70.6)	2 (2.4)	<.001
Extracorporeal membrane oxygenation	3 (2.9)	1 (5.9)	2 (2.4)	1.000
CRRT	6 (5.9)	5 (29.4)	1 (2.4)	<.001
Complications				
Shock	10 (9.8)	7 (41.1)	3 (3.5)	<.001
ARDS	20 (19.6)	15 (88.2)	5 (5.9)	<.001
Acute infection	17 (16.7)	14 (82.4)	3 (3.5)	<.001
Acute cardiac injury	15 (14.7)	12 (70.6)	3 (3.5)	<.001
Arrhythmia	18 (17.6)	12 (70.6)	6 (7.1)	<.001
Acute kidney injury	20 (19.6)	15 (88.2)	5 (5.9)	<.001
Acute liver injury	34 (33.3)	13 (76.5)	21 (24.7)	<.001
Lymphopenia	78 (76.5)	17 (100.0)	61 (71.8)	.028
Outcomes at discharge				
Discharge	85 (83.3)	
Died	17 (16.7)	
MODS	10 (58.8)	
ARDS	1 (5.9)	
Cardiac arrest	4 (23.5)	
Respiratory failure	2 (11.8)	

The results were presented as medians (IQRs) for continuous variables and n (%) for categorical variables. The different characteristics between nonsurvivors and survivors were tested by either the Mann-Whitney U test (continuous variables) or Chi-square test (categorical variables).

Abbreviations: ARDS, acute respiratory distress syndrome; BMI, body mass index; CNY, Chinese yuan; CRRT, continuous renal replacement therapy; ICU, intensive care unit; IQR, interquartile range; MODS, multiple organ dysfunction syndrome.

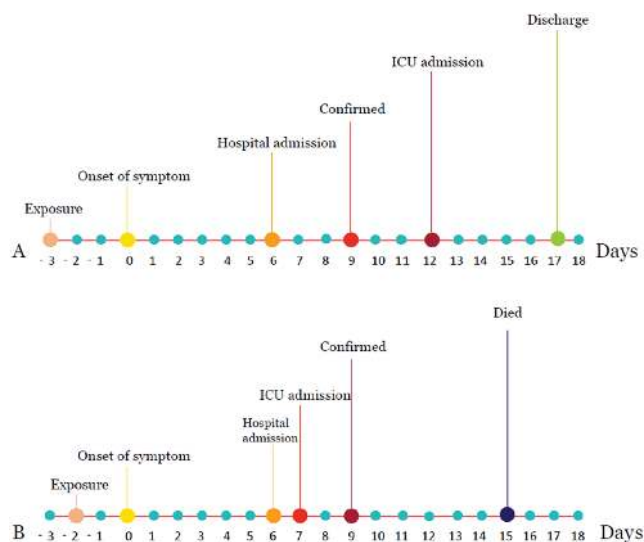


Figure 1. The timeline of SARS-CoV-2 onset in included patients. *A*, The timeline of SARS-CoV-2 onset in survivors ($n = 85$). *B*, The timeline of SARS-CoV-2 onset in nonsurvivors ($n = 17$). The onset of symptom was defined as Day 0. ICU admission time represents those patients who were admitted to an ICU ($n = 12$ in the survivors; $n = 6$ in the nonsurvivors). The points represent the median value. Abbreviations: ICU, intensive care unit; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

respectively. There were 18 patients (17.6%) admitted to the ICU, and 17 patients died (mortality, 16.7%; 95% confidence interval [CI], 9.4–23.9%; Table 1). The most common cause of death was multiple organ dysfunction syndrome (58.8%). In addition, 4 (23.5%) out of the 17 patient deaths were caused by cardiac arrest.

Mortality

The median duration from the onset of symptoms to death was 15 days (IQR, 9–21) and the median time from exposure to SARS-CoV2 to death was 17 days (IQR, 12–24). The patients who survived were younger (53 years [IQR, 47–66] vs 72 years [IQR, 63–81], respectively), were more likely to be health-care workers (28.2% vs 0, respectively), and were less likely to suffer from comorbidities (hypertension [20.0% vs 64.7%, respectively], diabetes [5.9% vs 35.3%, respectively], and chronic kidney disease [1.2% vs 17.6%, respectively]) than the patients who died. Survivors were also less likely than nonsurvivors to have suffered from complications such as shock (3.5% vs 41.1%, respectively), ARDS (5.9% vs 88.2%, respectively), acute infection (3.5% vs 82.4%, respectively), acute cardiac injury (3.5% vs 70.6%, respectively), arrhythmia (7.1% vs 70.6%, respectively), acute kidney injury (5.9% vs 88.2%, respectively), acute liver injury (24.7% vs 76.5%, respectively), and lymphopenia (71.8% vs 100.0%, respectively; Table 1). There were no differences in drug treatment rates between the survival and nonsurvival groups (antiviral therapy [$P = .749$], antibiotic treatment [$P = .369$], glucocorticoid therapy [$P = .184$], intravenous immunoglobulin therapy

[$P = .253$], and Chinese medicine treatment [$P = 1.000$]; Table 3). As shown in Table 3, survivors were more likely than nonsurvivors to have received treatment with Arbidol (37.6% vs 5.9%, respectively; $P = .011$) and less likely to have received treatment with carbapenems (17.6% vs. 58.8%; $P < .001$) and linezolid (2.4% vs. 17.6%; $P = .040$).

Patients who survived were less likely than nonsurvivors to have required admission to the ICU (14.1% vs 35.3%, respectively). They required longer hospital stays (11 days [IQR, 7–14] vs 9 days [IQR, 6–17], respectively) and had lower hospital expenses (14 464 Chinese yuan [IQR, 8707–28 605] vs 50 779 Chinese yuan [30 134–116 821]). During hospitalization, those patients who did not survive were more likely to have elevated procalcitonin (100.0% vs 76.5%, respectively), cystatin-C (100.0% vs 70.6%, respectively), alanine aminotransferase (82.2% vs 41.1%, respectively), D-dimer (100.0% vs 47.1%, respectively), troponin I (80.0% vs 40.0%, respectively), and N-terminal pro-brain natriuretic peptide (100.0% vs 80.0%, respectively) than those patients who survived (Table 2). Chest imaging examinations showed that patients who died were more likely than those who survived to have ground-glass opacity (41.2% vs 12.9%, respectively).

DISCUSSION

The mortality rate was high among the COVID-19 patients described in our cohort. Patient characteristics seen more frequently in those who died were the development of systemic complications following onset of the illness and a severity of disease requiring admission to the ICU. Furthermore, more intensive supportive care in the ICU might improve outcomes; however, the mortality rate was higher for those who were transferred to the ICU, likely reflecting their underlying disease severity and comorbidities [8].

Our findings and previous studies [1, 2, 9–11] show that lymphopenia is common in cases with SARS-CoV-2 infection, suggesting that SARS-CoV-2 consumes many immune cells and inhibits the body's cellular immune function. In this study, most of the deaths were caused by multiple organ dysfunction syndrome, suggesting that the impaired immune function is an important cause of death. Furthermore, we have reason to believe that the immune system was mobilized and a cytokine storm was formed [1, 12]. In a SARS-CoV infected mouse model, researchers showed that apart from the respiratory system, the heart was also infected with the coronavirus, with a down-regulated expression of ACE2 [13]. In this study, we confirmed that nearly a quarter of our patient deaths were caused by cardiac arrest.

Our data support those described by others, indicating that COVID-19 infection results from human-to-human transmission, including familial clustering of cases, and from nosocomial transmission [2, 9, 10]. We showed that 33.3% of the included

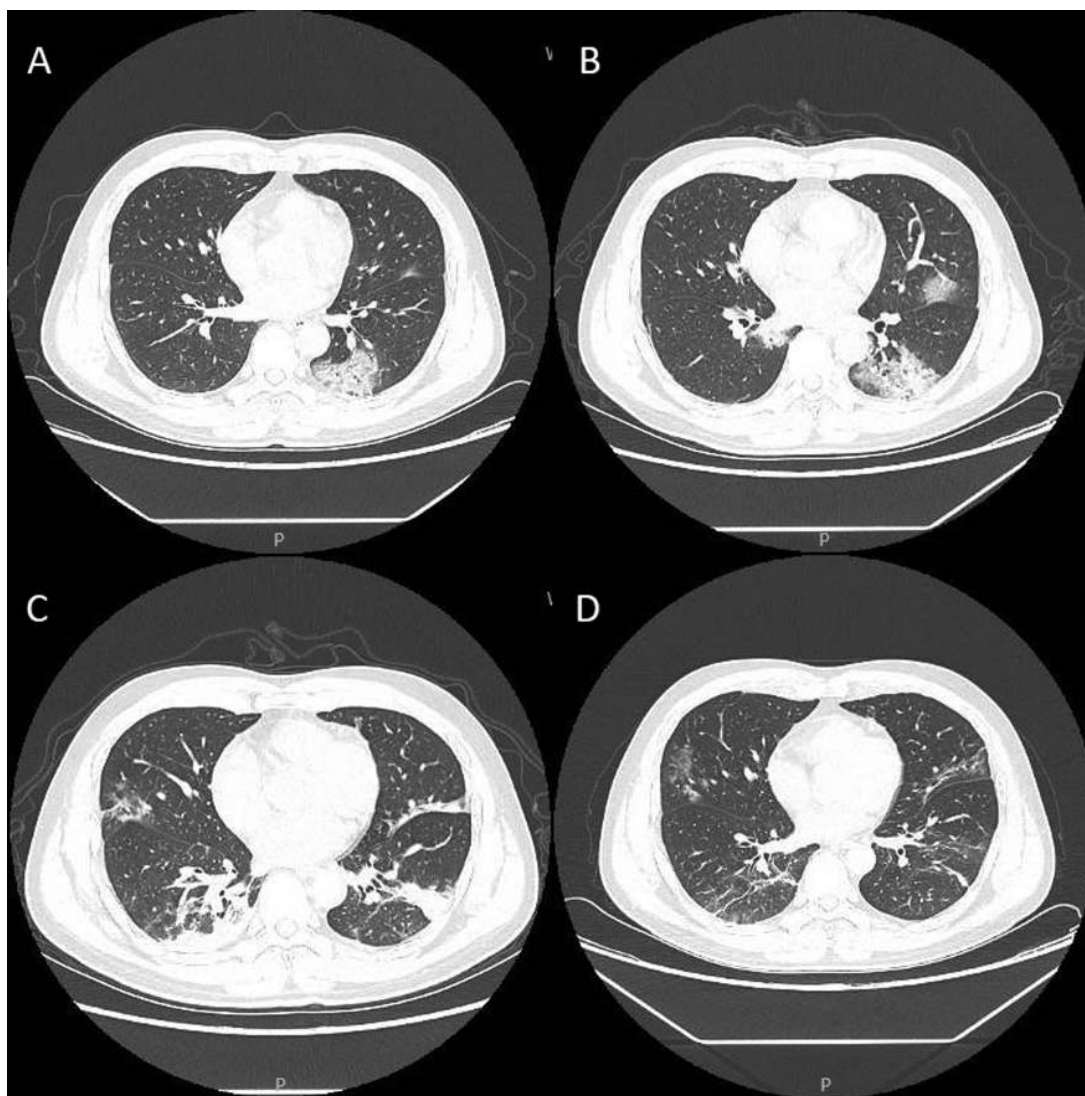


Figure 2. Chest computed tomographic images of a 42-year-old patient infected with SARS-CoV-2. *A*, Computed tomography images on Day 5 after symptom onset. *B*, Computed tomography images on Day 8 after symptom onset. *C*, Computed tomography images on Day 14 after symptom onset. *D*, Computed tomography images on Day 18 after symptom onset. This patient recovered and was discharged on Day 26 after symptom onset. Abbreviation: SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

patients were exposed to SARS-CoV-2 in the hospital setting. It might be due to the fact that many of our infected staff were admitted to our hospital. It was sad that in the early days of the COVID-19 outbreak, we did not know much about the disease, and hospitals and doctors did not have adequate protection. Beginning 20 January 2020, all medical workers in our hospital started to use protective clothing and goggles. Furthermore, since coronavirus diffusion takes place by droplet transmission, aerosolization during hospital procedures, like intubation or bronchoscopy, might represent a big concern, exposing other patients and health-care staff to an increased risk of infection, as during the flu pandemic [14]. However, in our study, some potential confounders, such as a small sample size, a single patient type (mainly hospital staff and patients with moderate to severe symptoms), and a lack of discharge information should not be

ignored. Further studies are warranted to explore the natural history of COVID-19.

In this study, the mortality was 16.7%, which was higher than rates reported in previous studies (range, 4.3% to 11.0%) [1, 9, 10]. It should be noted that a significant number of patients were still in hospital at the time of those reports, and the mortality continued to rise following those previous studies [1, 9, 10]. All the patients in our study had been discharged or died. Our results were more likely close to real results. As of 28 February 2020, the national official statistic shows that the mortality rates in Wuhan and China are 4.47% (2169/48 557) and 3.58% (2835/79 251), respectively [15]. Furthermore, 4691 patients with SARS-CoV-2 infections were reported overseas (51 countries), with 67 fatal cases (1.43%) [16]. Those results showed that the mortality rate of COVID-19 was lower than those of

Table 2. Radiologic and Laboratory Findings of Patients With Coronavirus Disease 2019 on Admission to Hospital and During Hospitalization

	All, n = 102		Non-survivors, n = 17	
	Admission	Hospitalization	Admission	Hospitalization
Radiologic findings (X-ray and CT)				
Local patchy shadowing	30 (29.4)	No change, 20 (66.7)	3 (17.6)	No change, 0 (0)
...		Bilateral infiltrate, 10 (33.3)	...	Bilateral infiltrate, 3 (100.0)
Bilateral patchy shadowing	72 (70.6)	NA	14 (82.4)	NA
Ground-glass opacity	18 (17.6)	NA	7 (41.2)	NA
Laboratory findings				
Lymphocyte count, $\times 10^9/L$	0.9 (0.8–1.2)	0.7 (0.6–1.1)	0.8 (0.7–1.2)	0.6 (0.5–1.0)
$\leq 1.1 \times 10^9/L$	65/102, 63.7%	78/102, 76.5%	11/17, 64.7%	17/17, 100%
C-reactive protein, mg/L	24.8 (6.7–55.7)	32.9 (13.0–84.7)	118.8 (39.9–160.0)	145.7 (102.0–256.3)
≥ 10 mg/L	52/102, 51.0%	64/102, 62.7%	16/16, 100%	12/12, 100%
Procalcitonin level, ng/mL				
≥ 0.1 ng/mL	35/82, 42.7%	48/82, 58.6%	13/17, 76.5%	17/17, 100%
ALT, U/L	23 (16–40)	38 (19–72)	40 (21–56)	72 (44–92)
≥ 40 U/L	25/101, 24.8%	48/101, 47.5%	7/17, 41.1%	14/17, 82.2%
Blood urea nitrogen, mmol/L	4.33 (3.45–5.46)	5.01 (3.78–7.39)	6.68 (4.80–9.37)	21.33 (10.11–36.73)
≥ 7.6 mmol/L	13/101, 12.9%	27/101, 26.7%	7/17, 41.4%	17/17, 100.0%
UA, umol/L	269 (228–347)	280 (236–387)	396 (304–485)	501 (389–597)
≥ 360 umol/L	24/101, 23.8%	30/101, 29.7%	9/17, 52.9%	14/17, 82.4%
Cys-C, mg/L	0.99 (0.82–1.13)	1.03 (0.84–1.31)	1.39 (1.14–2.45)	3.00 (1.82–4.74)
≥ 1.2 mg/L	20/101, 19.8%	35/101, 34.7%	12/17, 70.6%	17/17, 100.0%
D-dimer, mg/L	195 (133–432)	525 (255–595)	276 (204–474)	1050 (745–1740)
≥ 500 mg/L	21/101, 20.8%	53/101, 52.5%	8/17, 47.1%	17/17, 100.0%
Hypersensitive troponin I, pg/mL	7.6 (3.2–11.0)	8.0 (3.0–35.7)	21.5 (9.4–44.1)	208.5 (35.7–580.2)
≥ 26 pg/mL	7/55, 12.7%	15/55, 27.3%	6/15, 40.0%	12/15, 80.0%
BNP, pg/mL	12.2 (0–63.1)	44.4 (10.0–175.8)	46.1 (14.7–221.4)	273.7 (44.4–1325.1)
≥ 100 pg/mL	5/35, 14.3%	12/39, 30.8%	6/15, 40.0%	10/15, 66.7%
NT-proBNP, pg/mL	417 (132–1800)	448 (231–2100)	1165 (686–10 700)	4740 (2580–23 850)
≥ 900 pg/mL	6/16, 37.5%	10/16, 62.5%	12/15, 80.0%	15/15, 100.0%

If 1 patient had several blood samples tested during hospitalization, we would choose the highest one. The threshold of those blood markers is determined by the laboratory of our hospital.

Abbreviations: ALT, alanine aminotransferase; BNP, brain natriuretic peptide; CT, computed tomography; Cys-C, cystatin-C; NT-proBNP, N-terminal pro-brain natriuretic peptide; UA, uric acid.

Table 3. The Drug Treatment of Patients With Coronavirus Disease 2019 During Hospitalization

	All	Non-survivors	Survivors	P
n	102	17	85	
Antiviral therapy	100 (98.0)	17 (100.0)	83 (97.6)	.749
Arbidol hydrochloride capsules	33 (34.3)	1 (5.9)	32 (37.6)	.011
Oseltamivir	66 (64.7)	14 (82.4)	52 (61.2)	.095
Lopinavir and ritonavir tablets	28 (27.5)	4 (23.5)	24 (28.2)	.921
Antibiotic treatment	101 (99.0)	17 (100.0)	84 (98.8)	.369
Cephalosporins	34 (33.3)	5 (29.4)	29 (34.1)	.707
Quinolones	87 (85.3)	13 (76.5)	74 (87.1)	.453
Carbapenems	25 (24.5)	10 (58.8)	15 (17.6)	<.001
Linezolid	5 (4.9)	3 (17.6)	2 (2.4)	.040
Intravenous immunity therapy	11 (10.8)	0 (0)	11 (12.9)	.253
Immunoglobulin	11 (10.8)	0 (0)	5 (5.9)	.682
Thymosin alpha for injection	9 (8.8)	0 (0)	9 (10.6)	.349
Glucocorticoid therapy				
Methylprednisolone sodium succinate	51 (50.0)	11 (64.7)	40 (47.1)	.184
Chinese medicine treatment				
Lianhuaqingwen capsule	3 (2.9)	0 (0)	3 (3.5)	1.000

The results were presented as n (%) for categorical variables. The different characteristics between nonsurvivors and survivors were tested by Chi-square test (categorical variables).

SARS and Middle East respiratory syndrome coronavirus, which had mortality rates up to 10% and 37%, respectively [1]. Wu et al [17] estimated a risk of fatality among hospitalized patients with COVID-19 at 14% (95% CI, 3.9–32%). Importantly, Liu et al [18] showed that the reproductive numbers (R) of SARS-CoV-2 and SARS were 2.90 (95% CI, 2.32–3.63) and 1.77 (95% CI, 1.37–2.27, respectively). Those results illustrated that SARS-CoV-2 may have a higher pandemic risk than the SARS from 2003 [19]. Therefore, international collaboration among scientists is essential to address these risks and prevent the next pandemic [20].

As SARS-CoV-2 is an emerging virus, an effective treatment has not been confirmed. Russell et al [21] suggested that corticosteroid treatment should not be used for the treatment of a COVID-19-induced lung injury or shock outside of a clinical trial. In this study, we found that most forms of treatment had no impact on survival. Even patients who died received more carbapenems ($P < .001$) and linezolid ($P = .040$). It was exciting that Arbidol seems to improve the prognosis ($P = .011$). Furthermore, 1 study had identified 4 small molecular drugs (prulifloxacin, nelfinavir, bictegravir, nelfinavir) with high binding capacities with the SARS-CoV main protease by high-throughput screening [22]. Furthermore, remdesivir and chloroquine could effectively inhibit SARS-CoV-2 in vitro [23] and baricitinib also has been suggested as a potential treatment for COVID-19 [24]. Further clinical trial studies need to validate these hypotheses.

Our study suffers from the usual limitations of small samples and a single center. Our hospital is 1 of the major tertiary teaching hospitals and is responsible for treating critically ill patients with COVID-19. Thus, our cohort might represent the more severe COVID-19 cases and overestimate the real mortality rates. A recent large-sample, multicenter study showed that only 5.00% of the included COVID-19 patients were admitted to an ICU and that 1.36% died [25]. Also, we only recorded 17 patient deaths. Therefore, we did not perform logistic regression analyses to assess the risk factors for death. Thus, continued observations of the natural history of the disease are needed. Third, our study mainly includes adult patients, which might cause a selective bias. Pregnant women [26] and children [27] also are equally sensitive to the SARS-CoV-2 virus. Lastly, we only included laboratory-confirmed patients. In fact, RT-PCR assays have a considerable percentage of false negatives [28]. Huang et al [28] suggested the use of chest computed tomography in combination with a negative RT-PCR assay for the SARS-CoV-2 should cause a high clinical suspicion of an infection.

CONCLUSION

In conclusion, the mortality rate was high among the COVID-19 patients described in our cohort who met our criteria for inclusion in this analysis. The patient characteristics seen more

frequently in those who died were the development of systemic complications following onset of the illness and a severity of disease requiring admission to the ICU. Our data support those described by others indicating that COVID-19 infections result from human-to-human transmission, including familial clustering of cases, and from nosocomial transmission. There were no differences in mortality among those who did or did not receive antimicrobial or glucocorticoid drug treatments.

Notes

Author contributions. J. C. and W.-J. T. contributed equally to this work as coauthors, had full access to all of the data in the study, conducted the statistical analysis, and take responsibility for the integrity of the data and the accuracy of the data analysis. W.-J. T., X. H., and Q. L. contributed equally as senior authors. J. C., X. H., W. C., L. Y., W.-J. T., and Q. L. conceived of and designed the study. J. C., X. H., W. C., Y.-K. L., L. Y., W.-J. T., and Q. L. acquired, analyzed, or interpreted the data. J. C., X. H., W.-J. T., and Q. L. drafted the manuscript. W. C., Y.-K. L., and L. Y. critically revised the manuscript for important intellectual content. J. C., X. H., W. C., W.-J. T., and Q. L. provided administrative, technical, or material support. J. C., X. H., and W. C. supervised the research. W.-J. T. and Q. L. obtained the study funding. The data available can be obtained from the corresponding author.

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