Characteristics of patients with COVID-19 during epidemic ongoing outbreak in Wuhan, China

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Abstract

Background Since Dec 2019, SARS-CoV-2 has caused about fifty thousand patients and over two thousand deaths in Wuhan, China. We reported characteristics of patients with COVID-19 during epidemic ongoing outbreak in Wuhan.

Methods Data of COVID-19 patients with clinical outcome in a designated hospital in Wuhan, were retrospectively collected from electronic medical records. Characteristics were compared between patients who died or recovered, and between patients with different disease severity.

Results By Feb 25, 2020, 403 patients were enrolled, 100 died and 303 recovered. Most of non-survivors tended to be males, old aged, or with chronic diseases. Duration from illness onset to admission was 9 (7-12) days. Patients with severe or critical illness had more days from onset to admission compared to those with ordinary illness. Lymphopenia, anemia, hypoproteinemia, and abnormal serum sodium were presented in 52.6%, 54.6%, 69.8%, and 21.8% cases, respectively. Patients who died or with severe/critical illness showed increased white blood cell and neutrophil count, serum total bilirubin, creatinine, hypersensitive troponin I, D-dimer, procalcitonin, and C-reactive protein, and decreased red blood cell, lymphocyte, platelet count, and serum albumin on admission compared to those who recovered or with ordinary illness. Complications of acute organ injury and secondary infection were common in patients with COVID-19, especially in non-survivors.

Conclusions Multiple homeostasis disturbances were common in patients with severe or critical illness at admission. Early support should be provided, especially for old men with chronic disease, which is vital to control disease progression and reduce mortality of COVID-19 during epidemic ongoing outbreak.

Key words COVID-2019; SARS-CoV-2; homeostasis disturbance; inflammatory response; early intervention

Introduction

In late December 2019, cluster cases of pneumonia of unknown etiology were reported by local health facilities in Wuhan, China.[1] A novel coronavirus was identified as the pathogen of this acute respiratory illness, which was named SARS-Cov-2 by World Health Organization (WHO) for its similarity in gene sequence to severe acute respiratory syndrome coronavirus (SARS-CoV).[2,3] The disease caused by SARS-Cov-2 was latterly designated COVID-19.[4] The SARS-CoV-2 epidemic has rapidly spread from Wuhan to other areas. On 30 Jan 2020, WHO declared SARS-CoV-2 epidemic a public health emergency of international concern.[3] As a newly discovered virus, SARS-CoV-2 has brought about much more serious global health threats than the two former major pandemics by other members of coronavirus family, SARS-CoV and Middle East respiratory syndrome coronavirus (MERS-CoV).[5] As of Mar 6, 2020, a total of 98192 cases of COVID-19 have been confirmed globally, including 80711cases in China and 17481 cases in other 88 countries/territories/areas. Of them, 3380 patients died, 3045 in China and 335 in abroad.[6]

Data from the early epidemic revealed that common symptoms of SARS-CoV-2 infection included fever, fatigue, dry cough, and shortness of breath, and older males with comorbidities are more susceptible to the SARS-CoV-2 infection and progressive to severe and even fatal respiratory diseases.[7,8] Human-to-human transmission has been confirmed mainly through respiratory droplets and close contact.[8,9] The management of patients with SARS-CoV-2 infection is mainly symptomatic treatment and life support for severely and critically ill patients. Anti-viral treatment has been listed in protocols issued by the Chinese National Health Commission (NHC), however, these antiviral agents have yet to be confirmed effective for SARS-CoV-2.[9] Currently, SARS-CoV-2 has infected most patients and caused most deaths in Wuhan, China. However, little information focusing on final outcome and disease severity on admission can be obtained associated with clinical characteristics during the ongoing outbreak of SARS-CoV-2 pneumonia. In this retrospective study, we aimed to describe and compare the clinical features of patients with COVID-19 related to final outcome and disease severity on admission during its ongoing outbreak in Wuhan, China.

Materials and methods

Study design and participants

For this retrospective, single-center study, patients who admitted to Eastern Campus of Renmin Hospital, Wuhan University, were recruited from Jan 30 to Feb 25, 2020. Renmin Hospital of Wuhan

University is a teaching hospital affiliated to Wuhan University in Wuhan, China. With epidemic

ongoing outbreak, Eastern Campus of our hospital was requisitioned as one of designated hospitals for

SARS-CoV-2 infection on Jan 25, 2020. After being transformed into an infectious disease hospital,

Eastern Campus began to admit patients who were diagnosed as COVID-19 according to protocol[9] on

Jan 30, 2020. Clinical outcome of all patients was reviewed till Feb 25, 2020. Patients who were still

hospitalized by that time were excluded in this study. This study was approved by the Research Ethics

Committee of Renmin Hospital of Wuhan University (approval number: WDRM 2020-K038). Written

consent was not required for patients since it was a retrospective, observational study.

Data collection

Information of each patient was obtained mainly through screening Electronic Health Records and

Laboratory Information Management System supplied by DHC Software Co., Ltd (Beijing, China).

Nursing records were also reviewed if necessary. Patients' demographic information, history of

smoking or drinking, chronic diseases, and onset symptoms, were collected, as well as duration from

illness onset to hospital admission and disease severity on admission. Indexes of laboratory test on

admission were evaluated, including complete blood count, C–reactive protein (CRP), blood

biochemistry, myocardial enzymes, and coagulating function. Findings of chest computed tomographic

scan (CT) or x-ray film on admission were reviewed and listed. Disease severity (ordinary, severe, or

critical) was determined according to the guidance issued by NHC[9]. Treatment measures evaluated in

this research contained high-flow nasal cannula oxygen therapy (HFNC), non-invasive mechanical

ventilation (NIMV) or invasive mechanical ventilation (IMV), blood purification treatment, application

of antiviral agents, antibacterial agents, anti-fungal agents, corticosteroids, and immunoglobulin. The

primary endpoint of this study was patients' clinical outcome (died or recovered), and secondary

endpoint was disease severity on admission. Criteria for recovery were as follows: temperature

returned to normal for more than 3 days, respiratory symptoms improved significantly, and negative

results of two consecutive SARS-CoV-2 RNA detection in respiratory samples (sampling interval at

least 1 day).[9]

Statistical analysis

Categorical variables were presented as number (%), and continuous variables were described as mean

(standard deviation, SD) for normally distributed data or median (interquartile range, IQR) for

abnormally distributed data. Independent t test was used to compare continuous variables with normal

distribution; otherwise, the Mann-Whitney test was used. Chi-squared test was conducted to compare

categorical variables. All statistical analyses were performed using IBM SPSS Statistics 19.0 (SPSS

Inc.). A two-sided test of $\alpha < 0.05$ was considered statistical significance.

Results

By Feb 25, 2020, 985 patients with confirmed COVID-19 were admitted to Eastern Campus of Renmin

Hospital of Wuhan University. Five hundred and seventy four patients were still in hospitalization at

that time. Four hundred and eleven patients had clinical outcome, among them 8 died on admission day.

Consequently, 403 cases were finally enrolled in this study, 100 died and 303 recovered (figure 1).

The median age was 56 (39–68) years, 193 (47.9%) were males. Non-survivors had a significantly

older age than survivors (median 71 (65–80) years vs. 49 (37–62) years, p<0.001). The mortality was

obviously higher in males than in females. Twenty nine (7.2%) patients had history of smoking, and 41

(10.2%) had history of drinking. History of smoking or drinking was not associated with clinical

outcome. One hundred and seventy five (43.4%) patients had chronic diseases. Non-survivors had a

higher incidence of chronic diseases compared to survivors (77.0% vs. 32.3%, p<0.001). One hundred

and thirteen (28%) patients had hypertension. Thirty nine (9.7%) patients had cerebrovascular diseases.

Fifty seven (14.1%) patients had diabetes. Besides, coronary heart disease, chronic pulmonary disease,

cirrhosis, anemia, and chronic kidney disease appeared in 36 (8.9%), 28 (6.9%), 25 (6.2%), 15 (3.7%),

and 7 (1.7%) patients, respectively. Fever accounted for the most main symptom of onset (79.2%).

Other main onset symptoms were cough (10.2%) and fatigue (3.5%). The median duration from onset

of symptoms to hospital admission was 9 (7–12) days. On admission, all patients had abnormal lung

image manifested by chest computed tomography (CT) scan or x-ray film. The typical abnormality in

chest CT image was bilateral ground glass opacity and subsegmental consolidation. Bilateral

involvement appeared in 376 (93.3%) patients (table 1).

Laboratory findings of patients with COVID-19 were listed in table 2 and table 3. Results of complete

blood cell count test showed anemia in 220 (54.6%) and lymphopenia in 212 (52.6%) cases.

Non-survivors exhibited significantly higher white blood cell and neutrophil count, but lower red blood

cell, lymphocyte and platelet count on admission than survivors. Two hundred and seventy out of 387

patients (69.8%) had serum albumin level below 40 g/L on admission. Increased serum total bilirubin

and creatinine were observed in 10.9% (42/387) and 12.3% (48/390) patients, respectively.

Non-survivors had much larger proportion of elevated total bilirubin (27.6% vs. 5.2%, p<0.001) and

creatinine (28.6% vs. 6.8%, p<0.001) than survivors. Raised serum levels of hypersensitive troponin I

(median 0.035 (0.015–0.181) ng/mL vs. 0.006 (0.006-0.008) ng/mL, p<0.001), D-dimer (median 5.38

(1.21–17.78) mg/L vs. 0.50 (0.27–1.07) mg/L, p<0.001), C-reactive protein (median 95.0 (58.9–178.7)

mg/L vs. 23.8 (5.0–49.9) mg/L, p<0.001), and procalcitonin (median 0.199 (0.116–0.949) ng/mL vs.

0.044 (0.026–0.070) ng/mL, p<0.001) were observed in non-survivors compared to survivors.

Abnormal serum sodium was observed in 21.8% (88) cases. Non-survivors showed lower serum

sodium (median 141.1 (136.6–144.2) mmol/L vs. 143.2 (139.6–144.3) mmol/L, p=0.001) and higher

incidence of abnormal serum sodium (48% vs. 13.2%, p<0.001) compared to survivors on admission .

Two hundred and five (50.9%) cases were diagnosed as severe or critical illness, and 198 were

ordinary illness on admission, according to the criteria issued by NHC[9]. Patients with severe or critical

illness presented older age (median 66 (56–74) years vs. 43 (33–56) years, p <0.001), higher

proportions of male gender (56.6% vs. 38.9%, p <0.001) or chronic diseases (58.0% vs. 28.3%, p

<0.001), and more delayed hospitalization (median 10 (7–13) days vs. 8 (5–11) days, p <0.001) than

those with ordinary illness. Decreased lymphocyte (median 0.90 (0.54–1.24) $\times 10^9$/L vs. 1.22

(0.94–1.70) $\times 10^9$/L, p <0.001) and platelet count (median 179 (136–245) $\times 10^9$/L vs. 205 (154–261) $\times$

10^9/L, p <0.001), and serum albumin level (median 34.7 (32.3–37.9) g/L vs. 39.9 (37.3–42.2) g/L, p

<0.001) were observed in patients with severe or critical illness compared to those with ordinary

disease. The incidence of increased serum total bilirubin (16.6% vs. 4.0%, p <0.001) and creatinine

(16.7% vs. 4.3%, p =0.002) in patients with severe or critical disease were obviously higher than

patients with ordinary disease. Patients with severe or critical illness showed markedly elevated serum

level of hypersensitive troponin I (median 0.012 (0.006–0.062) ng/mL vs. 0.006 (0.006–0.006) ng/mL,

p <0.001), D-dimer (median 1.22 (0.52–7.46) mg/L vs. 0.39 (0.23–0.83) mg/L, p <0.001), C-reactive

protein (median 59.7 (20.7–103.5) mg/L vs. 6.8 (5.0–25.7) mg/L, p <0.001), and procalcitonin (median

0.10 (0.05–0.26) ng/mL vs. 0.04 (0.02–0.06) ng/mL, p <0.001) compared to those with ordinary illness.

Decreased serum sodium appeared in patients with severe or critical illness compared to those with

ordinary illness (median 141.2 (137.9–143.9) mmol/L vs. 143.5 (142.2–145.0) mmol/L, p<0.001). The

proportion of abnormal serum sodium was significantly lower in patients with ordinary illness

compared to those with severe or critical illness (12.2% vs. 32.2%, p<0.001).

Complications of acute organ injury and secondary infection were common in patients with COVID-19,

especially in non-survivors. The incidence of acute respiratory distress syndrome (90% vs. 17.5%),

acute liver injury (71% vs. 5.3%), cardiac injury (65% vs. 5.9%), kidney injury (43% vs. 4.6%), and secondary infection (58% vs. 6.3%) in patients who died was significantly higher than those who recovered (all $p<0.001$).

HFNC was used in 106 (26.3%) cases. NIMV and IMV were provided in 56 (13.9%) and 23 (5.7%) cases, respectively. Thirty nine (9.7%) patients received blood purification treatment. Antibiotics and anti-fungal agents were given to 349 (86.6%) and 14 (3.5%) cases respectively. Antiviral agents were given to 394 (97.8%) cases. Specifically, abidol was used in 324 (80.4%), oseltamivir in 123 (30.5%), ribavirin in 81 (20.1%), ganciclovir in 53 (13.2%), and recombinant interferon in 72 (17.9%). Almost half (49.1%) patients received immunoglobulin injection. One hundred and sixty-six (41.2%) patients received systemic application of glucocorticoids.

Discussion

To our knowledge, this report presented the largest sample of COVID-19 patients with clinical outcome. Our results confirmed the findings by Chen that male gender, older age, and chronic disease contributed to death caused by SARS-CoV-2 infection.[7] Patients with severe or critical illness on admission presented a more delay to admission, implying the effect of delayed admission on disease deterioration. The ongoing outbreak of this epidemic incurred a sharply increased patients in Wuhan, China, and most of them couldn't be admitted to hospitals in time. Frequent visits to fever clinics, inappropriate home quarantine, and delayed hospitalization further aggravated the disease progression and transmission of the virus. Multiple homeostasis disturbances, including lymphopenia, anemia, hypoproteinemia and abnormal serum sodium were observed on admission, especially in patients with severe or critical illness. Many factors might contribute to these disturbances, such as eating less due to poor appetite for food, augment of organic consumption, and simultaneous gastrointestinal problems. Insufficient nutrient intake and augmented consumption might deteriorate tissue metabolism and damage one's immunity to virus.

Our study showed that multiple organ damage, such as ARDS, acute kidney injury, acute cardiac injury, and acute liver injury, were common complications in patients with COVID-19. Previous studies have showed that angiotensin-convertion enzyme 2 (ACE2), the receptor for SARS-CoV-2, is expressed in organs including lung, heart, liver, and kidney. By binding to ACE2, SARS-CoV-2 can provide direct attacks to these organs.[10-12] Serum CRP level on admission was evidently elevated in patients with COVID-19, especially in those with severe or critical illness, indicating excessive inflammatory stress,

which was consistent with the elevated serum pro-inflammatory cytokines observed in COVID-19

patients.[13,14] Meanwhile, serum levels of some laboratory indexes such as total bilirubin, creatinine and

hypersensitive troponin I were increased in patients on admission. This trend was more obvious in

patients with severe or critical illness, indicating signs of organ damage. Though the pathological

mechanism of COVID-19 is yet to be defined, direct attacks from SARS-CoV-2 and organ damage

caused by excessive inflammatory response (cytokines storm) might be responsible for the

pathogenesis of disease progression.[14] Secondary infection, another main complication in this study,

appeared mostly in non-survivors, indicating an important booster of disease progression.

Comprehensive treatments were provided for patients in this study. No antiviral treatment for

coronavirus infection has been proven to be effective, however, antiviral agents were applied in almost

(97.8%) patients with COVID-19. The main agents used included abidol, oseltamivir, ribavirin,

ganciclovir, and recombinant interferon. Remdesivir and chloroquine are considered two promising

agents for SARS-CoV-2 infection,[15] which were given to some patients in randomized, double-blind,

placebo-controlled clinical trials. However, it's hardly to assess the efficacy and safety of remdesivir

and chloroquine until results of the trials are released. Short-term (3 to 5 days) application of

glucocorticoids is suggested for severe patients with COVID-19 in the protocol issued by NHC.[9] In this

study, glucocorticoids were applied in 166 (41.2%) cases, most of whom were patients with severe or

critical illness. However, glucocorticoids use was found to be correlated with increased mortality in

SARS patients and delayed virus clearance.[16,17] In this context, glucocorticoids are not routinely

recommended in treatment of SARS-CoV-2 pneumonia.[18] In this study, secondary infection appeared

mostly in patients with glucocorticoids use and might contribute to death, which was indicated by its

higher incidence in patients who died (58.0% vs. 6.3%, p<0.001). And so, glucocorticoids should be

used more cautiously in patients with COVID-19.

High-flow nasal cannula oxygen therapy (HFNC) and mechanical ventilation are recommended for

patients with ARDS.[19] Non-invasive positive pressure ventilation (NIPPV) has advantage in improving

oxygenation of patients with moderately or severely hypoxic respiratory failure (with oxygenation

index PaO2/FiO2 100 to 150), however, it does not reduce rate of endotracheal intubation and ICU

mortality.[20] For patients with ARDS, invasive ventilation with strategy of pulmonary protection is

strongly recommended.[21] In this study, many patients with severe ARDS were not provided IMV

treatment. Inadequate application of invasive ventilation in this study was mainly due to the shortage of

intensive care unit (ICU) resource for so many critically ill patients accumulated during epidemic ongoing outbreak. The worry about aerosol transmission of virus during endotracheal intubation was possibly another reason for some medical staffs.[22]

There are some limitations in our study. First, current criteria for recovery are debatable since positive result of SARS-CoV-2 RNA detection appeared in some discharged patients after one to two weeks of continuous quarantine.[23] Hence, some discharged patients may not truly recover. Second, the agents given to patients such as antibiotics, antivirus agents and anti-fungals, might exert toxicity to organs, which confounded judgment of organ damage by virus or excessive inflammatory stress. Besides, chronic diseases might also have effect on laboratory results on patients' admission, especially in those with severe or critical illness. Third, inflammatory mediators, such as IL-1, IL-6, TNF-α, commonly being used to predict cytokines storm,[14] were not included due to detection in just a small fraction of patients in this study. However, serum CRP level was detected for most patients in this study, which can also reflect inflammatory stress.

Since Dec 2019, SARS-CoV-2 has infected about fifty thousand patients and caused over two thousand deaths in Wuhan, China. With the strengthened support from central and local governments and national medical staffs, SARS-CoV-2 epidemic has been preliminary controlled in Wuhan, China at current. Many other countries such as Italy, Iran, and Korea, however, are showing an ongoing outbreak,[4] which will inevitably bring about shortage of medical resources. To learn characteristics of patients with COVID-19 in Wuhan, China, during ongoing outbreak of SARS-CoV-2 epidemic, is helpful for others to make appropriate strategies and thus to reduce mortality of COVID-19 globally.

In conclusion, findings of this study suggested multiple homeostasis disturbances in patients with severe or critical illness on admission, including lymphopenia, anemia, hypoproteinemia, abnormal serum sodium, and elevated inflammatory stress. Acute organ injuries were common in non-survivors, which might cause by SARS-CoV-2 attacks and excessive inflammatory response. Early supportive therapy should been provided to control disease progression and reduce the mortality of COVID-19.

Acknowledgments

Declaration of interests

All authors declare that they have no competing interests.

Author Contributions

XL, LY and BZ had the idea for and designed the study and had full access to all data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. WZ, HX, BW and WY contributed to writing of the report. XL contributed to critical revision of the report. BW and TG contributed to the statistical analysis. All authors contributed to data acquisition, analysis, or interpretation, and reviewed and approved the final version.

Data sharing

The data sets that support findings of the current study are available from the corresponding author on reasonable request.

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Figure legends

Figure 1. study flow char

Table 1 Demographic and epidemicological characteristics of COVID-19 patients

	All patients (n=403)	Died (n=100)	Recovered (n=303)	P
Age, years	56 (39-68)	71 (65-80)	49 (37-62)	0.000
< 60	232 (57.6%)	20 (20%)	212 (70.0%)	0.000
≥ 60	171 (42.4%)	80 (80%)	91 (30.0%)	
Gender				0.035
Male	193 (47.9%)	57 (57%)	136 (44.9%)	
Female	210 (52.1%)	43 (43%)	167 (55.1%)	
Smoking	29 (7.2%)	9 (9%)	20 (6.6%)	0.421
Drinking	41 (10.2%)	10 (10%)	31 (10.2%)	0.947
With chronic diseases	175 (43.4%)	77 (77%)	98 (32.3%)	0.000
Hypertension	113 (28.0%)	60 (60%)	53 (17.5%)	0.000
Cerebrovascular diseases	39 (9.7%)	22 (22%)	17 (5.6%)	0.000
Coronary heart disease	36 (8.9%)	16 (16%)	20 (6.6%)	0.004
Diabetes	57 (14.1%)	25 (25%)	32 (10.6%)	0.000
Chronic pulmonary disease	28 (6.9%)	17 (17%)	11 (3.6%)	0.000
Cirrhosis	25 (6.2%)	9 (9%)	16 (5.3%)	0.181
Chronic kidney disease	7 (1.7%)	3 (3%)	4 (1.3%)	0.501
Anemia	15 (3.7%)	7 (7%)	8 (2.6%)	0.091
Main symptom of illness onset				0.122
Fever	319 (79.2%)	87 (87%)	232 (76.6%)	
Cough	41 (10.2%)	4 (4%)	37 (12.2%)	
Fatigue	14 (3.5%)	2 (2%)	12 (4.0%)	
Sore throat	8 (2.0%)	0 (0)	8 (2.6%)	
Shortness of breath	8 (2.0%)	2 (2%)	6 (2.0%)	
Dyspnea	5 (1.2%)	4 (4%)	1 (0.3%)	
Diarrhea	4 (1.0%)	1 (1%)	3 (1.0%)	
Vomit	2 (0.5%)	0 (0)	2 (0.7%)	
Myalgia	2 (0.5%)	0 (0)	2 (0.7%)	
Days from onset to admission	9 (7-12)	9 (7-13)	9 (6-11)	0.188
Abnormal imaging				0.433
Bilateral lungs	376 (93.3%)	95 (95%)	281 (92.7%))	
Single lung	27 (6.7%)	5 (5%)	22 (7.3%)	

Age and days from onset to admission were presented as median (interquartile range) and compared with

Mann-Whitney test between patients who died or recovered. Chi-squared test was conducted to compare other

categorical variables between two groups.

Table 2. Laboratory findings of COVID-19 patients on admission according to clinical outcome

		All patients (n=403)	Died (n=100)	Recovered (n=303)	P
White blood cell count, $\times 10^9$/L	(3.5-9.5)	5.50 (4.11-7.30)	8.58 (5.13-12.34)	5.03 (3.98-6.50)	0.000
Neutrophil count, $\times 10^9$/L	(1.8-6.3)	3.57 (2.54-5.48)	7.09 (4.00-11.00)	3.10 (2.39-4.43)	0.000
Lymphocyte, $\times 10^9$/L	(1.1-3.2)	1.06 (0.75-1.49)	0.73 (0.46-0.92)	1.18 (0.90-1.62)	0.000
< 1.1		212/403 (52.6%)	82/100 (82.0%)	130/303 (42.9%)	0.000
Red blood cell count, $\times 10^{12}$/L	(4.3-5.8)	4.14 (0.65)	3.96 (0.71)	4.20 (0.62)	0.002
Hemoglobin, g/L	(130-175)	126.9 (17.7)	125.0 (19.6)	127.5 (17.1)	0.225
< 130		220/403 (54.6%)	61/100 (61.0%)	159/303 (52.5%)	0.138
Platelet count, $\times 10^9$/L	(125-350)	192 (148-251)	169 (121-219)	205 (153-264)	0.000
< 125		58/403 (14.4%)	28/100 (28%)	30/303 (9.9%)	0.000
Albumin , g/L	(40-55)	37.4 (33.3-40.8)	33.2 (31.2-36.5)	38.5 (35.4-41.7)	0.000
< 40		270/387 (69.8%)	91/98 (92.9%)	179/289 (61.9%)	0.000
Total bilirubin, mg/dL	(0-23)	12.8 (10.0-17.5)	17.4 (12.7-24.7)	11.9 (9.5-15.4)	0.000
> 23		42 /387 (10.9%)	27/98 (27.6%)	15/289 (5.2%)	0.000
Creatinine, μmol/L	(57-97)	69.0 (60.0-86.0)	82.0 (62.8-104.8)	68.0 (59.3-82.0)	0.000
> 97		48/390 (12.3%)	28/98 (28.6%)	20/292 (6.8%)	0.000
Hypersensitive troponin I, ng/mL	(0-0.04)	0.006 (0.006-0.028)	0.035 (0.015-0.181)	0.006 (0.006-0.008)	0.000
> 0.04		65/304 (21.4%)	47/96 (49.0%)	18/208 (8.7%)	0.000
D-dimer, mg/L	(0-0.55)	0.73 (0.36-3.65)	5.38 (1.21-17.78)	0.50 (0.27-1.07)	0.000
≤ 0.55		133/327 (40.7%)	4/96 (4.2%)	129/231 (55.8%)	0.000
< 0.55 to ≤ 5		125/327 (38.2%)	44/96 (45.8%)	81/231 (35.1%)	
> 5		69/327 (21.1%)	48/96 (50%)	21/231 (9.1%)	
C-reactive protein, mg/L	(0-10)	25.5 (5.0-73.0)	95.0 (58.9-178.7)	23.8 (5.0-49.9)	0.000
≤ 10		123/326 (37.7%)	3/85 (3.5%)	120/241 (49.8%)	0.000
10 < to ≤ 100		148/326 (45.4%)	43/85 (50.6%)	105/241 (43.6%)	
≥ 100		55/326 (16.9%)	39/85 (45.9%)	16/241 (6.6%)	
Procalcitonin, ng/mL		0.057 (0.032-0.132)	0.199 (0.116-0.949)	0.044 (0.026-0.070)	0.000
≤ 0.1		241/355 (67.9%)	20/96 (20.8%)	221/259 (85.3%)	0.000
0.1< to ≤ 0.5		78/355 (22.0%)	45/96 (46.9%)	33/259 (12.7%)	
> 0.5		36/355 (10.1%)	31/96 (32.3%)	5/259 (1.9%)	
Sodium, mmol/L	(137-147)	142.9 (138.8-144.2)	141.1 (136.6-144.2)	143.2 (139.6-144.3)	0.001
< 137		57/403 (14.1%)	34/100 (34.0%)	23/303 (7.6%)	0.000
137 ≤ to ≤ 147		315/403 (78.2%)	52/100 (52.0%)	263/303 (86.8%)	
> 147		31/403 (7.7%)	14/100 (14.0%)	17/303 (5.6%)	

Red blood cell count and hemoglobin were presented as mean (SD) and compared with independent t test between the died and the recovered. Other variables were presented as median (interquartile range) and compared with Mann-Whitney test.

Table 3. Clinical characteristics of COVID-19 patients according to disease severity on admission

	Ordinary (n=198)	Severe or critical (n=205)	p
Age, years	43 (33-56)	66 (56-74)	0.000
Male gender	77 (38.9%)	116 (56.6%)	0.000
With chronic diseases	56 (28.3%)	119 (58.0%)	0.000
Days from onset to admission	8 (5-11)	10 (7-13)	0.002
White blood cell count, $\times 10^9$/L	4.91 (3.97-6.19)	6.46 (4.47-8.93)	0.000
Neutrophil count, $\times 10^9$/L	2.94 (2.40-4.11)	4.62 (2.97-7.97)	0.000
Lymphocyte count, $\times 10^9$/L	1.22 (0.94-1.70)	0.90 (0.54-1.24)	0.000
< 1.1	74/198 (37.4%)	138/205 (67.3%)	0.000
Red blood cell count, $\times 10^{12}$/L	4.25 (0.59)	4.03 (0.68)	0.000
Hemoglobin, g/L	128.8 (16.8)	125.0 (18.4)	0.031
< 130	98/198 (49.5%)	122/205 (59.5%)	0.043
Platelet count, $\times 10^9$/L	205 (154-261)	179 (136-245)	0.000
< 125	16/198 (8.1%)	42/205 (20.5%)	0.000
Albumin, g/L	39.9 (37.3-42.2)	34.7 (32.3-37.9)	0.000
< 40	94/184 (51.1%)	176/203 (86.7%)	0.000
Total bilirubin, mg/dL	11.6 (9.2-14.2)	14.8 (10.9-20.8)	0.000
> 23	8/184 (4.3%)	34/203 (16.7%)	0.000
Creatinine, μmol/L	68.0 (59.0-82.0)	71.0 (61.0-88.0)	0.028
> 97	13/187 (7.0%)	35/203 (17.2%)	0.002
Hypersensitive troponin I, ng/mL	0.006 (0.006-0.006)	0.012 (0.006-0.062)	0.000
> 0.04	13/127 (10.2%)	52/177 (29.4%)	0.000
D-dimer, mg/L	0.39 (0.23-0.83)	1.22 (0.52-7.46)	
≤ 0.55	80/135 (59.3%)	53/192 (27.6%)	0.000
0.55 < to ≤ 5	42/135 (31.1%)	83/192 (43.2%)	0.000
> 5	13/135 (9.6%)	56/192 (29.2%)	
C-reactive protein, mg/L	6.8 (5.0-25.7)	59.7 (20.7-103.5)	
≤ 10	88 (44.4%)	33 (16.1%)	0.000
10 < to ≤ 100	103 (52.0%))	125 (61.0%)	0.000
≥ 100	7 (3.5%)	47 (22.9%)	
Procalcitonin, ng/mL	0.04 (0.02-0.06)	0.10 (0.05-0.26)	
≤ 0.1	142/161 (88.2%)	99/194 (51.0%)	0.000
0.1 < to ≤ 0.5	18/161 (11.2%)	60/194 (30.9%)	0.000
> 0.5	1/161 (0.6%)	35/194 (18.0%)	
Sodium, mmol/L	143.5 (142.2-145.0)	141.2 (137.9-143.9)	
< 137	10/198 (5.1%)	47/205 (22.9%)	0.000
≤ 137 to ≤147	176/198 (88.9%)	139/205 (67.8%)	0.000
> 147	12/198 (6.1%)	19/205 (9.3%)	

Red blood cell count and hemoglobin were presented as mean (SD) and compared with independent t test. Other continuous variables were presented as median (interquartile range) and compared with Mann-Whitney test. Chi-squared test was conducted to compare categorical variables.

Table 4. Complications and treatments of COVID-19 patients

	All patients (n=403)	Died (n=100)	Recovered (n=303)	P
Complications				
Acute respiratory distress syndrome	143 (35.5%)	90 (90.0%)	53 (17.5%)	0.000
Acute kidney injury	57 (14.1%)	43 (43.0%)	14 (4.6%)	0.000
Acute cardiac injury	83 (20.6%)	65 (65.0%)	18 (5.9%)	0.000
Acute liver injury	87 (21.6%)	71 (71.0%)	16 (5.3%)	0.000
Secondary infection	77 (19.2%)	58 (58.0%)	19 (6.3%)	0.000
Treatment measures				
High-flow nasal cannula oxygen therapy	106 (26.3%)	74 (74.0%)	32 (10.6%)	0.000
Non-invasive mechanical ventilation	56 (13.9%)	48 (48.0%)	8 (2.6%)	0.000
Invasive mechanical ventilation	23 (5.7%))	16 (16.0%)	7 (2.3%))	0.000
Blood purification	39 (9.7%)	16 (16.0%)	23 (7.6%)	0.014
Antiviral agents	394 (97.8)	96 (96.0%)	298 (98.3%)	0.323
Abidol	324 (80.4%)	66 (66.0%)	258 (85.1%)	0.000
Oseltamivir	123 (30.5%)	32 (21.0%)	91 (30.0%)	0.711
Ribavirin	81 (20.1%)	26 (26.0%)	55 (18.2%)	0.089
Ganciclovir	53 (13.2%)	17 (17.0%)	36 (11.9%)	0.189
Recombinant interferon	72 (17.9%)	8 (8.0%)	64 (21.1%)	0.003
Antibiotics	349 (86.6%)	99 (99.0%)	250 (82.9%)	0.000
Anti-fungal agents	14 (3.5%)	11 (11.0%)	3 (1.0%)	0.000
Glucocorticoids	166 (41.2%)	74 (74.0%)	92 (30.4%)	0.000
Immunoglobulin	198 (49.1%)	66 (66.0%)	132 (43.6%)	0.000

Chi-squared test was conducted to compare categorical variables presented as number (%).

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┌─────────────────────────────────┐
│  985 patients with confirmed COVID-19  │
│         by Feb 25, 2020         │
└─────────────────────────────────┘
                │            ┌──────────────────────────┐
                │            │ 574 excluded             │
                │            │ still in hospitalization │
                │            └──────────────────────────┘
                ▼
┌─────────────────────────────────┐
│     411 with clinical outcome    │
└─────────────────────────────────┘
                │            ┌──────────────────────────┐
                │            │ 8 excluded               │
                │            │ died on admission day    │
                │            └──────────────────────────┘
                ▼
┌─────────────────────────────────┐
│     403 enrolled in the study    │
│   100 died and 303 recovered     │
└─────────────────────────────────┘
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