Clinical Features of 69 Cases with Coronavirus Disease 2019 in Wuhan, China

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Summary: In this retrospective case series that included 69 adults in Wuhan, 29% of patients showed dyspnea and 20% of cases showed SpO_2<90%. Patients with SpO_2<90% had a significantly higher risk of death. Abidol showed initial therapeutic effect.

ABSTRACT

Background

From December 2019 to February 2020, 2019 severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has caused a serious outbreak of coronavirus disease 2019 (COVID-19) in Wuhan, China. Related clinical features are needed.

Methods

We reviewed 69 patients who were hospitalized in Union hospital in Wuhan between January 16 to January 29, 2020. All patients were confirmed to be infected with SARS-CoV-2 and the final date of follow-up was February 4, 2020.

Results

The median age of 69 enrolled patients was 42.0 years (IQR 35.0-62.0), and 32 patients (46%) were men. The most common symptoms were fever (60[87%]), cough (38[55%]), and fatigue (29[42%]). Most patients received antiviral therapy (66 [98.5%] of 67 patients) and antibiotic therapy (66 [98.5%] of 67 patients). As of February 4, 2020, 18 (26.9%) of 67 patients had been discharged, and five patients had died, with a mortality rate of 7.5%. According to the lowest SpO_2 during admission, cases were divided into the $SpO_2 \geq 90\%$ group (n=55) and the $SpO_2 < 90\%$ group (n=14). All 5 deaths occurred in the $SpO_2 < 90\%$ group. Compared with $SpO_2 \geq 90\%$ group, patients of the $SpO_2 < 90\%$ group were older, and showed more comorbidities and higher plasma levels of IL6, IL10, lactate dehydrogenase, and c reactive protein. Arbidol treatment showed tendency to improve the discharging rate and decrease the mortality rate.

Conclusions

COVID-19 appears to show frequent fever, dry cough, and increase of inflammatory cytokines, and induced a mortality rate of 7.5%. Older patients or those with underlying comorbidities are at higher risk of death.

Key words: coronavirus, pneumonia, Wuhan.

Introduction

Coronaviruses are enveloped, single-stranded, positive-sense RNA viruses that are phenotypically and genotypically diverse, and widespread in bats around the world. Meanwhile, coronaviruses can also be found in many other species as well, including humans, other mammals, and birds. They may cause respiratory, enteric, hepatic, or neurologic diseases [1, 2]. In humans, there are four prevalent coronaviruses (229E, OC43, NL63, and HKU1), which typically cause common respiratory symptoms. Usually, the symptoms caused by coronaviruses are mild. But, in the past two decades, two other strains — severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) had caused two large-scale epidemics, with mortality rates of 10% for SARS-CoV and 37% for MERS-CoV [3].

In late December 2019, a series of cases with respiratory symptoms and typical chest computed X-ray tomography (CT) features were reported in Wuhan, Hubei, China. A previously unknown betacoronavirus was then discovered through the use of full-genome sequencing in samples from these patients, and was believed to be the pathogen of coronavirus disease 2019 (COVID-19) [4, 5]. The new betacoronavirus was named 2019 severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and formed another clade within the subgenus sarbecovirus, Orthocoronavirinae subfamily. COVID-19 has caused an outbreak in China since late December 2019 and continues to evolve with the number of suspected cases and deaths increasing daily in and outside of China.

We describe the epidemiological, clinical, laboratory, and radiologic features, treatment, and

prognosis of patients with confirmed infection of SARS-CoV-2 who were hospitalized in Union Hospital, Wuhan. The difference of clinical features between the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group would also been compared.

Methods

Patients

In late December 2019, several hospitals of Wuhan reported clusters of patients with pneumonia of unknown cause, which was identified as SARS-CoV-2 soon after. The local government proclaimed a list of designated hospitals to treat patients infected with SARS-CoV-2, including the Union hospital. Generally, the patients of Union hospital come from all regions of Wuhan, especially the Hankou district. In this program, all consecutive patients with confirmed COVID-19 admitted to the main campus of Union hospital from January 16 to January 29, 2020 were enrolled. All patients with COVID-19 enrolled in this study were diagnosed and admitted in accordance with the guideline of the national health commission of China [6]. The final date of follow-up was February 4, 2020.

Data collection

We reviewed clinical charts, nursing records, laboratory results, and chest CT characteristics for all patients. Epidemiological, clinical, imaging, and serological records and treatment and outcomes data were collected from the electronic medical network of Union hospital. To ensure the accuracy of data, two independent researchers were arranged to review and check the data form.

Real-time reverse transcription polymerase chain reaction tests (RT-PCR)

The confirmation of COVID-19 is achieved by RT-PCR detection of throat swab samples of suspected patients. Following the recommendation of China National Center for Disease Control, two target genes were set as described previously [5], including open reading frame1ab (ORF1ab) and nucleocapsid protein (N), and simultaneously amplified and tested during the real-time RT-PCR assay. Target 1 (ORF1ab): forward primer CCCTGTGGGTTTTACACTTAA; reverse primer ACGATTGTGCATCAGCTGA; and the probe 5'-FAM-CCGTCTGCGGTATGTGGAAAGGTTATGG-BHQ1-3'. Target 2 (N): forward primer GGGGAACTTCTCCTGCTAGAAT; reverse primer CAGACATTTTGCTCTCAAGCTG; and the probe 5'-FAM-TTGCTGCTGCTTGACAGATT-TAMRA-3'. A cycle threshold value (Ct value) less than 37 was defined as a positive record, and a Ct-value exceeds 40 was defined as a negative test.

Statistical analysis

Continuous variables were reported as median (IQR), and compared with the Mann-Whitney U test. Categorical variables were reported as number and percentages, and compared by χ^2 test or Fisher's exact test between the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group, or the groups treated specific agent or not. Statistical analysis was performed with SPSS software (version 26.0). A P value of less than 0.05 was considered to indicate statistical significance. All probabilities are two-tailed.

Results

The enrolled 69 patients were all confirmed infected with SARS-CoV-2 with PCR tests of throat swabs. The median age of the patients was 42.0 years (IQR 35.0-62.0, Table 1). Among them, 35 (51%) were aged 30-49 years, and 19 (28%) were aged 50-69 years (Figure 1). 32 patients (46%) were men, and 37 patients (54%) were women. According to the lowest SpO_2 records during admission, we divided these patients into two groups: the $SpO_2 \geq 90\%$ group (n=55) and the $SpO_2 < 90\%$ group (n=14). The median age of the $SpO_2 \geq 90\%$ group was 37.0 years (IQR 32.0-51.0), whereas the median age of the $SpO_2 < 90\%$ group was 70.5 years (IQR 62.0-77.0). In the $SpO_2 < 90\%$ group, the median occurrence time of lowest SpO_2 was 1 day (IQR 0-2.0) after admission, and the median interval from onset of illness to time of lowest SpO_2 during admission was 8.5 days (IQR 7.0-11.0). Of the 69 patients, less than half had underlying comorbidities (25[36%]), including hypertension (9[13%]), cardiovascular disease (8[12%]), and diabetes (7[10%]), et al. Patients of the $SpO_2 < 90\%$ group showed more underlying comorbidities when compared with the $SpO_2 \geq 90\%$ group, such as hypertension (5[36%] vs 4[7%], p=0.014), cardiovascular disease (5[36%] vs 3[5%], p=0.07), and diabetes (6[43%] vs 1[2%], p<0.001).

The most common clinical feature at the onset of illness was fever (60[87%]), 40 (58%) of them had a temperature reached 38.1-39.0°C and 13 (19%) of them had a temperature exceeded 39.0°C. On day 10 from onset of illness, the cases of fever declined to 30 (43%), and 15 (22%) of them had a temperature within 38.1-39.0°C. Other common clinical manifestations included cough (38[55%]), fatigue (29[42%]), and myalgia (21[33%]). Less common symptoms were sputum production, oppression in chest, dyspnea, diarrhea, and headache, et al (Table1).

Compared with the $SpO_2 \geq 90\%$ group, patients of the $SpO_2 < 90\%$ group tend to show more frequency of fever and dyspnea.

The blood counts of patients on admission showed decrease in neutrophils (26 [39%] of 67 patients), lymphocytes (28 [42%] of 67 patients), and eosinophils (48 [72%] of 67 patients), among which, the number of eosinophils in 31 patients was zero. Concurrently, patients of the $SpO_2 < 90\%$ group showed more frequency of lymphopenia than those of the $SpO_2 \geq 90\%$ group (11 [79%] of 14 patients vs 17 [32%] of 53 patients, p=0.002). The enrolled patients showed increase in alanine aminotransferase (23 [33%] of 69 patients) and aspartate aminotransferase (19 [28%] of 69 patients), most of which count less than 100 U/L. In terms of inflammation indicators, patients on admission showed increase in lactate dehydrogenase (25 [41%] of 61 patients), c reactive protein (42 [67%] of 63 patients), and erythrocyte sedimentation rate (30 [52%] of 58 patients), but not procalcitonin. The proportion and extent of the increase of lactate dehydrogenase, c reactive protein, and erythrocyte sedimentation rate are more prominent in the $SpO_2 < 90\%$ group (Table 2).

At the time of admission, all the 69 patients had abnormal chest CT findings. Figure 2 shows a typical evolution of chest CT of COVID-19, which belongs to a 74-year-old female. As shown in Figure 2A, the most common CT manifestation on admission is ground glass density enhancement along the outer bands of both lungs. Several days later, the ground glass opacity began to solidify, as shown in Figure 2B. With the development of disease, the consolidation absorbed gradually, as shown in Figure 2C and 2D. In general, the reabsorption takes much longer than consolidation. Figure 3 showed chest CT images of four patients, A/B in the

$SpO_2 \geq 90\%$ group and C/D in the $SpO_2 < 90\%$ group. The opacity area of the ground glass is larger and the consolidation degree is more serious in the $SpO_2 < 90\%$ group.

Plasma proportion of CD4-positive T lymphocytes, CD8-positive T lymphocytes, and B lymphocytes in patients with COVID-19 was within the normal range. The IL6 and IL10 levels in plasma exceeded the upper limit of normal value in both the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group. Notably, the level of IL6 in 7 (100%) of 7 patients in the $SpO_2 < 90\%$ group exceeded 20 pg/ml (Table 3).

The outcomes of two cases with hemopathy were lost because they were transferred to an infection specialized hospital and out of contact. So, they were not enrolled in the analysis of treatment and prognosis in Table 4. The median time from onset of symptoms to admission was 6.0 days (IQR, 4.0-9.0). The majority of patients needed oxygen support (43 [64.2%] in 67 patients). Most patients received antiviral therapy (66 [98.5%] of 67 patients) and antibiotic therapy (66 [98.5%] of 67 patients). Meanwhile, the use of antifungal drugs (8 [11.9%] of 67 patients) and corticosteroids (10 [14.9%] of 67 patients) was limited. Most antibiotics and antiviral therapy were empiric. 57 (85%) patients received interferon therapy and 39 (58%) patients received moxifoxacin treatment. 29 (43%) patients were examined for sputum culture and 5 were positive, including 2 cases of candida albicans, 2 cases of enterobacter cloacae and 1 case of acinetobacter baumannii. 28 (42%) patients were tested for respiratory pathogen antibody with blood samples and 4 were positive, including 2 cases of chlamydia IgG, 1 case of syncytial virus IgM, 1 case of adenovirus IgM. No immunosuppressive therapies were used during the anti-infection course.

In a multicenter double-blind randomized placebo-controlled study, compared with the placebo, arbidol reduced the time to elimination of symptoms and abated the positive rate of influenza on day 4 (25 vs 53%, $p < 0.05$) [7]. Arbidol was licensed for upper respiratory tract infection caused by influenza A / B virus in China. In this work, 36 (53.7%) patients received treatment of arbidol from the beginning of hospitalization at of dose of 0.4 g for three times a day. The median duration of arbidol was 9 days. By the time of February 4, 2020, 18 (26.9%) of 67 patients had been discharged, and five patients had died, with a mortality rate of 7.5% in this cohort. The principle of discharge was based on relief of symptoms, obvious absorption of inflammation in chest CT, abatement of fever, and viral clearance with throat swabs for two consecutive times. Usually, RT-PCR assays would be carried out when the previous 3 conditions were met, and the time interval between two RT-PCR assays was at least one day. Further, we evaluated the efficacy of arbidol. Resultly, 12 (33%) of 36 patients had been discharged in the arbidol-treated group, whereas 6 (19%) of 31 patients had been discharged in the arbidol-untreated group. All deaths occurred in the arbidol-untreated group. It appears that Arbidol treatment could improve the discharging rate and decrease the mortality rate (Table 5). The efficiency of antifungal agent and corticosteroids were also analyzed in Supplementary Table 1 and Table 2. Antifungal agent did not show influence on the outcome of COVID-19 patients. And use of corticosteroids were associated with a higher risk of death.

Discussion

Our experience with these 69 patients confirms that COVID-19 is a kind of epidemic pneumonia with fever, dry cough and fatigue as the most common onset symptoms. Most patients have mild manifestations and excellent prognosis. The elderly and the patients with underlying comorbidities are prone to develop severe condition.

From the existing data, human-to-human transmission of SARS-CoV-2 among close contacts has occurred since the middle of December and spread out gradually within a month after that [8]. Although Wuhan, origin of the epidemic, had been blocked since January 23, 2020, the number of patients infected with SARS-CoV-2 still raised rapidly. Based on the imported cases from Wuhan into other cities, the basic reproductive number for SARS-CoV-2 was 2.68. The epidemic doubling time was estimated to be 6.4 days [9]. The Chinese government has taken various measures to control population flow and prevent cross infection. But considering the imbalance between large number of infected patients and limited medical resources in Wuhan, family clustering cases should be worried [10].

There is a certain degree of similarity between 2019-CoV and SARS-CoV. Both of them caused frequent fever, cough, and fatigue, whereas the upper respiratory symptoms like pharyngalgia and rhinorrhea were less common [11, 12]. SARS-CoV targets human angiotensin- converting enzyme 2 (hACE2) and infects intrapulmonary epithelial cells more than cells of the upper airways [13, 14]. MERS-CoV attaches to its receptor, an exopeptidase called dipeptidyl peptidase 4 to infect its host [15]. It appears that SARS-CoV-2 uses the same cellular receptor as SARS-CoV did [16], so the replication of SARS-CoV-2 is more likely to happen in the lower respiratory tract rather than the throat. This hypothesis may be the reason for the false negative

results of throat swab specimens for RT-PCR assays of SARS-CoV-2. But whether efficient transmission of SARS-CoV-2 will only occur after signs of lower respiratory tract disease develop is still to be elucidated. SARS-CoV was able to infect multiple cell types in several organs, including circulating lymphocytes, the epithelial cells of the respiratory tract, and the mucosa of the intestine [17]. To be noticed, SARS-CoV-2 caused diarrhea in this cohort, which was also reported in two other researches [5, 18]. Given SARS-CoV-2 and SARS-CoV share 75%-80% gene sequence and genomic sequence was detected in the faeces of patients infected with SARS-CoV-2, it is likely that SARS-CoV-2 would infect more than just the respiratory system, which may complicate the treatment and prophylaxis of COVID-19 [16].

Consistent with SARS-CoV [19, 20] and MERS-CoV [21, 22], SARS-CoV-2 induced high level of cytokines in plasma. Evidence from SARS and MERS indicates that receiving corticosteroids did not improve mortality, but rather delayed viral clearance [23, 24]. In this cohort, only ten (14.9%) of 67 patients received corticosteroid treatment. Other two hospitals in Wuhan reported utilization rates of corticosteroids were 22% and 44.9% respectively [3, 5]. In fact, as far as we know, doctors in Wuhan are generally very cautious in using corticosteroids when treating COVID-19. According to published data from Chinese government, the mortality rate of COVID-19 was 4.1% for Wuhan and 2.0% for the whole mainland of China, which were much lower than that of SARS and MERS, indicating the limited using of corticosteroids was reasonable. Because of the limited sample in this work, the correlation between use of corticosteroids and death should be interpreted with caution. Currently, COVID-19 has no particularly effective treatment. The effect of antiviral therapy is still

controversial [25]. Here we tried to treat COVID-19 patients with arbidol. The mechanism of arbidol, a small indole-derivative molecule, involves inhibition of virus-mediated fusion with target membrane and a resulting block of virus entry into target cells [26, 27]. In this cohort, arbidol showed tendency to improve discharging rate and reduce mortality. Due to the limited sample size of this study, this tendency should be verified with a randomized controlled trial to assess the efficacy and safety of arbidol in patients infected with SARS-CoV-2 before it works for guiding treatment.

According to the guideline for management of COVID-19 from the national health commission of China [28], two consecutive times of negative nucleic acid tests of respiratory samples are needed before discharge. And the sampling time should be at least 24 hours apart. But, some patients still showed positive RT-PCR tests with SARS-CoV-2 after discharge [29]. Therefore, apart from the two times of negative tests for discharge, additional RT-PCR tests may be needed for some patients depending on viral burden, timing during the course of infection, and the sensitivity of the assays being used. After charge, patients are strongly suggested to be quarantined for monitoring health condition for two weeks.

As the size of this cohort is limited, the statistical analysis results should be interpreted with caution, and the p value without statistical significance does not necessarily reflect the exact situation of the whole population. Larger sample size of clinical studies is needed to elucidate the epidemiology, clinical characteristics and prognostic factors of COVID-19. Moreover, due to the outcomes data of two patients in the SpO_2<90% group was missing, the prognosis comparation between the SpO_2<90% group and the SpO_2≥90% group may be biased.

COVID-19 is becoming a global health threat. Finding the source of infection and studying the behavior of SARS-CoV-2 are crucial for understanding this epidemic. Although there are some false negatives, throat swab sampling for RT-PCR detection has been widely used in the screening of suspected patient. CT examination of the chest also provides great help in diagnosis and evaluation of the curative effect. Early diagnosis, timely isolation, and appropriate treatment are the keys in fighting this infection.

Declaration of interests

We declare no competing interests.

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Table 1: Characteristics and symptoms of patients infected with SARS-CoV-2.

	All patients(n=69)	SpO_2≥90%(n=55)	SpO_2<90%(n=14)	p value
Age(years)	42.0(35.0-62.0)	37.0(32.0-51.0)	70.5(62.0-77.0)	<0.001
Sex				
Male	32(46%)	25(45%)	7(50%)	0.761
Female	37(54%)	30(55%)	7(50%)	..
From admission to time of lowest SpO_2 (days)	2.0(1.0-3.0)	2.0(1.0-3.0)	1.0(0-2.0)	0.205
From onset to time of lowest SpO_2 (days)	8.0(7.0-11.0)	8.0(6.0-11.0)	8.5(7.0-11.0)	0.892
Comorbidity				
Hypertension	9(13%)	4(7%)	5(36%)	0.014
Cardiovascular disease	8(12%)	3(5%)	5(36%)	0.007
Diabetes	7(10%)	1(2%)	6(43%)	<0.001
Chronic obstructive Pulmonary disease	4(6%)	2(4%)	2(14%)	0.181
Malignancy	4(6%)	3(5%)	1(7%)	0.605
Asthma	2(3%)	2(4%)	0	0.633
Chronic hepatitis	1(1%)	1(2%)	0	0.797
Fever at onset of illness	60(87%)	47(85%)	13(93%)	0.674
Highest temperature, °C	38.5(38.0-38.8)	38.5(38.0-38.8)	38.3(38.0-38.8)	0.525
<37.3	9(13%)	8(15%)	1(7%)	0.579
37.3-38	7(10%)	5(9%)	2(14%)	..
38.1-39	40(58%)	31(56%)	9(64%)	..
>39	13(19%)	11(20%)	2(14%)	..
Fever on day 10 from onset of illness	30(43%)	21(38%)	9(64%)	0.079
Temperature, °C	37.1(36.8-38.0)	37.5(37.2-38.1)	37.0(36.7-38.0)	0.038
<37.3	39(57%)	34(62%)	5(36%)	0.296
37.3-38	13(19%)	9(16%)	4(29%)	..
38.1-39	15(22%)	11(20%)	4(29%)	..
>39	2(3%)	1(2%)	1(7%)	..
Symptoms at onset of illness				
Cough	38(55%)	30(55%)	8(57%)	0.553
Fatigue	29(42%)	22(40%)	7(50%)	0.499
Myalgia	21(30%)	19(35%)	2(14%)	0.124
Sputum production	20(29%)	16(29%)	4(29%)	0.624
Dyspnea	20(29%)	13(24%)	7(50%)	0.057
Oppression in chest	14(20%)	12(22%)	2(14%)	0.418
Diarrhea	10(14%)	8(15%)	2(14%)	0.674
Headache	10(14%)	10(18%)	0	0.086

anorexia	7(10%)	6(11%)	1(7%)	0.564
Chest pain	6(9%)	4(7%)	2(14%)	0.352

Continued: Table 1: Characteristics and symptoms of patients infected with SARS-CoV-2.

	All patients(n=69)	$SpO_2 \geq 90\%$(n=55)	$SpO_2 < 90\%$(n=14)	p value
Pharyngalgia	6(9%)	5(9%)	1(7%)	0.648
Dizziness	5(7%)	4(7%)	1(7%)	0.734
Palpitation	5(7%)	4(7%)	1(7%)	0.734
Vomiting	3(4%)	2(4%)	1(7%)	0.499

Abbreviations: SARS-CoV-2, 2019 severe acute respiratory syndrome coronavirus 2. Data are median (IQR) or n/N (%), where N is the total number of patients with available data. P values are comparing the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group from Mann-Whitney U test, χ^2 test, or Fisher's exact test. $P < 0.05$ was considered statistically significant.

Table 2: Laboratory records of patients infected with SARS-CoV-2 on admission.

	All patients(n=69)	SpO_2≥90%(n=55)	SpO_2<90%(n=14)	p value
White blood cell count, × 10^9 /L	3.82(2.98-5.57)	3.57(2.96-4.93)	6.52(4.30-7.73)	0.006
<4	36/67(54%)	33/53(62%)	3/14(21%)	0.007
4-10	30/67(45%)	20/53(38%)	10/14(71%)	..
>10	1/67(1%)	0/53	1/14(7%)	..
Neutrophil count, × 10^9 /L	2.35(1.62-367)	2.16(1.60-2.70)	5.24(2.90-6.44)	<0.001
≤2	26/67(39%)	24/53(45%)	2/14(14%)	0.034
>2	41/67(61%)	29/53(55%)	12/14(86%)	..
Lymphocyte count, × 10^9 /L	1.15(0.82-1.46)	1.19(0.95-1.46)	0.61(0.37-1.00)	0.002
<1.1	28/67(42%)	17/53(32%)	11/14(79%)	0.002
≥1.1	39/67(58%)	36/53(68%)	3/14(21%)	..
Monocyte count, × 10^9 /L	0.31(0.23-0.44)	0.31(0.24-0.46)	0.27(0.14-0.41)	0.22
Eosinophil count, × 10^9 /L	0.01(0.00-0.02)	0.01(0.00-0.02)	0.00(0.00-0.01)	0.195
<0.02	48/67(72%)	37/53(70%)	11/14(79%)	0.518
≥0.02	19/67(28%)	16/53(30%)	3/14(21%)	..
Haemoglobin, g/L	130.00(118.00-140.00)	131.00(121.00-141.00)	128.00(117.00-136.00)	0.507
Platelet count, × 10^9 /L	171.00(142.00-211.00)	172.00(138.00-206.00)	167.00(144.00-215.00)	0.829
Alanine aminotransferase, U/L	25.00(17.00-40.00)	24.00(16.00-40.00)	31.50(23.00-52.00)	0.119
≤35	46/69(67%)	38/55(69%)	8/14(57%)	0.527
>35	23/69(33%)	17/55(31%)	6/14(43%)	..
Aspartate aminotransferase, U/L	28.00(22.00-42.00)	26.00(21.00-39.00)	40.50(24.00-62.00)	0.03
≤40	50/69(72%)	43/55(78%)	7/14(50%)	0.048
>40	19/69(28%)	12/55(22%)	7/14(50%)	..
Creatinine, μmol/L	66.35(58.00-79.65)	65.30(58.00-78.50)	71.50(52.50-80.40)	0.623
Lactate dehydrogenase, U/L	224.00(183.00-291.00)	207.00(181.00-274.00)	517.50(267.00-549.00)	0.001
≤245	36/61(59%)	34/49(69%)	2/12(17%)	0.002
>245	25/61(41%)	15/49(31%)	10/12(83%)	..
C reactive protein, mg/L	13.20(6.78-49.00)	11.30(6.53-26.30)	81.55(48.85-105.90)	<0.001
<8	21/63(33%)	20/51(39%)	1/12(8%)	<0.001
8-50	28/63(44%)	26/51(51%)	2/12(17%)	..
50-100	8/63(13%)	2/51(4%)	6/12(50%)	..
≥100	6/63(10%)	3/51(6%)	3/12(25%)	..
Procalcitonin, μg/L	0.13(0.13-0.15)	0.13(0.13-0.15)	0.13(0.13-0.15)	0.78
<0.5	58/62(94%)	46/50(92%)	12/12(100%)	0.578
≥0.5	4/62(6%)	4/50(8%)	0/12	..

Continued: Table 2: Laboratory records of patients infected with SARS-CoV-2 on admission.

	All patients(n=69)	$SpO_2 \geq 90\%$(n=55)	$SpO_2 < 90\%$(n=14)	p value
Erythrocyte sedimentation rate, mm/h	20.00(8.00-31.00)	17.00(7.00-25.00)	30.00(27.00-49.00)	0.001
<20	28/58(48%)	27/45(60%)	1/13(8%)	0.001
≥20	30/58(52%)	18/45(40%)	12/13(92%)	..

Abbreviations: SARS-CoV-2, 2019 severe acute respiratory syndrome coronavirus 2. Data are median (IQR) or n/N (%), where N is the total number of patients with available data. P values are comparing the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group from Mann-Whitney U test, χ^2 test, or Fisher's exact test. $P < 0.05$ was considered statistically significant.

Table 3: Lymphocytes and cytokines records of patients infected with SARS-CoV-2 on admission.

	All patients(n=43)	$SpO_2 \geq 90\%$(n=36)	$SpO_2 < 90\%$(n=7)	p value
CD4-positive T lymphocytes (%)	38.19(32.14-43.5)	38.25(31.98-43.15)	37.90(32.82-54.21)	0.508
CD8-positive T lymphocytes (%)	26.00(20.62-30.03)	27.51(21.12-30.10)	22.86(18.64-25.08)	0.12
B lymphocytes (%)	11.43(7.54-14.63)	11.02(7.46-14.37)	12.68(8.30-23.00)	0.488
IL2, pg/ml	2.63(2.43-2.77)	2.63(2.43-2.77)	2.77(2.43-3.32)	0.156
IL4, pg/ml	2.00(1.81-2.26)	1.95(1.76-2.21)	2.26(1.95-2.31)	0.137
IL6, pg/ml	8.54(4.68-20.58)	6.69(4.44-12.43)	51.69(34.31-161.65)	<0.001
<3	3/43(7%)	3/36(8%)	0/7	<0.001
3-20	29/43(67%)	29/36(81%)	0/7	..
≥20	11/43(26%)	4/36(11%)	7/7(100%)	..
IL10, pg/ml	4.23(3.43-5.45)	4.18(3.31-5.275)	6.92(4.21-11.53)	0.013
<5	27/43(63%)	25/36(69%)	2/7(29%)	0.041
≥5	16/43(37%)	11/36(31%)	5/7(71%)	..
TNFα, pg/ml	2.08(1.93-2.34)	2.08(1.93-2.35)	2.14(1.90-2.34)	0.86
TNFγ, pg/ml	2.19(1.88-2.78)	2.19(1.91-2.72)	2.23(1.84-9.53)	0.392

Abbreviations: SARS-CoV-2, 2019 severe acute respiratory syndrome coronavirus 2. Data are median (IQR) or n/N (%), where N is the total number of patients with available data. P values are comparing the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group from Mann-Whitney U test, χ^2 test, or Fisher's exact test. $P < 0.05$ was considered statistically significant.

Table 4: Treatments and outcomes of patients infected with SARS-CoV-2.

	All patients(n=67)	$SpO_2 \geq 90\%$(n=55)	$SpO_2 < 90\%$(n=12)	p value
Onset of symptom to admission	6.0(4.0-9.0)	6.0(4.0-9.0)	7.0(4.0-9.0)	0.928
Oxygen support	43(64.2%)	31(56.4%)	12(100.0%)	0.003
Prognosis				
Hospitalization	44(65.7%)	38(69.1%)	6(50.0%)	<0.001
Discharge	18(26.9%)	17(30.9%)	1(8.3%)	..
Death	5(7.5%)	0	5(41.7%)	..
Involved treatment				
Antiviral therapy	66(98.5%)	55(100.0%)	11(91.7%)	0.179
Antibiotic therapy	66(98.5%)	54(98.2%)	12(100.0%)	0.638
Antifungal therapy	8(11.9%)	3(5.5%)	5(41.7%)	<0.001
Use of corticosteroids	10(14.9%)	6(10.9%)	4(33.3%)	0.048
Arbidol	36(53.7%)	32(58.2%)	4(33.3%)	0.118

Abbreviations: SARS-CoV-2, 2019 severe acute respiratory syndrome coronavirus 2. Data are median (IQR) or n/N (%), where N is the total number of patients with available data. P values are comparing the $SpO_2 \geq 90\%$ group and the $SpO_2 < 90\%$ group from Mann-Whitney U test, χ^2 test, or Fisher's exact test. $P < 0.05$ was considered statistically significant.

Table 5: Outcomes of COVID-19 patients treated with arbidol.

Prognosis	Arbidol-treated group (n=36)	Arbidol-untreated group (n=31)	p value
Hospitalization	24(67%)	20(65%)	0.03
Discharge	12(33%)	6(19%)	..
Death	0	5(16%)	..

Abbreviations: COVID-19, coronavirus disease 2019. Data are n/N (%), where N is the total number of patients with available data. P values are comparing the arbidol-treated group and the arbidol-untreated group from χ^2 test. $P < 0.05$ was considered statistically significant.

Figure 1

Title: Age distribution of 69 enrolled patients with coronavirus disease 2019 (COVID-19).

Legend: Number of hospital admissions by age group.

Figure 2

Title: Chest computed X-ray tomography (CT) images of a 74-year-old patient with coronavirus disease 2019.

Legend: (A) Chest CT images from a 74-year-old woman showing bilateral multiple ground glass density enhancement along the outer bands of both lungs on January 15, 2020. The ground glass opacity began to solidify on January 18, 2020 (B). With the development of disease, the consolidation absorbed gradually on January 21, 2020 (C) and January 26, 2020 (D).

Figure 3

Title: Chest computed X-ray tomography (CT) images of 4 cases with coronavirus disease 2019.

Legend: (A/B) Chest CT images of patients in the $SpO_2 \geqslant 90\%$ group showed bilateral ground glass density enhancement. (C/D) Chest CT images of patients in the $SpO_2 < 90\%$ group showed multiple areas of consolidation.

Figure 1

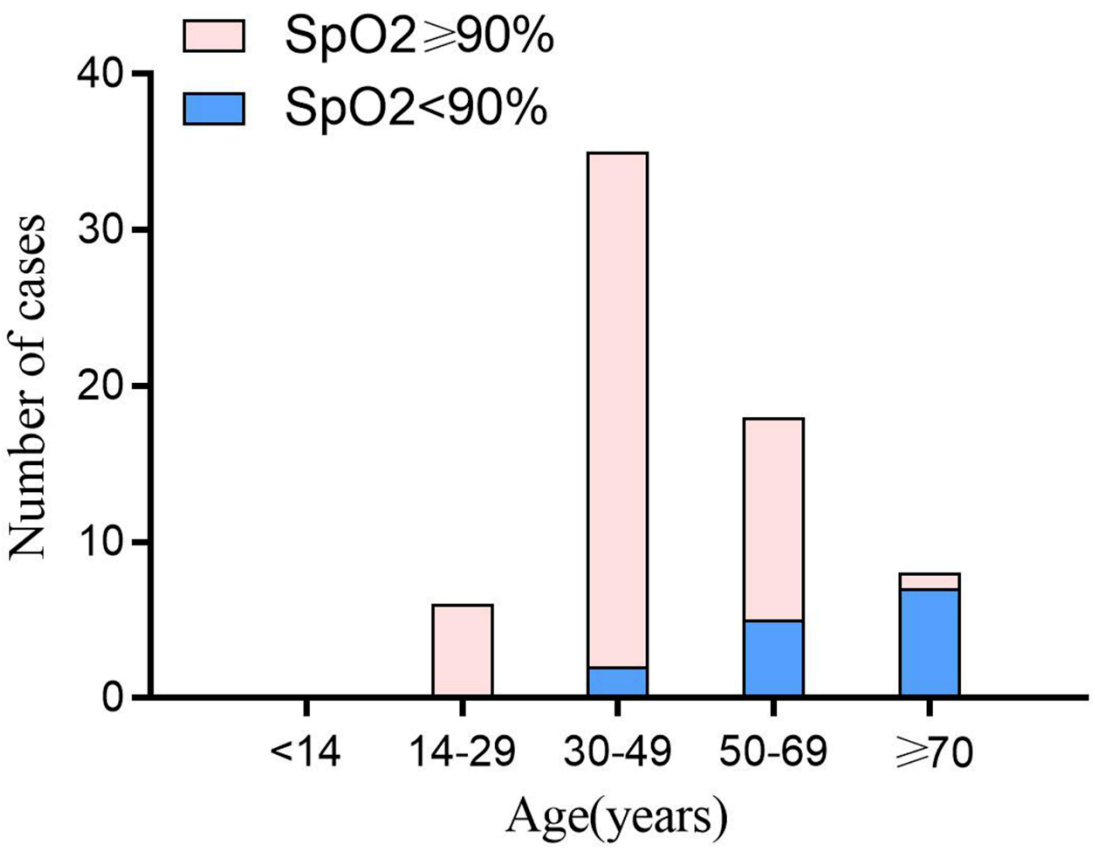

Figure 2

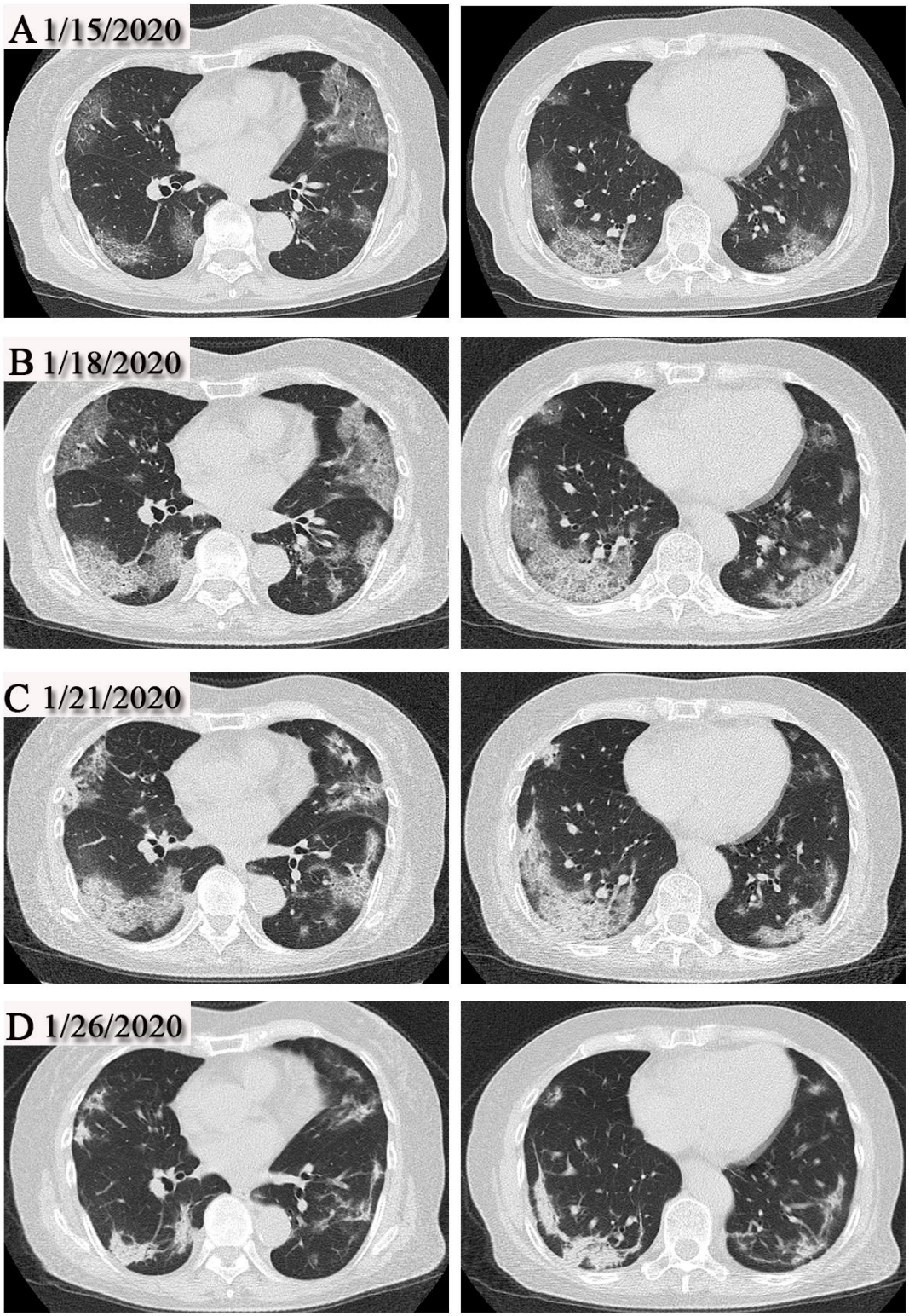

Figure 3