Clinical course and outcome of novel coronavirus COVID-19 infection in 107 patients discharged from the Wuhan hospital

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Abstract

Background In December 2019, Coronavirus Disease 2019 (COVID-19) outbreak was reported from Wuhan, China. Information on the clinical progress and prognosis of COVID-19 was not thoroughly described. We described the clinical courses and prognosis in COVID-19 patients.

Methods Retrospective case series of COVID-19 patients from Zhongnan Hospital of Wuhan University in Wuhan, and Xi-shui Hospital, Hubei Province, China, up to February 10, 2020. Epidemiological, demographic and clinical data were collected. Clinical progress of survivors and non-survivors were compared. Risk factors for death were analyzed.

Results A total of 107 discharged patients with COVID-19 were enrolled. The clinical progression of COVID-19 presented as a tri-phasic pattern. Week 1 after illness onset was characterized by fever, cough, dyspnea, lymphopenia and radiological multilobar pulmonary infiltrates. In severe cases, thrombocytopenia, acute kidney injury, acute myocardial injury or adult respiratory distress syndrome were observed. During week 2, in mild cases, fever, cough and systemic symptoms began to resolve and platelet count rose to normal range, but lymphopenia persisted. In severe cases, leukocytosis, neutrophilia and deteriorating multi-organ dysfunction were dominant. By week 3, mild cases had clinically resolved except for lymphopenia. However, severe cases showed persistent lymphopenia, severe acute respiratory dyspnea syndrome , refractory shock, anuric acute kidney injury, coagulopathy, thrombocytopenia and death. Older age and male sex were independent risk factors for poor outcome of the illness.

Conclusions A period of 7–13 days after illness onset is the critical stage in COVID-19 progression. Age and male gender were independent risk factors for death of COVID-19.

Background

In late 2019 a novel coronavirus, designated severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was identified as the cause of COVID-19 in Wuhan, a city in the Hubei province of China.[1, 2] Full-genome sequencing and phylogenic analysis indicated that SARS-CoV-2 is a betacoronavirus in the same subgenus as the SARS virus, but in a different clade.[2] The Middle East respiratory syndrome (MERS) virus, another beta-coronavirus, was more distantly related.[3] SARS-CoV-2 is 96% identical at the whole-genome level to a bat coronavirus, and suggesting that bats are the primary source.[4, 5] Epidemiologic investigations of initial cases showed COVID-19 was linked with exposure to Wuhan seafood market which also sold live rabbits, snakes, and other animals.[6] Subsequently, human-to-human transmission among close contacts has been the primary mechanism of transmission.[7] The disease has spread rapidly in China and more than 60,000 cases of COVID-19 have been reported. Sporadic cases have also been confirmed in other countries, mainly among travelers from Wuhan and their contacts.[8, 9]

The incubation period of COVID-19 is thought to be up to14 days following exposure.[6,7,10] The principal presenting features of COVID-19 are fever, cough, dyspnea and bilateral infiltrates on chest imaging.[11, 12]

Approximately 20 percent of patients progress to multi-organ dysfunction (including respiratory failure, septic shock, acute cardiac injury or acute renal failure. [11-13] However, a complete picture of the clinical progression of COVID-19 has not been reported. Except for infection control and supportive therapy, there is no specific therapy of COVID-19. Multiple organ support therapy is the corner stone in the treatment of critically ill patients with COVID-19.[13] Early recognition of risk factors for death would be useful to identify those potentially needing critical care at an early stage. Accordingly, a dynamic study was conducted to track clinical progression along the entire disease course. Risk factor analysis was performed to reveal important clinical features associated with the poor outcome.

Methods

Study Design And Participants

This case series was approved by the institutional ethics board of Zhongnan Hospital of Wuhan University and Xishui People's Hospital (No. 2020020). All the discharged (alive and dead) patients with confirmed COVID-19 from Zhongnan Hospital of Wuhan University and Xishui People's Hospital up to February 10, 2020, were enrolled. Oral consent was obtained from patients or patients' relatives. Zhongnan Hospital, located in Wuhan, Hubei Province, the endemic areas of COVID-19, is one of the major tertiary teaching hospitals and responsible for the treatments for COVID-19 assigned by the government. Xishui People's Hospital located in Huanggang city, another early endemic centre of COVID-19 in Hubei province. All patients with COVID-19 enrolled in this study were diagnosed according to World Health Organization interim guidance.[14] The methodology of RT-PCR used has been previously reported.[13]

Data Collection

The medical records of patients were analyzed by the research team of the Department of Critical Care Medicine, Zhongnan Hospital of Wuhan University. Epidemiological, clinical, laboratory, and radiological characteristics and treatment and outcomes data were obtained with data collection forms from electronic medical records and reviewed by a trained team of physicians. Information recorded included demographic data, medical history, exposure history, underlying comorbidities, symptoms, signs, laboratory findings, chest computed tomographic (CT) scans, treatment measures (ie, antiviral therapy, corticosteroid therapy, respiratory support, kidney replacement therapy) and outcome. The date of disease onset was defined as the day when the first symptom was noticed. acute respiratory distress syndrome (ARDS) was defined according to the Berlin definition. [15] Acute kidney injury (AKI) was identified according to the Kidney Disease: Improving Global Outcomes definition.[16] Cardiac injury was defined if the serum levels of cardiac biomarkers (eg, troponin I) were above the 99th percentile of upper reference limit or if new abnormalities were shown in echocardiography. Times from onset of disease to hospital admission, dyspnea, ARDS, ICU admission and hospital discharge were recorded.

Statistical analysis

Categorical variables were described using frequencies and percentage, while continuous variables were described using mean, median, and interquartile range (IQR) values. Means for continuous variables were compared using independent group Student's t tests when the data were normally distributed and the Mann-Whitney test when they were not. Proportions for categorical variables were compared using the χ2 test, although Fisher's exact test was used when the data were sparse. Univariate analyses were performed to evaluate the risk factors associated with death. Multiple logistic regression analysis was used to identify independent predictors of mortality. All the tests were two-tailed and p-value less than 0.05 was considered statistically significant. All analyses were processed by SPSS for Windows version 17.0 (SPSS, Chicago, IL, USA).

Results

Basic Characteristics

Basic characteristics of the 107 patients (95 from Zhongnan and 12 from Xi-Shui) are shown in Table 1. There were 88 survivors and 19 non-survivors. Median age was 51 years (IQR, 36−65; range, 19−92 years), 57 (53.3%) were male. Median times from first symptoms to hospital admission, dyspnea, and ARDS were 7 days (IQR, 3.5-9), 5.5 days (IQR, 2-9.3), and 7.5 days (IQR, 4.3−11), respectively. Median length of hospital stay was 11 days (IQR, 7−15). In this cohort of 107 patients, hypertension (26 [24.3%]), cardiovascular disease (13 [12.1%]) and diabetes (11 [10.3%]) were the most common coexisting conditions. The most common symptoms at onset of illness were fever (104 [97.2%]), dry cough (67 [62.6%]), fatigue (69 [64.5%]), dyspnea (35 [32.7%]), anorexia (33[30.8%]) and myalgia (33[30.8%]). Less common symptoms were sore throat, headache, dizziness, abdominal pain, diarrhea, nausea, and vomiting. At hospital admission median respiratory rate was 20/minute [IQR, 19−21] and mean arterial pressure was 89 mmHg [IQR, 83−98].

Table 1
Basic Characteristics of COVID-19 Patients.

Characteristics	Total (n = 107)	Survivors (n = 88)	Non-survivors (n = 19)	P Value
Age, years	51.0(36.0–65.0)	44.5(35.0-58.8)	73.0(64.0–81.0)	< 0.001*
Sex				0.003*
Male	57(53.3)	41(46.6)	16(84.2)	
Female	50(46.7)	47(53.4)	3(15.8)	
Comorbidity				
Hypertension	26(24.3)	16(18.2)	10(52.6)	0.001*
Cardiovascular disease	13(12.1)	6(6.8)	7(36.8)	0.002*
Diabetes	11(10.3)	6(6.8)	5(26.3)	0.024*
Chronic liver disease	6(5.6)	5(5.7)	1(5.3)	1.000
Cerebrovascular disease	6(5.6)	3(3.4)	3(15.8)	0.068
COPD	3(2.8)	2(2.3)	1(5.3)	0.447
Chronic kidney disease	3(2.8)	2(2.3)	1(5.3)	0.447
Symptoms and signs				
Fever	104(97.2)	85(96.6)	19(100.0)	1.000
Dry cough	67(62.6)	56(63.6)	11(57.9)	0.639
Fatigue	69(64.5)	55(62.5)	14(73.7)	0.356
Dyspnea	35(32.7)	20(22.7)	15(78.9)	< 0.001*
Anorexia	33(30.8)	25(28.4)	8(42.1)	0.241
Myalgia	33(30.8)	28(31.8)	5(26.3)	0.638
Pharyngalgia	12(11.2)	11(12.5)	1(5.3)	0.689

Data expressed as median (IQR) or N (%)

Abbreviations: COPD, chronic obstructive pulmonary disease; ARDS, acute respiratory distress syndrome; bpm, beats per minute.

Data are median (IQR), n (%). p values indicate differences between survivors and non-survivors. P < 0.05 was considered as significant. Vital signs including heart rate, respiratory rate and mean arterial pressure were collected at admission.

Characteristics	Total (n = 107)	Survivors (n = 88)	Non-survivors (n = 19)	P Value
Headache	7(6.5)	7(8.0)	0(0)	0.348
Dizziness	7(6.5)	7(8.0)	0(0)	0.348
Diarrhea	7(6.5)	3(3.4)	4(21.1)	0.018*
Nausea	6(5.6)	6(6.8)	0(0)	0.588
Vomiting	3(2.8)	2(2.3)	1(5.3)	0.447
Abdominal pain	2(1.9)	1(1.1)	1(5.3)	0.325
Onset of symptom to admission (d)	7.0(3.5-9.0)	7.0(3.0-9.8)	6.0(4.0−7.0)	0.405
Onset of symptom to dyspnea (d)	5.5(2.0-9.3)	7.0(3.3−10.8)	4.0(1.8−7.5)	0.103
Onset of symptom to ARDS (d)	7.5(4.3−11.0)	10.0(6.0−13.0)	7.0(3.5-9.0)	0.081
Length of hospital stay (d)	11.0(7.0−15.0)	10.5(7.0−14.0)	14.0(6.0−17.0)	0.561
Heart rate (bpm)	86(75−96)	85(75−96)	90(78−100)	0.240
Respiratory rate	20(19−21)	20(19−21)	22(20−24)	0.003*
Mean arterial pressure (mmHg)	89(83−98)	88(83−96)	95(89−101)	0.019*
Data expressed as median (IQR) or N (%)				
Abbreviations: COPD, chronic obstructive pulmonary disease; ARDS, acute respiratory distress syndrome; bpm, beats per minute.				
Data are median (IQR), n (%). p values indicate differences between survivors and non-survivors. P < 0.05 was considered as significant. Vital signs including heart rate, respiratory rate and mean arterial pressure were collected at admission.				

In comparison to the 88 hospital survivors, the 19 non-survivors were significantly older (median age, 73 years [IQR, 64−81] vs 44.5 years [IQR, 35-58.8]; p < .001) and were predominantly male (16 [84.2%] vs 41 [46.6%]; p = .003). Non-survivors were more likely to have underlying comorbidities, including hypertension (10 [52.6%] vs 16 [18.2%]; P = .001) and other cardiovascular disease (7 [36.8%] vs 6 [6.8%]; P = .002). Compared with the survivors, non-survivors were more likely to report dyspnea (15[78.9%] vs 20 [22.7%]; P < .001) and diarrhea (4[21.1%] vs 3[3.4%]; P = .018) at presentation. At hospital admission respiratory rate was higher in survivors than in non-survivors (22 [IQR 20−24] vs 20 [19−21]; p = .003). Similarly, mean arterial pressure was higher in non-survivors than in survivors (95 mmHg [IQR 89−101] vs 88 mmHg [83−96]; P = .019).

Laboratory Values And Radiographic Findings

Laboratory values and radiographic findings at hospital admission are shown in Table 2. Lymphopenia (0.9 × 10⁨/L [0.7−1.2]) and prolonged prothrombin time (12.8[11.9−13.5]) at admission were prominent features. 90 (84.1%) patients showed multi-lobar involvement on initial radiographs. 105 (98.1%) patients showed bilateral involvement on chest CT scan during hospitalization. A representative CT scan is shown in Fig. 1. Compared with survivors, on admission non-survivors had higher neutrophil counts (5.4 × 10⁨/L[3.2−8.5] vs 2.8 × 10⁨/L [2-3.9], P⁨0.001, lower platelet count (122 × 10⁨/L [83−178] vs 178 [139−207], P = 0.006) and higher D-dimer level (439 mg/L [202−1991] vs 191 mg/L [108−327], P = 0.003). Admission values of blood urea, creatinine, highly sensitive troponin I, serum creatine kinase, creatine kinase-MB, lactate dehydrogenase, alanine aminotransferase, aspartate aminotransferase were also significantly higher in the non-survivors.

Table 2
Initial Laboratory Values and Radiographic Findings of COVID-19 Patients.

	Normal range	Total (n = 107)	Survivors (n = 88)	Non-survivors (n = 19)	P Value
White blood cell count, × 10⁹/L	3.5–9.5	4.6(3.7–6.1)	4.4(3.4–5.8)	6.7(4.6–10.3)	0.004*
Neutrophil count, × 10⁹/L	1.8–6.3	3.1(2.1–4.7)	2.8(2.0-3.9)	5.4(3.2–8.5)	< 0.001*
Lymphocyte count, × 10⁹/L	1.1–3.2	0.9(0.7–1.2)	0.9(0.7–1.3)	0.8(0.5–1.1)	0.121
Platelet count, × 10⁹/L	125–350	175(129–200)	178(139–207)	122(83–178)	0.006*
Prothrombin time, s	9.4–12.5	12.8(11.9–13.5)	12.9(12.0-13.5)	12.6(11.9–13.5)	0.813
Activated partial thromboplastin time, s	25.1–36.5	31.7(29.4–33.9)	31.7(29.5–33.5)	32.7(27.5–37.0)	0.850
D-dimer, mg/L	0-500	203(121–358)	191(108–327)	439(202–1991)	0.003*
Creatine kinase, U/L	< 171	90(54–138)	86(53–121)	142(87–325)	0.022*
Creatine kinase-MB, U/L	< 25	14(10–18)	13(9–16)	18(13–44)	0.008*
Lactate dehydrogenase, U/L	125–243	236(176–369)	227(171–329)	456(254–588)	0.010*
Alanine aminotransferase, U/L	9–50	23(16–39)	22(15–34)	47(22–66)	0.002*
Aspartate aminotransferase, U/L	15–40	31(24–47)	29(23–41)	67(38–90)	< 0.001*
Total bilirubin, mmol/L	5–21	9.8(8.4–14.1)	9.5(8.4–12.9)	11.3(9.4–20.7)	0.069
Blood urea nitrogen, mmol/l	2.8–7.6	4.2(3.2–5.6)	3.9(3.0-4.7)	6.1(4.9–10.5)	< 0.001*
Creatinine, μmol/L	64–104	71(60–86)	68(58–83)	87(71–130)	< 0.001*

Abbreviations: MB, muscle and brain type; NA, not available.

Data are median (IQR), or n (%). P values indicate differences between survivors and non-survivors. P < 0.05 was considered as significant. Laboratory values and radiographic findings were collected at admission except that the bilateral involvement on radiographs was collected during hospitalization.

	Normal range	Total (n = 107)	Survivors (n = 88)	Non-survivors (n = 19)	P Value
Hypersensitive troponin I, > 26.2 pg/mL, No. (%)	< 26.2	6(5.6)	1(1.1)	5(26.3)	0.001*
Multilobar involvement on initial radiographs, No. (%)	NA	90(84.1)	73(83.0)	17(89.5)	0.731
Bilateral involvement on radiographs during hospitalization, No. (%)	NA	105(98.1)	86(97.7)	19(100.0)	1.000
Abbreviations: MB, muscle and brain type; NA, not available.					
Data are median (IQR), or n (%). P values indicate differences between survivors and non-survivors. P < 0.05 was considered as significant. Laboratory values and radiographic findings were collected at admission except that the bilateral involvement on radiographs was collected during hospitalization.					

Complications, Treatments And Outcome

Common complications included ARDS (28[26.2%]), shock (22 [20.6%]), AKI (14[13.1%]) and acute cardiac injury (12[11.2%]). Non-survivors were more likely to have one of these complications than survivors. Secondary infection was uncommon. Almost all patients received antiviral therapy (105 [98.1%]). Glucocorticoids were administered in 62 [57.9%] patients. Oxygen therapy was applied in (80 [74.8%] patients. In total, 20 patients required invasive mechanical ventilation. On day 1 of invasive mechanical ventilation, the median PaO2/FiO2 ratio was 103 (IQR 58–172) and the median APACHE II score was 25 (IQR 17–32). Three patients received extracorporeal membrane oxygenation (ECMO) therapy, Two of them survived and were discharged at day 26 and day 32, and one died due to sudden cardiac arrest after connection to the ECMO circuit. The causes of death included refractory ARDS (15 [78.9%]), septic shock (1 [5.3%]), sudden cardiac arrest (1 [5.3%]), hemorrhagic shock (1 [5.3%]) and acute myocardial infarction (1 [5.3%]).

Risk Factors Associated With Death For Covid-19

On univariate analysis, risk factors associated with death at hospital admission were older age, male gender, hypertension, diabetes, cardiovascular disease, raised white blood cell counts, elevated level of neutrophil counts, thrombocytopenia, creatine kinase-MB, lactate dehydrogenase, alanine aminotransferase, aspartate aminotransferase and creatinine (Table 3). On multivariable analysis, older age and male gender remained significant independent risk factors for death (Table 4).

Table 3

Univariate Analysis of Variable Associated With Death For COVID-19 Patients.

Variable	Univariable	
	OR (95% CI)	P Value
Age	1.102(1.054−1.152)	< 0.001*
Male	6.114(1.662−22.485)	0.006*
Hypertension	5.000(1.748−14.301)	0.003*
Diabetes	4.881(1.310-18.184)	0.018*
Cardiovascular disease	7.972(2.290-27.753)	0.001*
White blood cell count	1.239(1.055−1.455)	0.009*
Neutrophil count	1.257(1.073−1.472)	0.005*
Lymphocyte count	0.234(0.051−1.075)	0.062
Platelet count	0.987(0.977−0.997)	0.009*
Prothrombin time	1.084(0.737−1.595)	0.683
Activated partial thromboplastin time	0.998(0.979−1.017)	0.829
Creatine kinase	1.001(0.999−1.002)	0.277
Creatine kinase-MB	1.043(1.008−1.079)	0.015*
Lactate dehydrogenase	1.006(1.002−1.010)	0.004*
Alanine aminotransferase	1.020(1.002−1.038)	0.031*
Aspartate aminotransferase	1.034(1.015−1.054)	< 0.001*
Total bilirubin	1.070(0.995−1.149)	0.066
Blood urea nitrogen	1.001(0.985−1.016)	0.943
Creatinine	1.037(1.015−1.058)	0.001*
Abbreviations: MB, muscle and brain type.		
P < 0.05 was considered as significant and labeled with an asterisk (*) at the top corner of the P value.		

Table 4

Univariate and Multivariate Analysis of Risk Factors Associated With Death (19 patients.

Variable	Univariable		Multivariable	
	OR (95% CI)	P Value	OR (95% CI)	P Value
Age (years)	1.102(1.054–1.152)	< 0.001*	1.111(1.042–1.184)	0.001*
Male	6.114(1.662–22.485)	0.006*	7.224(1.298–40.190)	0.024*
Hypertension	5.000(1.748–14.301)	0.003*	1.099(0.264–4.580)	0.897
Cardiovascular disease	7.972(2.290-27.753)	0.001*	1.188(0.182–7.765)	0.857
Creatinine concentration	1.037(1.015–1.058)	0.001*	1.012(0.987–1.037)	0.342

P < 0.05 was considered as significant and labeled with an asterisk (*) at the top corner of the P value.

Discussion

Studies on COVID-19 have generally been limited to the description of the initial clinical, haematological, radiological and microbiological findings. Herein, we firstly described the clinical progression of virologically confirmed COVID-19. This study enrolled 107 discharged patients with COVID-19 which included 88 survivors and 19 non-survivors. We also analyzed the prognosis factors and found that age and male gender were the independent risk factor for mortality.

This study showed the clinical progression of COVID-19 presented as a tri-phasic pattern. Week 1 was characterized by fever, cough, dyspnea and other systemic symptoms. Most positive NAAT results could be obtained in week 1, which suggested that the symptoms were largely related to the effect of viral replication. In surviving patients, laboratory abnormalities included lymphopenia and prolonged prothrombin time. In non-survivors, emergence of systemic inflammation was evidenced by higher fever, respiratory rate, WBC counts and neutrophils count. Subsequently, multiple organ dysfunction syndrome (MODS) occurred with thrombocytopenia, renal failure, acute myocardial injury and ARDS. Notably, there was an obvious drop in body temperature around day 7, probably in relation to the widespread use of methylprednisolone as a rescue therapy.

During weeks 2 of illness, NAAT test became negative in surviving patients at a median of 13 days after illness onset. At the same time fever, cough and systemic symptoms began to resolve. However, lymphocyte counts still remained low, even as symptomatic illness resolved. This suggests that the lymphocytes are the main target of SARS-CoV-2 infection and the lymphocyte counts needs some time to recover. In the non-survivors, clinical status deteriorated and MODS developed during the second week.

In week 3, the organ functions improved in survivors, but continued to deteriorate the non-survivors. The lymphocyte counts dropped further and immune dysfunction became obvious in the non-survivors. These

patients developed severe ARDS necessitating ventilation and even ECMO support, septic shock supported by vasopressors, and an- end stage renal failure requiring continuous renal replacement therapy. Coagulation dysfunction and thrombocytopenia also developed. Death was inevitable due to multiorgan failure.

Notably, most non-survivors in our study were old male. Multivariate analysis showed older age and male gender were independent risk factors for death. A recent study examining single-cell RNA expression profiling of angiotensin converting enzyme 2 (ACE2), the cellular receptor of SARS-CoV-2, showed that Asian males had an extremely large number of ACE2-expressing cells in the lung.[17,18] A finding that might underlie the higher risk of death in this population.

After the incubation period, the frequent manifestations of COVID-19 were fever, cough, dyspnea, and bilateral infiltrates on chest imaging.[11-13] Evidence has shown that SARS-CoV-2 was found in the loose stool of a patient and potential transmission through the faecal–oral route should be considered.[19, 20] Consistent with the finding, some patients showed digestive symptoms (e.g. abdominal pain, diarrhea, nausea, and vomiting) at the illness onset. Multi-lobar involvement on initial chest CT was shown in most of our patients, consistent with a primary pulmonary method of acquisition.

Until now, no fully proven and specific antiviral treatment for the SARS-CoV-2 infection exists. Organ support therapy is the corner stone in the treatment of critically ill patients with SARS-CoV-2 infection. Remdesivir, a novel nucleotide analog antiviral drug has been used in the first case with COVID-19 in the US and a clinical trial of remdesivir in SARS-CoV-2 infection is in progress.[21] Remdesivir and chloroquine have been shown to effectively inhibit the SARS-CoV-2 in Vero E6 cells.[22] Moreover, the effects of abidol, oseltamivir or methylprednisolone in SARS-CoV-2 infection have not been fully evaluated.

This study has several limitations. First, the virus loads were not detected. We can't determine if the MODS or severity of illness were correlated with the sustained viral load. Secondly, due to the retrospective study, data about the values of creatine kinase, creatine kinase-MB and lactate dehydrogenase from day 11 to day 17 were missing. The enzyme activity couldn't be analyzed in week 3 after illness onset. Further study should be conducted to clarify the dynamic change of the three lab index. Third, only 107 patients with confirmed SARS-CoV-2 infection were enrolled in this study. Future study should be needed to enroll larger sample sizes to evaluate the clinical progression and analyze the risk factor for death in COVID-19.

Conclusions

Our experience in Wuhan revealed a period of 7–13 days after the onset of illness as the critical stage in COVID-19 progression. Age and male gender were independent risk factors for death of COVID-19.

Abbreviations

COVID-19, Coronavirus Disease 2019; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; MERS, Middle East respiratory syndrome; AKI, acute kidney injury; ARDS, acute respiratory dyspnea syndrome; IQR, interquartile range; NAAT, nucleic acid amplification test; PT, prothrombin time; APACHE II score, Acute Physiology and Chronic Health Evaluation II score; ECMO, extracorporeal membrane oxygenation; MODS, multiple organ dysfunction syndrome; ACE2, angiotensin converting enzyme 2

Declarations

Ethics approval and consent to participate

This case series was approved by the institutional ethics board of Zhongnan Hospital of Wuhan University and Xishui People's Hospital (No. 2020020).

Consent for publication

No individual participant data is reported that would require consent to publish from the participant (or legal parent or guardian for children).

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Both Drs Wang and Peng designed this project. Drs. Yin, Liu, Zhang, Zhou and B.Hu collected the data. Dr C Hu and M Jian were responsible for the statistical analysis. Dr Wang wrote the draft and Drs Xu, Li, Prowly, and Peng revised this draft. Dr Peng finalized this manuscript and all the authors approved the final version of this manuscript.

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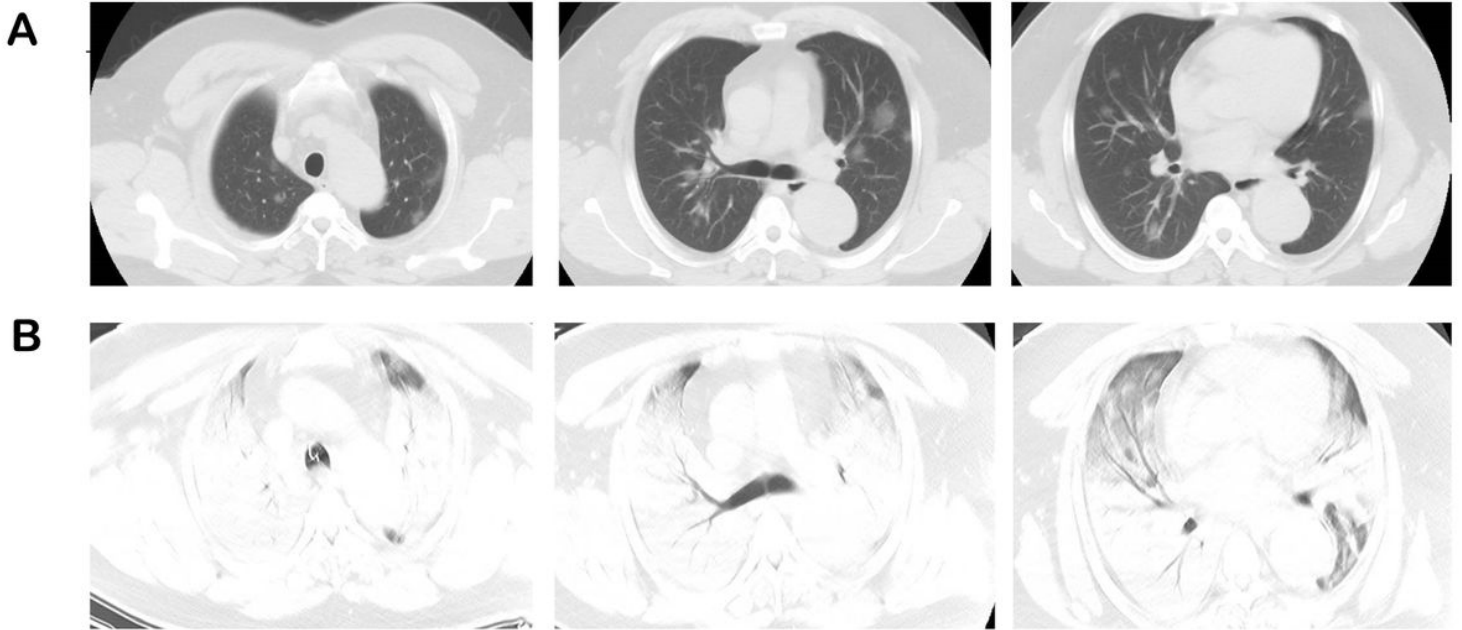
Figures

Figure 1

Chest Computed Tomographic Images of a 31-Year-Old Patient With COVID-19. A. Computed tomography images on day 4 after symptom onset was obtained on January 30, 2020. The images show ground glass opacity in both lungs. B. Computed tomography images on day 10 after symptom onset was obtained on February 4, 2020. The images demonstrate severe bilateral airspace disease with massive loss of normal aerated lung tissue. The patient suffered cardiac arrest due to severe ARDS, then was intubated, and was on ECMO in the intensive care unit.
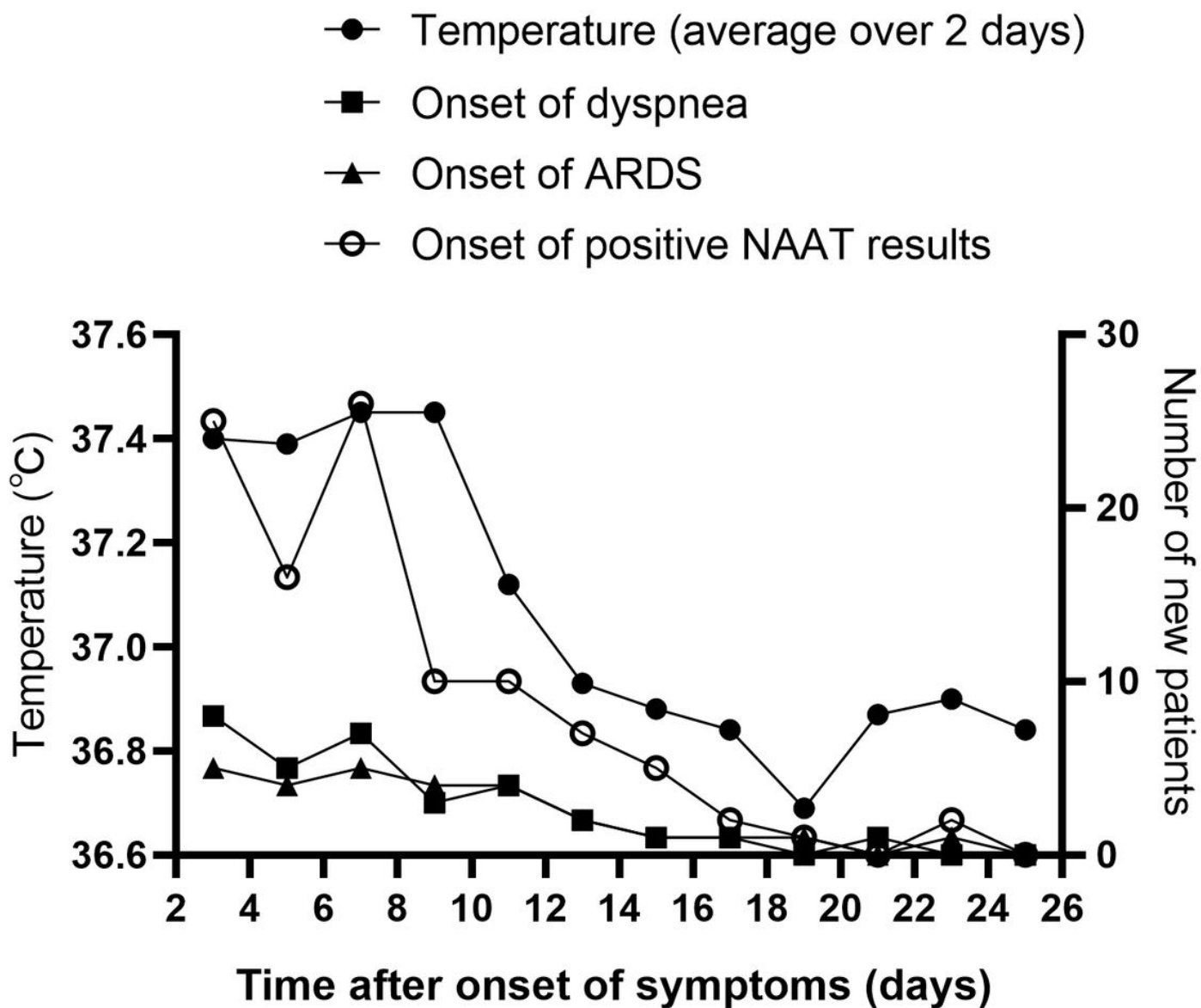
Figure 2

Temporal clinical profiles in 107 patients with COVID-19. Hollow symbols indicates the median time from illness onset to nucleic acid amplification test (NAAT) turning negative was 13 days.
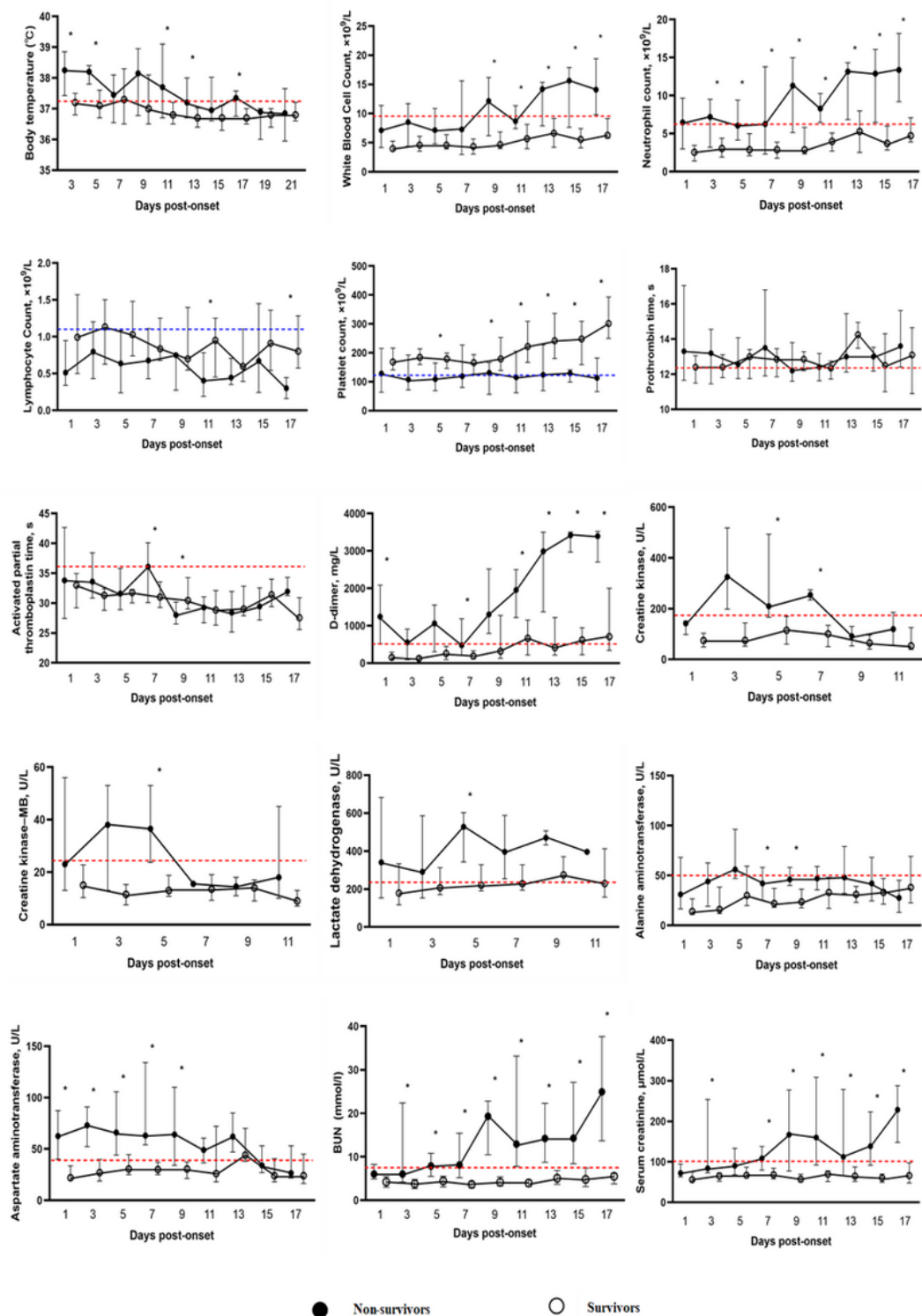
Figure 3

Dynamic Body Temperature and Laboratory Findings in 107 COVID-19 Patients. Timeline charts illustrate the temperature and laboratory parameters in 107 patients with COVID-19 (88 survivors and 19 non-survivors) every other day based on the days after the onset of illness. The dashed lines in red show the upper normal limit of each parameter, and the dashed line in blue shows the lower normal limit of lymphocyte count. * P <0 .05 for survivors vs non-survivors.