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

Jianlei Cao[1#], MD, PhD; Wen-Jun Tu[2#‡], MD, PhD; Wenlin Cheng[1], MD, PhD; Lei Yu[3], MD, PhD; Ya-Kun Liu[4], MD; Xiaoyong Hu[1‡], MD, PhD; Qiang Liu[2‡], PhD

1. Department of Cardiology, Zhongnan Hospital, Wuhan University, Wuhan, China
2. Institute of Radiation Medicine, China Academy of Medical Science & Peking Union Medical College, Tianjin, China
3. Department of Infectious Diseases, The Fourth Affiliated Hospital of Harbin Medical University, Harbin, China
4. Qilu Hospital of Shandong University, Jinan, China

[#] Contributed equally to this work

[‡]Corresponding author:
Wen-Jun Tu; Email: tuwenjun@irm-cams.ac.cn; Tel/fax: 86-022-81682291
Address: No.238, Baiti Road, Tianjin, 300192, P.R. China

Summary

The mortality rate was high among the COVID-19 patients as patient characteristics seen more frequently in those who died were development of systemic complications following onset of the illness and the severity of disease requiring admission to the ICU.

© The Author(s) 2020. Published by Oxford University Press for the Infectious Diseases Society of America. All rights reserved. For permissions, e-mail: journals.permissions@oup.com.

Abstract

Objective

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

Methods

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

Results

The study cohort included 102 adult patients. The median (IQR) age was 54 years (37-67years) and 48.0% were female. A total of 34 patients (33.3%) were exposed to source of transmission in the hospital setting (as health care workers, patients, or visitors) and 10 patients (9.8%) had a familial cluster. Eighteen patients (17.6%) were admitted to the ICU, and 17 patients died (mortality, 16.7%; 95% confidence interval [CI], 9.4%-23.9%). Among patients who survived, they were younger, more likely were health care workers and less likely suffered from comorbidities. They were also less likely suffered from complications. There was no difference in drug treatment rates between the survival and non-survival groups. Patients who survived less likely required admission to the intensive care unit (14.1% vs. 35.3%). Chest imaging examination showed that death patients more likely had ground-glass opacity (41.2% vs. 12.9%).

Conclusions

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

KEYWORDS: COVID-19; SARS-CoV-2; human-to-human transmission; nosocomial infections; outcome

Introduction

In December 2019, a cluster of patients with pneumonia of undetermined etiology was recognized in Wuhan, Hubei, China [1]; subsequently, a novel coronavirus (SARC-CoV-2) was identified from lower respiratory tract samples obtained from affected patients [2]. The virus and its associated disease were given the designation COVID-19 in February 2020, distinguishing this syndrome from the acute respiratory syndromes associated with two other betacoronaviruses (severe acute respiratory syndrome coronavirus [SARS-CoV] and Middle East respiratory syndrome coronavirus [MERS-CoV] that caused earlier outbreaks of severe disease in humans [3-4]. Structural analysis suggests that SARS-CoV-2 might be able to bind to the angiotensin-converting enzyme (ACE) 2 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 older patients (>65 years) with comorbidities and ARDS were at 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, the clinical features and short-term outcomes of patients with COVID-19 is still limited. In this study, we investigate clinical and laboratory features and short-term outcomes of patients with COVID-19.

Methods

Patients and Data Collection

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

Epidemiological, clinical, radiological characteristics, underlying diseases, laboratory tests at admission and during hospitalization, treatment, complications and outcomes data were collected [8-10]. Patients outcomes (discharge or death) and admitted to intensive care unit (ICU, yes or no) were followed up at discharge until Feb 15, 2020[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 37was defined as a positive test result, and a Ct-value of 40 or more was defined as a negative test. 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 such as age, sex, body mass index (BMI), exposure to source of transmission within 14 days (yes or no), the incubation period (defined as the time from exposure to source of transmission to onset of symptom), familial cluster (yes or no), health care workers (yes or no) and hospitalized patients/outpatients/visitors(yes or no) were collected. 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 treatment mainly included antiviral treatment, antibiotic treatment, glucocorticoid treatment, intravenous immunoglobulin therapy and Chinese medicine treatment. Other treatment options such as Oxygen inhalation, noninvasive ventilation, invasive mechanical ventilation, extracorporeal membrane oxygenation and continuous renal replacement therapy (CRRT) were also recorded. Clinical complications (lymphopenia, hypoxemia, shock, acute respiratory distress syndrome [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 median (IQR) for continuous variables and number (%) for categorical variables. The different characteristics between death and survival groups were

tested by Mann-Whitney U test (continuous variables) or Chi-square test (categorical variables). All statistical analyses were tested SPSS 22.0 (IBM). A two-sided a of less than 0·05 was considered statistically significant.

Results

Baseline characteristics

The initial study cohort included 104 adult patients. Two patients were excluded because of transfer during hospitalization, leaving 102 patients for analysis. Demographic details are shown in TABLE 1. The median (IQR) age was 54 years (37-67years) 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%], patients and/or visitors [9.8%]) and 10 patients (9.8%) had a familial cluster. Features of the signs and symptoms most commonly at admission were self-reported fever (81.4%), fatigue (54.9%), and dry cough (49.0%). The timeline of SARS-CoV-2 onset in included patients is shown in Figure 1.

Chest imaging examination showed that 18 patients (17.6%) had ground-glass opacity. Figure 2 showed chest computed tomographic images changing 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%), Cys-C (19.8%), Alanine aminotransferase (ALT, 24.8%) and NT-proBNP (37.5%). During hospitalization, the results of those biomarkers had increased to 76.5%, 62.7%, 34.7%, 47.5%, 62.5%. TABLE 2. All patients were treated in isolation. Patients treated with antiviral, antibiotic, glucocorticoid and mechanical ventilation were 98.0%, 99.0%, 50.0% and 19.6%, respectively. The median timing of initiation of antiviral therapy relative to onset of symptoms was 6 (3-7) days. As shown in the 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 therapy were Immunoglobulin (10.8%) and Methylprednisolone Sodium Succ (50.0%).

During hospitalization, 19.6%, 16.7% and 14.7% of patients had acute respiratory distress syndrome (ARDS), acute infection, acute cardiac injury, respectively. Eighteen patients (17.6%) were 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 death patients were caused by cardiac arrest.

Mortality

The median duration from the onset of symptoms to death and median time from exposure to SARS-CoV2 to death were 15(IQR, 9-21) days and 17(IQR, 12-24) days, respectively. Among patients who survived, they were younger (53[IQR, 47-66] vs. 72[63-81] years), more likely were health care workers (28.2% vs. 0) and less likely suffered from comorbidities (hypertension [20.0% vs. 64.7%], diabetes [5.9% vs. 35.3%] and chronic kidney disease[1.2% vs. 17.6%]). They were also less likely suffered from complications such as shock (3.5% vs. 41.1%), ARDS (5.9% vs. 88.2%), acute infection (3.5% vs. 82.4%), acute cardiac injury (3.5% vs. 70.6%), arrhythmia (7.1% vs. 70.6%), acute kidney injury (5.9% vs.88.2%), acute liver injury(24.7% vs.76.5%) and lymphopenia(71.8% vs.100.0%).TABLE 1. There was no difference in drug treatment rates between the survival and non-survival groups (antiviral therapy[P=0.749], antibiotic treatment[P=0.369], glucocorticoid therapy[P=0.184], intravenous immunoglobulin therapy [P=0.253] and Chinese medicine treatment[P=1.000]). TABLE 3. As shown in the table 3, survivors were more likely received treatment of Arbidol (37.6% vs. 5.9%; P=0.011) and less likely received

treatment of Carbapenems (17.6% vs. 58.8%; P<0.001) and Linezolid (2.4% vs. 17.6%; P=0.040) than those non-survivors.

Patients who survived less likely required admission to the ICU (14.1% vs. 35.3%). They required more length of hospital stay (11[7-14] *vs.* 9[6-17] days) and less hospital expenses (14464[8707-28605] vs. 50779[30134-116821] CNY). During hospitalization, non-survived patients more likely had elevated procalcitonin (100.0% vs. 76.5%), Cys-C (100.0% vs. 70.6), ALT (82.2% vs. 41.1), D-dimer (100.0% vs. 47.1%), troponin I (80.0% vs. 40.0%) and NT-proBNP (100.0% vs. 80.0%). TABLE 2. Chest imaging examination showed that death patients more likely had ground-glass opacity (41.2% vs. 12.9%).

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 development of systemic complications following onset of the illness and the 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 cytokine storm was formed [1, 12]. One study showed that 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 death patients were caused by cardiac arrest.

Our data support those described by others that COVID-19 infection results from human-to-human transmission, including familial clustering of cases, and nosocomial transmission [2, 9-10]. We showed that 33.3% of the included 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 Jan. 20, 2020, all medical workers in our hospital started to use protective clothing and goggles. Furthermore, since coronavirus diffusion takes place by droplet transmission, aerosolisation 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 pandemia [14]. However, in our study, some potential confounders such as small sample size, single patient type (mainly hospital staff and moderate to severe patients) and lack of discharge information should not be ignored. Further studies are warranted to explore natural history of COVID-19.

In this study, the mortality was 16.7%, which was higher than previous studies (range

from 4.3% to 11.0%) [1, 9-10]. It should be noted that a significant number of patients were still in hospital and mortality would continue to rise in previous studies [1, 9-10]. All the patients in our study had discharged or died. Our results were more likely close to real results. As of Feb 28, 2020, the national official statistic shows that the mortality in Wuhan and China are 4.47% (2169/48557) and 3.58% (2835/79251), respectively [15]. Furthermore, 4691 patients with SARS-CoV-2 infections were reported in overseas (51 countries), with 67 fatal cases (1.43%) [16]. Those results showed that the mortality rate of COVID-19 was less than SARS and MERS, which mortality up to 10% and 37% [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 number (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). Those results illustrated that SARS-CoV-2 may have a higher pandemic risk than SARS broken out in 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 2019-nCoV-induced lung injury or shock outside of a clinical trial. In this study, we found that most of treatment had no impact on survival. Even dead patients received more Carbapenems($P<0.001$) and Linezolid($P=0.040$). It was exciting that Arbidol seems to improve prognosis($P=0.011$). Furthermore, one study had identified 4 small molecular drugs [Prulifloxacin, Nelfinavir, Bictegravir, Nelfinaviras] with high binding capacity with 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 had been suggested as potential treatment for COVID-19 [24]. Further clinical trial studies need to validate these hypotheses.

Our study suffers from the usual limitations of the small samples and single-center. Our hospital is one of the major tertiary teaching hospitals and is responsible for the treating critically ill patients with COVID-19. Thus, our cohort might represent the more severe COVID-19 and the real mortality rates are overestimated. A recent large-sample and multicenter study showed that only 5.00% of the included COVID-19 patients were admitted to intensive care unit and 1.36% succumbed [25]. Second, we only recorded 17 died patients.

Therefore, we did not perform logistic regression analyses to assess 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 selective bias. Pregnant women [26] and children [27] also are equally sensitive to the SARS-CoV-2 virus. Lastly, we only include laboratory confirmed patients. In fact, RT-PCR assay had a considerable percentage of false negatives [28]. Huang et al. [28] suggested that use of chest CT in combination with negative RT-PCR assay for the SARS-CoV-2 but high clinical suspicion.

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

Author Contributions: Drs Cao and Tu contributed equally as the co-author. Drs Tu, Hu and Liu Q contributed equally as senior authors. Drs Cao and Tu had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Cao, Hu, Cheng, Yu, Tu, Liu Q

Acquisition, analysis, or interpretation of data: Cao, Hu, Cheng, Liu Y, Yu, Tu, Liu Q

Drafting of the manuscript: Cao, Hu, Tu, Liu Q

Critical revision of the manuscript for important intellectual content: Cheng, Liu Y, Yu.

Statistical analysis: Cao, Tu.

Administrative, technical, or material support: Cao, Hu, Cheng, Tu, Liu Q

Supervision: Cao, Hu, Cheng

Obtain funding: Tu, Liu Q

Acknowledgement: We thank all patients included in this study. We are really grateful to all the health workers around the world. Their expertise & humanity are fundamental to stop SARS-COV-2 from spreading further. Data available can be obtained from the corresponding author**.**

Disclaimer: The study funders/sponsors had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Funding/Support: This study was supported by funding from CAMS Innovation Fund for Medical Science (Dr, Liu Q, No.2017-I2M-1-016; Dr. Tu, 2019-I2M-2-006); Natural Science Foundation of Tianjin (Dr. Tu, No. 19JCYBJC26600) and China Postdoctoral Science Foundation funded project (Dr. Tu, No. 2019M660921).

Conflict of Interest Disclosures: None reported.

References

1. Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., ... & Cheng, Z. (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The Lancet. 395: 497-506.

2. Zhu, N., Zhang, D., Wang, W., Li, X., Yang, B., Song, J., ... & Niu, P. (2020). A novel coronavirus from patients with pneumonia in China, 2019. New England Journal of Medicine. DOI: 10.1056/NEJMoa2001017

3. Zhong, N. S., Zheng, B. J., Li, Y. M., Poon, L. L. M., Xie, Z. H., Chan, K. H., ... & Liu, X. Q. (2003). Epidemiology and cause of severe acute respiratory syndrome (SARS) in Guangdong, People's Republic of China, in February, 2003. The Lancet, 362(9393), 1353-1358.

4. Zaki AM, van Boheemen S, Bestebroer TM, Osterhaus ADME, Fouchier RAM. Isolation of a novel coronavirus from a man with pneumonia in Saudi Arabia. N Engl J Med 2012; 367: 1814–20.

5. Lu, R., Zhao, X., Li, J., Niu, P., Yang, B., Wu, H., ... & Bi, Y. (2020). Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. The Lancet. 395: 565-574.

6. Yang, X., Yu, Y., Xu, J., Shu, H., Liu, H., Wu, Y., ... & Wang, Y. (2020). Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. The Lancet Respiratory Medicine. https://doi.org/10.1016/S2213-2600(20)30079-5

7. Xu, X. W., Wu, X. X., Jiang, X. G., Xu, K. J., Ying, L. J., Ma, C. L., ... & Sheng, J. F. (2020). Clinical findings in a group of patients infected with the 2019 novel coronavirus (SARS-Cov-2) outside of Wuhan, China: retrospective case series. *bmj*, *368*.

8. Cao JL, Hu XR, Cheng W, Yu L, Tu WJ., & Liu Q. (2020). Clinical Features and Short-term Outcomes of 18 Patients with Corona Virus Disease 2019 in Intensive Care Unit. Intensive Care Medicine, DOI: 10.1007/s00134-020-05987-7.

9. Wang, D., Hu, B., Hu, C., Zhu, F., Liu, X., Zhang, J., ... & Zhao, Y. (2020). Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus–Infected

Pneumonia in Wuhan, China. JAMA. doi:10.1001/jama.2020.1585

10. Chen, N., Zhou, M., Dong, X., Qu, J., Gong, F., Han, Y., ... & Yu, T. (2020). Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. The Lancet. 395: 507-513.

11. Li, Q., Guan, X., Wu, P., Wang, X., Zhou, L., Tong, Y., ... & Xing, X. (2020). Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus–Infected Pneumonia. New England Journal of Medicine. DOI: 10.1056/NEJMoa2001316

12. Yang, Ming and Zhao, Jinming and Zhang, Zheng, More Than Pneumonia, The Potential Occurrence of Multiple Organ Failure in 2019 Novel Coronavirus Infection (February 5, 2020). Available at SSRN: http://dx.doi.org/10.2139/ssrn.3532272

13. Oudit GY, Kassiri Z, Jiang C *et al*: SARS-coronavirus modulation of myocardial ACE2 expression and inflammation in patients with SARS. Eur J Clin Invest 2009; 39(7):618-25.

14. Cabrini, L., Landoni, G., & Zangrillo, A. (2020). Minimise nosocomial spread of 2019-nCoV when treating acute respiratory failure. *The Lancet*. https://doi.org/10.1016/S0140-6736(20)30359-7

15.http://www.nhc.gov.cn/xcs/yqfkdt/202002/4ef8b5221b4d4740bda3145ac37e68ed.shtml

16.https://www.who.int/docs/default-source/coronaviruse/situationre-ports/20200228-sitrep-39-covid-19.pdf?sfvrsn=aa1b80a7_2

17. Wu, P., Hao, X., Lau, E. H., Wong, J. Y., Leung, K. S., Wu, J. T., ... & Leung, G. M. (2020). Real-time tentative assessment of the epidemiological characteristics of novel coronavirus infections in Wuhan, China, as at 22 January 2020. *Eurosurveillance*, *25*(3), 2000044.

18. Liu, T., Hu, J., Kang, M., Lin, L., Zhong, H., Xiao, J., ... & Deng, A. (2020). Transmission dynamics of 2019 novel coronavirus (2019-nCoV) (1/25/2020). Available at SSRN: https://ssrn.com/abstract=3526307

19. Wu, J. T., Leung, K., & Leung, G. M. (2020). Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan,

China: a modelling study. *The Lancet*. 395: 689-697.

20. Daszak, P., Olival, K. J., & Li, H. (2020). A strategy to prevent future pandemics similar to the 2019-nCoV outbreak. Biosafety and Health, https://doi.org/10.1016/j.bsheal.2020.01.003

26. Russell, C. D., Millar, J. E., & Baillie, J. K. (2020). Clinical evidence does not support corticosteroid treatment for 2019-nCoV lung injury. The Lancet. 395: 473-475.

22. Li, Y., Zhang, J., Wang, N., Li, H., Shi, Y., Guo, G., ... & Zou, Q. (2020). Therapeutic Drugs Targeting 2019-nCoV Main Protease by High-Throughput Screening. bioRxiv. doi: https://doi.org/10.1101/2020.01.28.922922

23. Wang, M., Cao, R., Zhang, L., Yang, X., Liu, J., Xu, M., ... & Xiao, G. (2020). Remdesivir and chloroquine effectively inhibit the recently emerged novel coronavirus (2019-nCoV) in vitro. *Cell Research*, https://doi.org/10.1038/s41422-020-0282-0

24. Richardson, P., Griffin, I., Tucker, C., Smith, D., Oechsle, O., Phelan, A., & Stebbing, J. (2020). Baricitinib as potential treatment for 2019-nCoV acute respiratory disease. The Lancet. DOI: https://doi.org/10.1016/S0140-6736(20)30304-4

25. Guan, W. J., Ni, Z. Y., Hu, Y., Liang, W. H., Ou, C. Q., He, J. X., ... & Du, B. (2020). Clinical characteristics of 2019 novel coronavirus infection in China. NEJM. DOI: 10.1056/NEJMoa2002032

26. Chen, H., Guo, J., Wang, C., Luo, F., Yu, X., Zhang, W., ... & Liao, J. (2020). Clinical characteristics and intrauterine vertical transmission potential of COVID-19 infection in nine pregnant women: a retrospective review of medical records. The Lancet. https://doi.org/10.1016/S0140-6736(20)30360-3

27. Shen, K., Yang, Y., Wang, T., Zhao, D., Jiang, Y., Jin, R., ... & Shang, Y. (2020). Diagnosis, treatment, and prevention of 2019 novel coronavirus infection in children: experts' consensus statement. World Journal of Pediatrics. https://doi.org/10.1007/s12519-020-00343-7

28. Huang, P., Liu, T., Huang, L., Liu, H., Lei, M., Xu, W., ... & Liu, B. (2020). Use of Chest CT in Combination with Negative RT-PCR Assay for the 2019 Novel Coronavirus but High Clinical Suspicion. Radiology. https://doi.org/10.1148/radiol.2020200330

Table 1: Baseline characteristics, complications and outcome of patients with COVID-19‡

	All	Non-survivors	Survivors	P
N	102	17	85	
Age, years	54(37-67)	72(63-81)	53(47-66)	<0.001
Sex-female	49(48.0)	4(23.5)	45(52.9)	0.0512
BMI, kg/m2	24.4(21.8-26.0)	26.0(23.4-28.7)	24.3(21.8-25.7)	0.088
Exposure to source of transmission within 14 days	47(46.1)	10(58.8)	37(43.5)	0.374
Familial cluster	10(9.8)	1(5.9)	9(10.6)	0.882
Infection				
Health care Workers	24(23.5)	0	24(28.2)	0.0284
Hospitalized patients and/or outpatients in past 14 days	10(9.8)	2(11.8)	8(9.4)	0.882
Signs and symptoms				
Fever	83(81.4)	12(70.6)	61(71.8)	0.844
Fatigue	56(54.9)	9(52.9)	47(55.3)	0.859
Dry cough	50(49.0)	8(47.1)	42(49.4)	0.863
Muscle ache	35(34.3)	5(29.4)	30(34.3)	0.641
Diarrhea	11(10.8)	3(17.6)	8(9.4)	0.568
More than one sign or symptom	92(90.2)	16(94.1)	76(89.4)	0.882
Comorbidities				
Any	47(46.1)	13(76.5)	34(40.0)	0.006
Hypertension	28(27.5)	11(64.7)	17(20.0)	<0.001
Diabetes	11(10.8)	6(35.3)	5(5.9)	<0.001
Cerebrovascular disease	6(5.9)	3(17.6)	3(3.5)	0.090
Cardiovascular disease	5(4.9)	3(17.6)	2(2.4)	0.040
Respiratory diseases	10(9.8)	4(23.5)	6(7.1)	0.101
Malignancy	4(3.9)	1(5.9)	3(3.5)	0.819
Chronic kidney disease	4(3.9)	3(17.6)	1(1.2)	0.012
Chronic liver disease	2(2.0)	1(5.9)	2(2.4)	0.462
Incubation period, days(n=47)	3(2-6)	3(2-4)	3(2-6)	0.563
Onset of symptom to, days				
Hospital admission	6(3-7)	6(3-8)	6(3-7)	0.690
Confirmed Diagnosis	8(5-14)	9(5-16)	8(5-13)	0.577
Transfer to ICU	18(17.6)	6(35.3)	12(14.1)	0.082
Length of hospitalized, days	11(7-15)	9(6-17)	11(7-14)	0.719
Cost of hospitalization, CNY	18138(8436-42450)	50779(30134-116821)	14464(8707-28605)	<0.001
Treatments				
Oxygen inhalation	76(74.5)	15(88.2)	61(71.8)	0.264
Noninvasive ventilation	5(4.9)	3(17.6)	2(2.4)	0.040
Invasive mechanical ventilation	14(13.7)	12(70.6)	2(2.4)	<0.001
Extracorporeal membrane oxygenation	3(2.9)	1(5.9)	2(2.4)	1.000
CRRT	6(5.9)	5(29.4)	1(2.4)	<0.001

Complications				
Shock	10(9.8)	7(41.1)	3(3.5)	<0.001
ARDS	20(19.6)	15(88.2)	5(5.9)	<0.001
Acute infection	17(16.7)	14(82.4)	3(3.5)	<0.001
Acute cardiac injury	15(14.7)	12(70.6)	3(3.5)	<0.001
Arrhythmia	18(17.6)	12(70.6)	6(7.1)	<0.001
Acute kidney injury	20(19.6)	15(88.2)	5(5.9)	<0.001
Acute liver injury	34(33.3)	13(76.5)	21(24.7)	<0.001
Lymphopenia	78(76.5)	17(100.0)	61(71.8)	0.028
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 median (IQR) for continuous variables and number (%) for categorical variables. The different characteristics between non-survivors and survivors were tested by Mann-Whitney U test (continuous variables) or Chi-square test (categorical variables).

MODS, multiple organ dysfunction syndrome; CRRT, continuous renal replacement therapy; ARDS, Acute respiratory distress syndrome; ICU, Intensive Care Unit; BMI, body mass index; CNY, China Yuan

Table 2 Radiologic and Laboratory Findings of patients with COVID-19 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) Bilateral infiltrate, 10(33.3)	3(17.6)	No change, 0(0) 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, *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)
≤1.1 * 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				
≥0.1ng/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)
≥7.6mmol/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)
≥1.2mg/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)
≥100pg/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(686-10700)	4740(2580-23850)
≥900pg/ml	6/16, 37.5%	10/16, 62.5%	12/15, 80.0%	15/15, 100.0%

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

ALT, Alanine aminotransferase; Cys-C, Cystatin-C; BNP, Brain natriuretic peptide; NT-proBNP, N-terminal pro brain natriuretic peptide

Table 3: The drug treatment of patients with COVID-19 during hospitalization[‡]

	All	Non-survivors	Survivors	P
N	102	17	85	
Antiviral therapy	100(98.0)	17(100.0)	83(97.6)	0.749
Arbidol Hydrochloride Capsules	33(34.3)	1(5.9)	32(37.6)	0.011
Oseltamivir	66(64.7)	14(82.4)	52(61.2)	0.095
Lopinavir and Ritonavir Tablets	28(27.5)	4(23.5)	24(28.2)	0.921
Antibiotic treatment	101(99.0)	17(100.0)	84(98.8)	0.369
Cephalosporins	34(33.3)	5(29.4)	29(34.1)	0.707
Quinolones	87(85.3)	13(76.5)	74(87.1)	0.453
Carbapenems	25(24.5)	10(58.8)	15(17.6)	<0.001
Linezolid	5(4.9)	3(17.6)	2(2.4)	0.040
Intravenous immunity therapy	11(10.8)	0(0)	11(12.9)	0.253
Immunoglobulin	11(10.8)	0(0)	5(5.9)	0.682
Thymosin Alpha for Injection	9(8.8)	0(0)	9(10.6)	0.349
Glucocorticoid therapy				
Methylprednisolone Sodium Succ	51(50.0)	11(64.7)	40(47.1)	0.184
Chinese medicine treatment				
Lianhuaqingwen capsule	3(2.9)	0(0)	3(3.5)	1.000

[‡]The results were presented as number (%) for categorical variables. The different characteristics between non-survivors and survivors were tested by Chi-square test (categorical variables).

Figure legends

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 non-survivors(N=17). The onset of symptom was defined as day 0. ICU admission time represent those patients were admitted to ICU (N=12 in the survivors; N=6 in the non-survivors). The points represent the median value. ICU, Intensive Care Unit; SARS-CoV-2, Severe Acute Respiratory Syndrome Coronavirus 2.

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 discharged on day 26 after symptom onset.

Figure 1

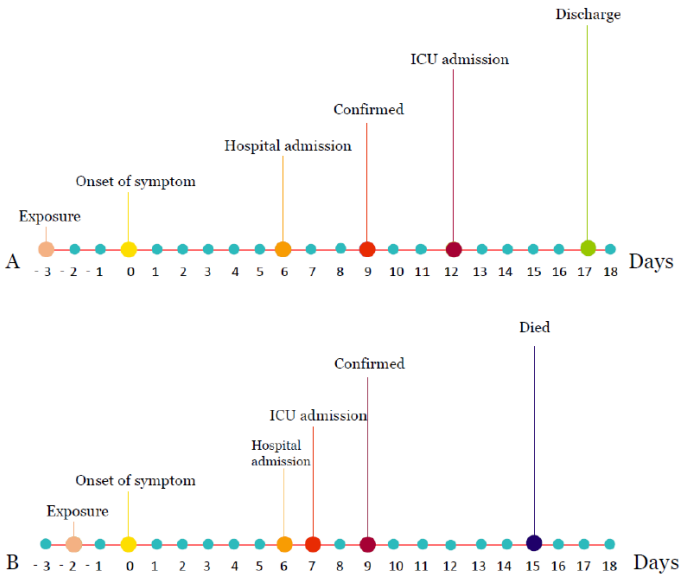
Discharge
ICU admission
Confirmed
Hospital admission
Onset of symptom
Exposure
A
-3 -2 -1 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
Days
Died
Confirmed
ICU admission
Hospital admission
Onset of symptom
Exposure
B
-3 -2 -1 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
Days

Figure 2