Clinical Features of 85 Fatal Cases of COVID-19 from Wuhan: A Retrospective Observational Study

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Abstract

Background: The global death toll from COVID-19 virus as of Mar 25, 2020 exceeds 21000. The risk factors for death were attributed to advanced age and co-morbidities but have not been accurately defined.

Objectives: To report the clinical features of 85 fatal cases with COVID-19 in two hospitals in Wuhan.

Methods: Medical records of 85 fatal cases of COVID-19 between January 9 and February 15, 2020 were collected. Information recorded included medical history, exposure history, comorbidities, symptoms, signs, laboratory findings, CT scans and clinical management.

Measurements and Main Results: The median age of the patients was 65.8 years and 72.9% were male. Common symptoms were fever (78 [91.8%]), shortness of breath (50 [58.8%]), fatigue (50 [58.8%]), and dyspnea (60 [70.6%]). Hypertension, diabetes and coronary heart disease were the most common comorbidities. Notably, 81.2% patients had very low eosinophil counts on admission. Complications included respiratory failure (80 [94.1%]), shock (69 [81.2%]), ARDS (63 [74.1%]), arrhythmia (51 [60%]), etc. Most patients received antibiotic (77 [90.6%]), antiviral (78 [91.8%]) and glucocorticoids (65 [76.5%]) treatments. Thirty-eight patients [44.7%] and 33 [38.8%] received intravenous immunoglobulin and interferon α2b respectively.

Conclusions: In this depictive study of 85 fatal cases of COVID-19, most cases were males aged over 50 years old with noncommunicable chronic diseases. The majority of the patients died of multiple organ failure. Early onset of shortness of breath may be used as an observational symptom for COVID-19 exacerbations. Eosinophilopenia may indicate a poor prognosis. The combination of anti-microbial

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drugs did not offer considerable benefit to the outcome of this group of patients.

Keywords: COVID-19; SARS-CoV-2; fatal cases; eosinophilopenia, co-pathogen; survival

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Introduction

A new type coronavirus was discovered to be the cause of unexplained pneumonia cases in Wuhan, China in late December, 2019 (1, 2). The virus belongs to the same genus as SARS-CoV and MERS-CoV and was thus named SARS-CoV-2 by the International Committee on Taxonomy of Viruses (ICTV) on February 11, 2020. However, SARS-CoV-2 is more infectious than SARS-CoV and MERS-CoV with over 80,000 cases in China and nearly 500,000 cases worldwide reported as of March 25, 2020, according to the Center for Systems Science and Engineering (CSSE) at John Hopkins University (JHU) (3). On February 11, 2020, WHO officially named the disease caused by the new coronavirus as COVID-19. SARS-CoV-2 is prone to transmit in family clusters (4, 5) or cause outbreaks in hospitals. Most COVID-19 patients present with mild and moderate symptoms, but severe cases can present with acute respiratory distress syndrome (ARDS), multiple organ dysfunction syndrome (MODS) and even death. It has been reported that fatality rate of COVID-19 varies from 1.4% (6) to 4.3% (7) in different regions or hospitals. Mounting evidence has shown that this virus induces excessive and aberrant non-effective host immune responses associated with severe lung injury (1, 8). Higher rates of ARDS are seen in elderly patients with comorbidities (4). Previously published research has described clinical characteristics of COVID-19 in Wuhan and other provinces, as well as SARS and MERS. Our current work is the largest cohort of fatal cases of COVID-19 in the literature thus far.

As in SARS and MERS patients, most COVID-19 patients have a characteristic ground glass appearance on chest CT scans (9). Pathological characteristics of a COVID-19 patient with biopsy samples from the lung, liver and heart show similar features as those seen in SARS-CoV and MERS-CoV infections (10). Along with ARDS, acute cardiac injury and acute kidney injury can also occur (1).

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Since COVID-19 is a new epidemic of SARS, the specific mechanisms and pathophysiology of the disease remain elusive. No effective vaccine or anti-viral treatment are currently available. As of March 25, 2020, there has been of total of 21,192 reported COVID-19-related deaths worldwide but no detailed analysis of this group has been reported. Here, we report on the characteristics of the largest series of fatal cases of COVID-19 in Wuhan city, the epicenter of the SARS-CoV-2 outbreak, and describe their clinical characteristics in the hope that this will help clinicians identify patients with poor prognosis at an early stage.

Methods

Study design and participants

This is a retrospective study conducted in two hospitals in Wuhan: Hannan Hospital and Wuhan Union Hospital. The study was approved by the Ethics Committees of both hospitals. All consecutive severe patients with confirmed COVID-19 admitted to the two hospitals between January 9 and February 15, 2020 were enrolled. All patients were diagnosed based on the recommendations by the National Institute for Viral Disease Control and Prevention, China (5th edition) which specifies that suspected cases who from Hubei Province, with Wuhan city as its capital, who have a contact history and present typical chest CT features may be diagnosed with COVID-19. A positive PCR test for SARS-CoV-2 is mandatory for patients located outside of Hubei province in order to make the diagnosis. Clinical outcomes (mortality) were monitored up to February 15, 2020, the final date of follow-up.

Data collection

Epidemiological, clinical, laboratory, radiological test results, as well as clinical management data,

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were obtained using data collection forms from electronic medical records. The data were reviewed by Wuhan-Beijing Medical Treatment Group for COVID-19 of physicians and scientists. Data collected included demographics, medical history, exposure history, underlying comorbidities, symptoms, signs, laboratory findings, chest CT scans, and clinical management (i.e. antiviral therapy, corticosteroid therapy, respiratory support, intravenous immunoglobulin, continuous renal replacement therapy). The date of disease onset was defined as the day symptoms (i.e. fever, shortness of breath, fatigue, dyspnea, anorexia, expectoration, dry cough, diarrhea, myalgia, headache, vomiting, abdominal pain, chest pain and pharyngalgia) first appeared. ARDS was defined according to the Berlin definition. Acute kidney injury and cardiac injury were defined according to the previous study (7). The duration from onset of disease to hospital admission, and death were also recorded.

We searched PubMed, Medline, and Google Scholar on March 25, 2020, for articles describing the clinical features of patients infected with SARS-CoV-2; previously known as 2019 novel coronavirus [2019-nCoV]), using the search terms "novel coronavirus" or "2019-nCoV" and "fatal cases" or "COVID-19", with no time restrictions. We also searched CNKI and Wanfang Data using the same terms in Chinese, with no time restrictions.

Statistical analysis

Continuous measurements such as mean (SD) and categorical variables were reported as numbers and percentages (%). For laboratory results, we also assessed whether or not measurements fell within the normal range. We used SPSS (version 26.0) for all analysis.

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Results

Presenting characteristics

Eight-five fatal cases with clinically diagnosed COVID-19 were included in this study. Thirty-three (38.8%) patients had positive SARS-CoV-2 PCR tests. The median age was 65.8 years (SD: 14.2; range: 14-86 years), and 62 (72.9%) were male (Table 1). Three patients had a history of exposure to the Huanan seafood market.

Of the 85 patients, 58 (68.2%) had one or more comorbidities. Hypertension (32 [37.6%]), diabetes (19 [22.4%]) and coronary heart disease (10 [11.8%]) were the most common comorbidities. On admission, most patients had fever (78 [91.8%]) and dyspnea (60[70.6%]), two thirds of the patients had shortness of breath (50 [58.8%]) and fatigue (50 [58.8%]). Almost half of the patients had anorexia and more than a third of the patients had expectoration (32 [37.6%]). Other symptoms included dry cough, diarrhea, myalgia, headache, vomiting, abdominal pain, chest pain and pharyngalgia. The duration from first symptoms to hospital admission and ARDS were 10.1 ± 6.2 days and 10.3 ± 6.6 days respectively (Table 1). The mean duration from hospital admission to death was 6.35 ± 4.51 (range 1 to 21) days.

The most common cause of death in 81 of the 85 patients was respiratory failure (38, 46.91%), followed by septic shock (16, 19.75%), multiple organ failure (13, 16.05%) and cardiac arrest (7, 8.64%). Acute coronary syndrome, malignant arrhythmia and DIC were rare causes of death (Table 1).

Laboratory findings

On admission, 69 (81.2%) patients had an eosinophil count below the normal range (0.02-0.52 x 10^9

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cells/L), and 10 (11.8%) and 38 (44.7%) patients had a white blood cell count below and above the normal range respectively. Fifty-one (60.0%) and 66 (77.6%) of the patients had neutrophils above and lymphocytes below the normal range respectively. Platelet counts below and above the normal range were noted in 35 (41.2%) and 6 (7.1%) patients respectively. Many patients had deceased hemoglobin and hematocrit (Table 2). D-dimer was higher than the normal range in 56 (65.9%) patients. Many patients showed decreased APTT (activated partial prothrombin time) and increased PT (Prothrombin time). Sixty-seven (78.8%) patients had albumin below the normal range. Many patients had varying degrees of abnormal liver function with increased alanine aminotransferase (ALT) or aspartate aminotransferase (AST). Most patients had abnormal myocardial zymograms characterized by increased creatine kinase in 31 (36.5%) and increased lactate dehydrogenase in 70 (82.4%) patients. Forty-eight (56.5%) patients had different degrees of impaired renal function with elevated blood urea nitrogen or serum creatinine. Seventy-eight (91.8%) patients had C-reactive protein and 19 (22.4%) had procalcitonin levels above the normal ranges.

On admission, co-pathogens were tested in a subset of patients. Most testing was serological analysis of blood samples. Mycoplasma IgM antibodies were detected in 9 of 34 (26.5%) patients that were tested, and Chlamydia was positive in 12 out of 35 (34.1%) patients tested. Two patients out of 22 patients (9.1%) tested were Influenza A positive and one out of 19 patients (5.3%) tested for Influenza B was positive. Three of nine (33.3%) patients tested for respiratory syncytial virus were positive. There were no patients positive for parainfluenza virus, adenovirus, coxsackievirus, tuberculosis, rickettsia, or legionella. With regard to sputum cultures, no bacterial cultures were positive in 12 patients tested, but 3 patients had positive fungal cultures.

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Chest CT findings

Chest CT scan was done in 80 patients on admission. Seventy-eight (97.5%) patients showed bilateral pneumonia and only two patients had unilateral pneumonia. Sixty-one (76.3%) patients showed multiple mottling and ground-glass opacities (Figure 1 and Table 4).

Complications and treatment

Patients presented with functional damage involving multiple vital organs, including respiratory failure (80 [94.1%]), shock (69 [81.2%]), ARDS (63 [74.1%]) arrhythmia (51 [60.0%]), acute myocardial injury (38 [44.7%]), acute liver injury (30 [35.3%]) and sepsis (28 [32.9%]) (Table 5).

Most patients received antibiotics (77 [90.6%]), antiviral treatment (78 [91.8%]) and glucocorticoids (65 [76.5%]). Thirty-eight (44.7%) and 33 [38.8%]) patients received intravenous infusions of immunoglobulin and recombinant human interferon α2b. Eleven patients received antifungal treatment. Two thirds of the patients had oxygen therapy. Forty-four and 18 patients received non-invasive and invasive mechanical ventilation respectively. Continuous renal replacement therapy was given to eight patients. One patient received plasma from a recovered COVID-19 patient and no patients received extracorporeal membrane oxygenation as rescue therapy (Table 5).

On admission, the median CURB-65 was 1.9 (SD: 1.1; range: 0-5). Eight (9.4%) patients had a CURB-65 score of 0, 27 (31.8%) patients had a score of 1 and 25 (29.4%) patients had a score of 2. These were classified as mild according to the CURB-65 guidelines. Only 25 patients were classified as severe on admission, of whom 20 (23.5%) patients had a score of 3, 3 (3.5%) had a score of 4, and 2 (2.4%) had a score of 5 (Table 6). Procalcitonin appears to increase with higher CURB-65 admission

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scores (Table 6). There was a 3.35 (95% CI 0.17, 6.53) increase in procalcitonin level for each increase in CURB-65 level (p = 0.0451).

Discussion

In this retrospective study, we report to this date the largest series of patients who have died from COVID-19, providing detailed clinical characteristics of this cohort of patients from two Wuhan hospitals. The 85 fatal cases of COVID-19 reported here account for 2.7% of the total mortality due to SARS-CoV-2 infection in Hubei province. Although the symptoms of the majority of patients in other provinces have been comparatively mild (11), the number of deaths from SARS-CoV-2 infection continues to increase, with more cases and fatalities occurring now in other provinces, countries and regions. As of March 25, 2020, there have been 3287 fatalities and 81,869 confirmed cases of COVID-19 in China, according to the WHO. Early diagnosis and timely treatment to reduce mortality is of crucial importance. It is hoped that this work will have value in helping clinicians identify patients with poor prognosis at an early stage by being aware of some of the alarming clinical characteristics presented by patients before they died from COVID-19, and help guide appropriate and effective management for future patients.

A recent study by Zhou *et al.* of 191 patients in which there were 54 deaths found that older age, high SOFA score and D-dimer greater than 1 μg/mL could assist in the early identification of patients who may have a poorer prognosis. In our study, the median age of non-survivors was 65.8 years, which is similar to the median age reported in non-survivors but higher than that of the survivors (52 years) reported in the previous study. Furthermore, our study found that the median level of D-dimer in non-survivors was 5.159 ug/ml (SD: 4.679, range: 0.27-26) and 70.6% or our patients had a D-dimer greater

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than 1 μg/mL, which was also consistent with the previous study. The SOFA score was not included in our study. However, we found that 29.4% patients had a CURB-65 score greater than 3, which was similar to the previous study (28% in non-survivors) (12). In our study, about a quarter of patients who died had an elevated procalcitonin level, which was consistent with the previous study, but Zhou's study also found that an elevated procalcitonin level greater than 0.5 was associated with a 93% chance of death. In a non-COVID-19 study on patients with pneumonia, the CURB-65 score on admission correlated with mortality risk (13).

It was difficult to find age- and sex-matched controls for our study. In comparing our data to the previous study mentioned above which did include survivors, we found that the hospital length of stays was the same in non-survivors between the two studies but almost double the survivors length of stay (12 days) in the previous study. Interestingly, the use of intravenous immunoglobulin was higher in non-survivors in both the previous study and our study (36 and 38%, respectively), but much lower in survivors (10%) in the previous study. The increased use of intravenous immunoglobulin may be due to the severity of illness, but also raises the question of whether intravenous immunoglobulin may be ineffective in severely ill patients. The use of corticosteroids was higher in our cohort of 85 patients than in the abovementioned study (65% vs 48% in non-survivors and 30% in survivors respectively). Lymphopenia was identified as a risk factor for death in the previous study, but eosinopenia was not mentioned.

Previous studies found that nearly half of COVID-19 patients are over the age of 50 and men are more likely to be infected than women (14). The mortality rate in males is higher than that in females. In patients who develop SARS, advanced age is an independent predictor for an adverse outcome, but

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gender is not (15). In this report, we observed that among the 85 deaths, there were 76 (89.3%) patients over the age of 50 years old and 62 (72.9%) were male.

The most common comorbidities of the COVID-19 patients in our cohort are hypertension and diabetes, which is similar to that of previous studies (4, 7). However, the most common comorbidities of the SARS patients were diabetes (16 [11%]) and cardiac disease (12 [8%]) (16). The increased prevalence of hypertension in China may play a role in COVID-19 related deaths.

Common clinical features of COVID-19 patients include fever (83%), cough (82%), shortness of breath (31%) and muscle ache (11%) (4). For SARS, the common clinical features included fever (99%), cough (69%), myalgia (49%), and dyspnea (42%)(16). It is worth noting that the overall rates of shortness of breath in our cohort were higher than that in SARS patients (4). We suggest that early onset of shortness of breath may be indicative of poor prognosis.

We found that the absolute eosinophil count in peripheral blood was reduced in almost all patients who died. The number of patients with reduced eosinophil count in non-severe and severe COVID-19 patients who survived has been reported elsewhere to be 39/82 (47.6%) and 34.56 (60.7%) respectively (17). Previous studies have reported that there is rapid and persistent decrease in the numbers of circulating eosinophils in acute infection or inflammation (18, 19). A study on 30-day mortality and eosinopenia showed that eosinopenia is an independent predictor of death in patients with pneumonia but no chronic respiratory disease (20). This effect was not related to steroid use. In the case of COVID-19, this may be related to CD8 T cell depletion and eosinophil consumption caused by SARS-CoV-2. IL-5, produced by CD8 T cells, contributes to eosinophil proliferation and activation in blood (21-23). Lower numbers of CD8 T cells has been found in SARS-CoV-2 infected patients (24).

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Moreover, ECP and EDN, two eosinophil granule proteins, can neutralize viruses (19, 25, 26). Therefore, the decrease of eosinophil in COVID-19 patients may be related to a higher viral load of SARS-CoV-2 and SARS-CoV-2 triggered eosinophil granule proteins consumption.

We thus speculate that eosinophilopenia may be used as a prognostic indicator for COVID-19 patients. In addition, the ratio of neutrophil to eosinophil counts may be another measure which can minimize variability in absolute eosinophil counts from different hospitals. Another laboratory abnormality found in this study was decreased total lymphocytes, which is consistent with the conclusions of existing research indicating that lymphocytopenia is more often seen in non-survivors of SARS-CoV-2 infection (7).

Similarly, prolonged prothrombin time, and elevated lactate dehydrogenase were noted in our cohort, whereas a previous study found that 13% of patients had creatine kinase and 3% patients had serum creatinine above the normal ranges at the time of admission (4). Wang, *et al.* reported that the levels of blood urea and creatinine progressively increased before death through dynamic profiling of laboratory data (7). We observed that 56.5% patients of our patients had renal dysfunction as indicated by the increased levels of blood urea or creatinine at the time of admission. Therefore, we suggest that an increased level of creatinine and urea nitrogen may also indicate poor prognosis.

Co-pathogens of COVID-19 patients has not been previously reported in the literature. Testing for co-pathogens was done in some but not all patients in our cohort, and we found that less than 10% of tested patients were positive for influenza A virus, influenza B virus, and parainfluenza virus. It is worth noting that the antibody-positive rate of mycoplasma and chlamydia were relatively high.

The initial admission CURB-65 of most patients was not high, and yet the outcome of all the

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patients was death. This indicates that the clinical course of COVID-19 develops rapidly, so the CURB-65 at the beginning of admission cannot be used as a guide of severity. COVID-19 patients need to be closely monitored after admission. A Kaplan-Meier curve is illustrated in Figure 2.

From a practical standpoint, doctors equipped with protective suits and helmets have great difficulties in closely examining patients with standard techniques such as auscultation and observing for signs of shortness of breath. Therefore, laboratory findings and chest CT scan become critical in monitoring disease progress and treatment outcome. We determined that the presence of bilateral pneumonia and progressive radiographic deterioration on follow-up CT may be risk factors for poor prognosis (25). It should be noted that the administration of multiple antibiotics did not change the outcome of the disease in our series. Rational use of antibiotics should thus be exercised. It is also not known if any of the therapies used in COVID-19, such as steroids may actually be counterproductive, and lead to increased morbidity or mortality.

This study has some limitations. First, only fatal cases of COVID-19 were included. A prospective study including fatal and non-fatal patients will provide more conclusive and valuable data. Second, pathological findings were not available. Third, although eosinophilopenia was found in almost all patients in this series, it can also occur in many non-fatal severe and moderate patients based on our clinical observations (unpublished data). Therefore, additional studies are needed to confirm the prognostic value of eosinophilopenia in COVID-19 patients.

Conclusion

In summary, most cases of death from COVID-19 were males over 50 years of age with noncommunicable chronic diseases such as hypertension, diabetes, coronary heart diseases. The

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patients mainly died of multiple organ failure. Early onset of shortness of breath might be predictive of demise. Eosinophilopenia may indicate a poor prognosis. The use of a combination of more than three anti-microbial drugs appears to offer no benefit to the outcome of this group of patients.

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Figure legends

Figure 1

(A) Chest CT Images of a 55-year-old male patient with COVID-19 taken on January 27, 2020, showing unilateral pneumonia.

(B) Chest CT Images of an 85-year-old male patient with COVID-19 taken on February 4, 2020, showing ground glass opacity in both lungs.

(C) Chest CT Images of a 23-year-old female patient with COVID-19 taken on January 24, 2020, showing diffusive ground glass opacity.

(D) Chest CT Images of a 72-year-old male patient with COVID-19 taken on January 30, 2020, showing bilateral pneumonia.

Figure 2

A Kaplan-Meier survival curve from the time of admission with COVID-19 to time of death

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Table1 – Clinical characteristics of patients with COVID-19

Clinical characteristics, symptoms or signs	n=85, n (%), mean +/- SD
Age, year	65.8±14.2
Age groups	
0-14	1 (1.2%)
15-49	8 (9.4%)
50-64	24 (28.2%)
≥ 65	52 (61.2%)
Sex	
Female	23 (27.1%)
Male	62 (72.9%)
Exposure to Huanan seafood market	3 (3.53%)
Comorbidities	
Any	58 (68.2%)
Hypertension	32 (37.6%)
Diabetes	19 (22.4%)
Coronary heart disease	10 (11.8%)
Cerebrovascular diseases	7 (8.2%)
Chronic liver disease	5 (5.9%)
Malignancy	6 (7.1%)
Chronic kidney disease	3 (3.5%)
Chronic obstructive pulmonary disease	2 (2.4%)
Signs and symptoms on admission	
Fever	78 (91.8%)
Short of breath	50 (58.8%)
Fatigue	50 (58.8%)
Dyspnea	60 (70.6%)
Anorexia	48 (56.5%)
Expectoration	32 (37.6%)
Dry cough	19 (22.4%)
Diarrhea	16 (18.8%)
Myalgia	14 (16.5%)
Headache	4 (4.7%)
Vomiting	4 (4.7%)
Abdominal pain	3 (3.5%)
Chest pain	2 (2.4%)
Pharyngalgia	2 (2.4%)
Onset of symptom to (days)	
Hospital admission	10.1 ± 6.2
Acute respiratory distress syndrome	10.3 ± 6.6
Onset of hospital admission to death (days)	6.35 ± 4.51
Cause of death	

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Respiratory failure	38/81(46.91%)
Multiple organ failure (MOF)	13/81(16.05%)
Septic shock	16/81(19.75%)
Cardiac arrest	7/81(8.64%)
Acute coronary syndrome (ACS)	4/81(4.94%)
Malignant arrhythmia	2/81(2.47%)
Disseminated intravascular coagulation (DIC)	1/81(1.23%)

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Table 2 – Laboratory findings of patients with COVID-19 on admission to hospital

	n=85, n (%), mean (SD)
White blood cell count; $\times10^9$/L; normal range 3.5-9.5	10.121 ± 6.266
Increased	38 (44.7%)
Decreased	10 (11.8%)
Neutrophil count; $\times10^9$/L; normal range 1.8-6.3	8.765 ± 6.181
Increased	51 (60.0%)
Decreased	11 (12.9%)
Lymphocytes; $\times10^9$/L; normal range 1.1-3.2	0.729 ± 0.419
Decreased	66 (77.6%)
Eosinophils; $\times10^9$/L; normal range 0.02-0.52	0.013 ± 0.025
Decreased	69 (81.2%)
Basophils; $\times10^9$/L; normal range <0.06	0.018 ± 0.035
Increased	4 (4.7%)
Monocytes; $\times10^9$/L; normal range 0.1-0.6	0.413 ± 0.305
Increased	16 (18.8%)
Decreased	7 (8.2%)
Platelets; $\times10^9$/L; normal range 125-350	162.6 ± 108.9
Increased	6 (7.1%)
Decreased	35 (41.2%)
Neutrophil-to-lymphocyte ratio	15.17±13.67
Eosinophil-to-lymphocyte ratio	0.027±0.056
Eosinophil-to-neutrophil ratio	0.003±0.014
Haemoglobin; g/L; normal range 130–175	129.1 ± 25.4
Decreased	41 (48.2%)
Hematocrit; %; normal range 40-50	38.05 ± 7.10
Increased	5 (5.9%)
Decreased	53 (62.4%)
D-dimer (μg/L; normal range 0.0–1.5)	5.159 ± 4.679
Increased	56 (65.9%)
Activated partial prothrombin time; s; normal range 28.0-43.5	39.22 ± 9.26
Increased	22 (25.9%)
Decreased	4 (4.7%)
Prothrombin time; s; normal range 11.0-16.0	15.41 ± 3.32
Increased	22 (25.9%)
Decreased	1 (1.2%)
Fibrinogen; g/L; normal range 2.0-4.0	6.321 ± 18.349
Increased	40 (47.1%)
Decreased	19 (22.4%)
Albumin; g/L; normal range 35.0–55.0	30.95 ± 9.85
Decreased	67 (78.8%)
Alanine aminotransferase; U/L; normal range 21-72	72.9 ± 199.5
Increased	14 (16.5%)

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Aspartate aminotransferase; U/L; normal range 17-59	94.4 ± 263.3
Increased	28 (32.9%)
Total bilirubin; μmol/L; normal range 5.1-19.0	18.44 ± 13.61
Increased	30 (35.3%)
Blood urea nitrogen; mmol/L; normal range 3.2-7.1	9.368 ± 7.360
Increased	42 (49.4%)
Serum creatinine; μmol/L; normal range 58-110	113.73 ± 149.70
Increased	16 (18.8%)
Creatine kinase; U/L; normal range 55–170	298.0 ± 401.8
Increased	31 (36.5%)
Lactate dehydrogenase; U/L; normal range 109-245	645.8 ± 596.9
Increased	70 (82.4%)
Glucose; mmol/L; normal range 4.1-5.9	9.383 ± 5.099
Increased	67 (78.8%)
Decreased	3 (3.5%)
Procalcitonin; μg/L; normal range <0.5;	3.650 ± 13.398
Increased	19 (22.4%)
C-reactive protein; mg/L; normal range <8.0	107.259 ± 117.215
Increased	78 (91.8%)

Increased means over the upper limit of the normal range and decreased means below the lower limit of the normal range

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Table 3 – Co-pathogens of fatal patients with COVID-19

Co-pathogens	n (%)
Blood sample	
Mycoplasma	9/34 (26.5%)
Chlamydia	12/35 (34.1%)
Respiratory syncytial virus	1/3 (33.3%)
Adenovirus	0/3 (0%)
Coxsackievirus	0/2 (0%)
Influenza A virus	2/22(9.1%)
Influenza B virus	1/19 (5.3%)
Parainfluenza virus	0/18 (0%)
tuberculosis	0/9 (0%)
Rickettsia	0/1 (0%)
Legionella	0/1(0%)
sputum culture	
Bacterial culture	0/12 (0%)
Fungal culture	3/9 (33.3%)

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Table 4 – Chest CT findings of patients with COVID-19

CT finding	n=80, n (%)
Unilateral pneumonia	2 (2.5%)
Bilateral pneumonia	78 (97.5%)
Multiple mottling and ground-glass opacity	61 (76.3%)

80 patients available

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Table 5 – Complications and management of patients with COVID-19

Complications	n=85, n (%)
Respiratory failure	80 (94.1%)
Shock	69 (81.2%)
ARDS	63 (74.1%)
Arrhythmia	51 (60%)
Acute cardiac injury	38 (44.7%)
Acute liver injury	30 (35.3%)
sepsis	28 (32.9%)
Treatment	
Oxygen therapy	57 (67.1%)
Non-invasive mechanical ventilation	61 (71.8%)
Invasive mechanical ventilation	18 (21.2%)
Kidney replacement therapy	8 (9.4%)
ECMO	0
Antibiotic treatment	77 (90.6%)
Antifungal treatment	11 (12.9%)
Antiviral treatment	78 (91.8%)
Glucocorticoids	65 (76.5%)
Interferon	33(38.8%)
Intravenous immunoglobulin therapy	38 (44.7%)
COVID recovery patient plasma treatment	1(1.2%)
Anti-infection treatment	
Antibiotics	
Meropenem	38 (44.7%)
Imipenem/cilastatin	1 (1.2%)
Moxifloxacin	40 (47.1%)
Levofloxacin	4 (4.7%)
Linezolid	18 (21.2%)
Vancomycin	2(2.4%)
Teicoplanin	2 (2.4%)
Tigecycline	2 (2.4%)
Piperacillin/Tazobactam	9 (10.6%)
Ceftriaxone sodium	3 (3.5%)
Cefoperazone/sulbactam	2 (2.4%)
Ceftazidime tazobactam	2(2.4%)
Antiviral	
Arbidol hydrochloride capsules	51 (60%)
Lopinavir and Ritonavir tablets	11 (12.9%)
Oseltamivir	9 (10.6%)
Paramivir	6 (7.1%)
Ganciclovir	5 (5.9%)
Ribavirin	4 (4.7%)

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Antifungal	
Caspofungin	2 (2.4%)
Voriconazole	8 (9.4%)
Fluconazole	3 (3.5%)

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Table 6 – CURB-65 of fatal patients with COVID-19

CURB-65	N=85[#], n (%)	Procalcitonin level* (N=85)	Data from Zhou *et al.* (N=54) [#]
Mean CURB-65	1.9 ± 1.1		
	Grade groups		
0	8 (9.4%)	1.00 ± 1.61	16 (30%)
1	27 (31.8%)	0.87 ± 1.81	
2	25 (29.4%)	1.10 ± 1.91	23 (43%)
3	20 (23.5%)	4.19 ± 8.52	15 (28%)
4	3 (3.5%)	29.44 ± 50.22	
5	2 (2.4%)	1.58 ± n.a.	

[#]85 patients from our study compared to 54 patients from Zhou's study

*p = 0.656, calculated using the Kruskal-Wallis H test for comparing multiple groups.

n.a. = not applicable

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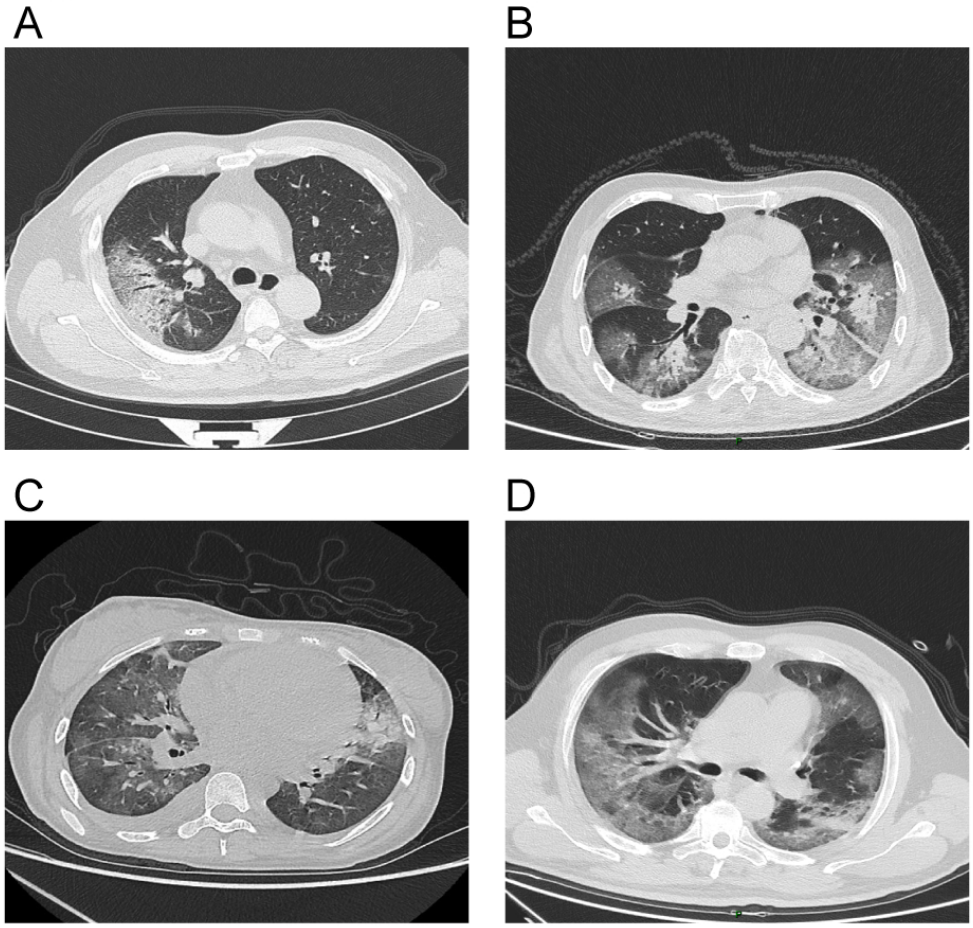
Figure 1



(A) Chest CT Images of a 55-year-old male patient with COVID-19 taken on January 27, 2020, showing unilateral pneumonia. (B) Chest CT Images of an 85-year-old male patient with COVID-19 taken on February 4, 2020, showing ground glass opacity in both lungs. (C) Chest CT Images of a 23-year-old female patient with COVID-19 taken on January 24, 2020, showing diffusive ground glass opacity. (D) Chest CT Images of a 72-year-old male patient with COVID-19 taken on January 30, 2020, showing bilateral pneumonia.

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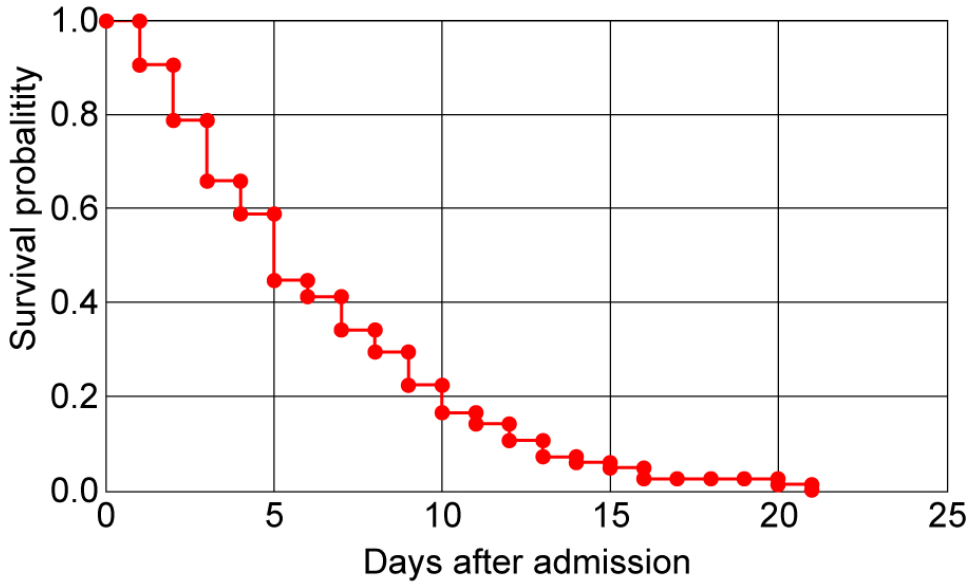
Figure 2



A Kaplan-Meier survival curve from the time of admission with COVID-19 to time of death.

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