Analysis of clinical characteristics of SARS-CoV-2 infected cases: a retrospective study of medical records

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Statistical Epidemiology

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Abstract

In order to identify the clinical characteristics of patients with Corona Virus Disease 2019 (COVID-19) and find out the characteristic effects of 2019 New Coronavirus (SARS-CoV-2) infection on changes in clinical and laboratory data, we analyzed the medical records of 80 suspected cases who admitted in the national designated hospital due to the relevant clinical manifestations of SARS-CoV-2 infection from January 22 to February 13, 2020. 62 (77.5%) confirmed cases and 18 (22.5%) negative cases were confirmed by SARS-CoV-2 nucleic acid test. Epidemiological investigation and statistical analysis were carried out on the clinical and laboratory data of all suspected cases of COVID-19, the specific indicators were found, and the clinical characteristics of COVID-19 were described. Compared with the patients with negative nucleic acid test, the patients with positive nucleic acid test showed shorter time of onset of symptoms, higher plasma CO_2 level, lower eosinophil ratio, lower platelet count and hematocrit, lower serum sodium level, higher serum creatinine, higher blood urea and plasma albumin levels (all *P*<0.05). Our results might provide some suggestions in diagnosis, clinical treatment and prevention for COVID-19.

Background

Corona Virus Disease 2019 (COVID-19) is an acute respiratory infectious disease that first broke out in Wuhan, China in December 2019. The disease has caused global concern because of its rapid spread throughout the country and worldwide ^[1]. The Chinese government and people have actively responded and effectively suppressed the spread of COVID-19. On January 30, 2020, the World Health Organization (WHO) announced that SARS-CoV-2 was listed as a public health emergency of international concern ^[2,3]. SARS-CoV-2 belongs to the coronavirus family and is classified as β-coronavirus 2b lineage. Whole-genome sequencing and phylogenetic analysis showed that SARS-CoV-2 is related to human severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) ^[4]. At the entire genome level, the homology of SARS-CoV-2 to bat coronavirus is 96%, and bats are currently considered to be the most likely source wild animals to carry SARS-CoV-2^[5]. Common clinical manifestations of patients with COVID-19 included fever, fatigue, dry cough, prone to dyspnea and anorexia in severe cases, decreased lymphocytes, prolonged prothrombin time,

elevated lactate dehydrogenase, and typical scrubs for imaging of pneumonia Glassy Cloudiness (GGOs) ^[4,6].

Classifying the clinical characteristics of COVID-19 patients is of great significance for the rapid diagnosis of SARS-CoV-2 as well as clinical treatment and care. Herein, we analyzed the clinical characteristics of 62 SARS-CoV-2 nucleic acid positive cases and 18 negative cases. Based on the contact history, clinical symptoms, blood and biochemical test results, chest computed tomography (CT) results, and combined with the gold standard for clinical detection of virus RNA to diagnose suspected cases, we diagnosed 80 suspected cases admitted to hospital, reviewed the physiotherapy records of all cases, and found that compared with SARS-CoV-2 nucleic acid test negative cases, positive cases had shorter onset time of symptoms and higher plasma CO₂ levels. And the proportion of eosinophils decreased, platelet count and hematocrit decreased, the level of serum sodium was lower, while the levels of serum creatinine, blood urea and plasma albumin were higher in positive cases, suggesting that viral infection could damage bone marrow hematopoiesis, liver and kidney function. The characteristic changes of these indexes related to blood coagulation, hematopoiesis, liver and kidney function provide a new perspective for the diagnosis, clinical treatment and nursing of COVID-19.

Results

Presenting Characteristics

80 hospitalized patients with acute respiratory tract symptoms and unnormal radiology manifestations are involved in this study. Among it, 62 (77.5%) of the patients were confirmed to infected with SARS-Cov-2, while 18 (22.5%) of which showed negative results In addition, 16 (20%) of the patients were aged 15-44 years, 38 (48%) were aged 45-64 years and 26 (32%) were aged 65 or older than 65 (**Table 1**). Further, 3 (4%) of the patients shared the BMI (body mass index) under 18.5, 45 (56%) were from18.5 to 24.9, 27 (34%) were from 25 to 29.9 and 5 (6%) were equal to or over 30. The median age and BMI index were 56.09±13.75(mean±SD) and 23.93±3.226 respectively, and 46 (58%) of the 80 patients were men. Moreover, 37 (46%) demonstrated a traveling history to Wuhan or contacted with individuals coming from Wuhan. The median duration from onset of symptom to

hospital admission was 8.5 days (6.25-10). Among the 80 patients, hypertension (19, 24%) and diabetes (13, 16%) were the most common comorbidities. The most common symptoms during hospital stay were fever (63, 79%), dry cough (58, 73%), chest tightness (46, 58%) and dyspnea (21, 26%). All patients had radiology manifestations, and 77 (96%) showed bilateral involvement. Compared with other patients, patients who were diagnosed with COVID-19 by viral nucleic acid assay had lower median duration from onset of symptom to hospital admission (8 days, IRQ, 5-10 vs 10 days, IRQ, 7-15; *P*=0.014).

Laboratory and clinical parameters in COVID-19 and other patients Compared with other patients, numerous differences in laboratory results were found in COVID-19 patients (Table2). These differences between COVID-19 and other patients included lower eosinophil count $(0x10^{9}/L, IRQ, 0.0.02 \text{ vs } 0.2x10^{9}/L, IRQ, 0.0.4; P=0.031)$, lower platelet count $(194.18 \times 10^9 \pm 75.912 / L \text{ vs } 264.61 \times 10^9 \pm 124.933 / L; P=0.004)$, lower thrombocytocrit (0.19 ± 0.975 vs 0.25±0.103; P=0.012), lower prothrombin time (13.2 s, IRQ, 12.7-13.525 vs 13.65 s, IRQ, 12.975-14.3; P=0.045), higher activated partial thromboplastin time (40.23±6.330 s vs 35.84 ± 6.839 s; P=0.013), higher high-sensitivity c-reactive protein (22.18 mg/L, IRQ, 18.2-23.12 vs 18.585 mg/L, IRQ, 3.9125-22.38; *P*=0.046), higher creatinine (80.10±26.995 µmol/L vs 63.56±19.970 µmol/L; *P*=0.027), lower sodium (135.57 \pm 4.682 mmol/L vs 138.85 \pm 4.018mmol/L; P=0.02), lower carbondioxide $(24.83\pm2.437 \text{ mmol/L vs } 26.86\pm2.144; P=0.005)$, higher uric acid $(246.02\pm86.101 \mu \text{mol/L vs})$ $187.44 \pm 51.815 \mu mol/L; P=0.002$, higher urea (3.995 mmol/L, IRQ, 3.29-5.985 vs 3.2 mmol/L, IRQ, 2.425-3.65; P=0.007) and higher albumin (37.94±5.515 g/L vs 34.13±4.086 g/L; P=0.013). Among COVID-19 patients, more than 50% of them showed that lymphocyte count (48,77%), lymphocyte percentage (34, 55%), eosinophil count (46, 74%), sodium (32, 65%), phosphorus (14, 64%) and albumin (31, 60%) were below normal range, and that direct bilirubin (30, 58%), highdensity lipoprotein cholesterol (38, 68%) high-sensitivity c-reactive protein (50, 98%), D-dimer (48, 77%) and fibrinogen (51, 82%) were over normal range (**Table 3**).

Of 80 patients, 75 (94%) patients received antiviral treatment, 70 (88%) were given antibiotic treatment, 21(26%) were administrated immunoglobulin treatment, 14 (18%) were underwent oxygen

therapy, 11 (14%) were supervised with traditional Chinese medicine, 5(6%) were given probiotics tablet, and 1 (1%) was presented corticosteroid (**Table 4**). As of February 22, 28(35%) patients had been discharged and no patients had died.

Results of the multivariable logistic regression model for determining risk factors for positive outcome of the viral nucleic acid assay are displayed **(Table 5)**. Age, hypersensitive C-reactive protein and albumin were positively associated with positive result of the viral nucleic acid assay, while time from symptom occurrence to admission was negatively related to that.

Discussion

Increasing evidence suggested that COVID-19 is highly contagious ^[7]. It has been reported that its basic reproduction number (RO) is 3.77, which is estimated to spread to 3.77 other people per patient on average ^[7], and the incubation period of the disease can be as long as two weeks ^[2]. However, our report indicates the onset of symptom to hospital admission in SARS-CoV-2 nucleic acid test positive cases is less than that in SARS-CoV-2 nucleic acid test negative cases. Which suggesting that although there is a long incubation period after contact with COVID-19 patients, nucleic acid test positive cases after admission to hospital with suspected symptoms more quickly compared with negative cases after admission to hospital with suspected symptoms such as fever and cough. That indicates the onset of COVID-19 is sudden, and patients may show atypical or SARS symptoms in the early stage. The strong infectivity, long incubation period, rapid onset and the diversity of clinical symptoms of COVID-19, increase the difficulty of diagnosis, treatment, nursing and clinical research for it. As a new severe infectious disease, there is no particularly effective prevention and treatment for it. Hence it is essential to classify the specific clinical features of COVID-19, which will play a certain role in the prevention and treatment of COVID-19.

It has been reported that SARS-CoV-2 has pulmonary aggressiveness ^[8]. Our report shows that the blood CO_2 level of nucleic acid test positive cases is significantly increased, which is a sign of a significant decrease in the ability of the lungs to exchange gas, which confirms the pulmonary aggressiveness of SARS-CoV-2. The scary thing is that SARS-CoV-2 infection can also cause multiple

organ damage to the heart, liver, kidney and bone marrow.

We found that the level of high sensitivity C-reactive protein was significantly increased in patients with positive SARS-CoV-2 nucleic acid test. The function of high-sensitivity C-reactive protein is similar to that of traditional C-reactive protein, and its elevated level is one of the manifestations of inflammation^[9].And it has been reported that elevated high-sensitivity C-reactive protein may also be one of the early markers of myocardial injury^[10,11], which indicates that SARS-CoV-2 infection may cause myocardial injury in patients, although further pathological studies are needed to prove this idea. In addition, we found that compared with the patients with negative nucleic acid test, the patients with positive nucleic acid test had lower platelet count and hematocrit, shorter prothrombin time and longer activated partial thromboplastin time, which indicated that the coagulation function of patients with positive nucleic acid test was damaged by virus infection. Good blood coagulation depends on good hematopoiesis and liver function, so we speculate that hematopoiesis and liver function are damaged by SARS-CoV-2 infection, which is in line with similar previous research conclusions^[10,11]; We found that the level of serum sodium, which is closely related and interdepende to water^[12], in patients with positive nucleic acid test was lower than that in patients with negative nucleic acid test, which may be due to high fever and sweating caused by viral infection, acute loss of water or loss of digestive juice caused by vomiting and diarrhea. Studies have shown that fever (91.7%), cough (75.0%), fatigue (75.0%) and gastrointestinal symptoms (39.6%) after SARS-CoV-2 infection are the most common clinical manifestations ^[13]. Sweating and loss of digestive juices both cause sodium to be excreted with the liquid. However, patients with negative nucleic acid test also have high fever, sweating and loss of digestive juice. whether the symptoms of hyponatremia in patients with positive nucleic acid test are the consequence of renal dysfunction caused by viral infection is a conjecture worth considering. The evidence to support this conjecture also includes that the blood creatinine, uric acid and urea in patients with positive nucleic acid test are significantly higher than those in patients with negative nucleic acid test (see Table2 for details). The level of plasma albumin in patients with positive nucleic acid test is higher than that in patients with negative

nucleic acid test, which is another situation that we should pay attention to. Plasma albumin is the most abundant protein in plasma, and it is also a kind of nutrient. in special cases, its decomposition can produce amino acids that participate in the synthesis of tissue proteins ^[14], or oxidative decomposition to produce energy ^[15]. In addition, plasma albumin can also play a role in stabilizing globulin, which is of great significance in antiviral effect ^[16]. High fever and sweating after virus infection, acute dehydration caused by diarrhea and vomiting will lead to an increase in the concentration of serum albumin. At the same time, in order to make up for the decrease of plasma osmotic pressure caused by the decrease of blood sodium, plasma albumin will also increase adaptively. The increase of albumin will protect the body in the early stage of SARS-CoV-2 infection, but some clinicians have found that the level of plasma albumin will continue to decrease with the progress of infection, which may be due to the difficulty of eating caused by the continuous progress of COVID-19, or it may be the liver function damage caused by SARS-CoV-2 infection that just discussed leads to the decrease of albumin.

In the process of analysis, we also found that the age of patients with positive nucleic acid test was higher than that of patients with negative nucleic acid test, and it was statistically significant. This shows that the older group is more likely to be infected with SARS-CoV-2, which is related to their declining immune function and physical fitness; comparing the number of eosinophils in the sample, it can be found that the number of patients with positive nucleic acid test is lower, which may be the result of treatment with corticosteroids during treatment, or it may be caused by excessive production of adrenocortical hormones under physical stress during the disease. These two phenomena are helpful for us to understand SARS-CoV-2 infection, but they are not meaningful for the treatment of COVID-19.

SARS-CoV-2 infection may cause damage to multiple organs of patients. SARS-CoV-2 with multiple organ invasion is the clinical characteristic description of our SARS-CoV-2 infection. The pathological changes of multiple organ damage caused by virus infection need to be further developed in the pathological study of COVID-19 infection, but the occurrence of this situation undoubtedly increases

the risk of death and the difficulty of clinical treatment of patients. In clinical treatment, we must pay attention to monitoring the changes of lung function, liver function, renal function and hematopoietic function and correct the symptoms of disorders in time, which will play a very positive role in improving the prognosis of patients.

Conclusion

SARS-CoV-2 nucleic acid test, there are 62 (77.5%) SARS-CoV-2 nucleic acid test positive cases and 18 (22.5%) nucleic acid test negative cases. By comparing the laboratory data of all positive cases and negative cases, we find that compared with nucleic acid test negative cases. The patients with positive nucleic acid test had higher blood CO2 level, lower eosinophil ratio, lower platelet count and crit, lower serum sodium level, and higher serum creatinine, blood urea and plasma albumin levels, suggesting that SARS-CoV-2 infection will cause damage to many organs, especially liver and kidney function and bone marrow hematopoietic function. This is our description of the clinical characteristics of SARS-CoV-2 infection. During clinical treatment, we must pay close attention to patients' blood CO2, blood urea, serum creatinine levels, blood coagulation function, timely detection of patients' liver and kidney function, and timely correction of disorders, which will play a positive role in improving the prognosis of patients.

Methods

Study design and participants

From January 22 to February 13, 2020, we enrolled consecutive patients with acute respiratory tract symptoms admitted to a hospital (Ezhou, Hubei, China). All enrolled patients were tested for nucleic acid of SARS-CoV-2 during the hospital stay. The diagnosis of SARS-CoV-2-infected pneumonia is made according to World Health Organization interim guidance. Written informed consent were acquired from the patients involved. This case series is approved by the Ethics Committee of Medical Department of Wuhan University.

Data collection

For this retrospective study, patients' clinical records were obtained and organized into tabular data. The information collected involved demographic data, exposure history, medical comorbidities, signs, symptoms, laboratory findings, treatment measures (including antiviral therapy, antibacterial

therapy, corticosteroid therapy, oxygen therapy and other supportive treatments) and outcomes of the enrolled patients. For ensuring data accuracy, the data forms were independently reviewed by two investigators of our research team.

Nucleic acid testing for SARS-CoV-2

Patients' throat swab samples were obtained and then placed into a collection tube with viral transport media. Total RNA was extracted from the samples within 2h through use of respiratory sample RNA isolation kit, followed by reverse transcription. Subsequently, real-time reverse transcription polymerase chain reaction (RT-PCR) assay was performed to quantified two target genes, nucleocapsid protein(N) and open reading frame 1ab (ORF1ab). Target gene 1 (N): forward primer GGGGAACTTCTCCTGCTAGAAT; reverse primer CAGACATTTTGCTCTCAAGCTG; and the probe 5`-FAM- TTGCTGCTGCTGACAGATT-TAMRA-3`. Target gene 2 (ORF1ab): forward primer CCCTGTGGGTTTTACACTTAA; reverse primer ACGATTGTGCATCAGCTGA; and the probe 5`-VIC-CCGTCTGCGGTATGTGGAAAGGTTATGG-BHQ1-3`. The conditions for performing the real-time RT-PCR were incubation at 50°C for 15 minutes and 95°C for 3 minutes, followed by 40 cycles of denaturation at 94°C for 15 second as well as extending and collecting fluorescence signal at 55 °C for 45 second. Diagnostic criteria of COVID-19 were that a positive result is defined as a cycle threshold value (Ct-value) lower than 37, and a negative result is defined as Ct-value equal to or higher than 40. A medium Ct-value ranging between 37 and less than 40 required retesting for further confirmation. Statistical Analysis

Continuous variables were described by values of mean and standard deviation (SD) as well as median and interquartile range (IQR), categorical variables were presented as number and percentage. For comparing continuous variables, mean (SD) and independent -samples tests were employed when the data fitted normal distribution; otherwise, median (IQR) and Mann-Whitney tests were applied. For categorical variables, their proportions were compared by using chi-square tests and Fisher's exact tests as appropriate. The variables associated with the diagnosis of COVID-19 were elevated by logistic regression models. The statistically significant variables (P-value of <0.05) were selected into the final model. The patients with negative results were used as the reference group to

calculate odds ratio. All statistical analyses were conducted with IBM SPSS Statistics 19.0.

Abbreviations

Corona Virus Disease 2019 (COVID-19)

World Health Organization (WHO)

severe acute respiratory syndrome (SARS)

- Middle East respiratory syndrome (MERS)
- pneumonia Glassy Cloudiness (GGOs)
- computed tomography (CT)
- reproduction number (RO)
- standard deviation (SD)
- interquartile range (IQR)
- cycle threshold value (Ct-value)

real-time reverse transcription polymerase chain reaction (RT-PCR)

Declarations

- Ethics approval and consent to participate: This case series is approved by the Ethics Committee of Medical Department of Wuhan University.
- 2. Consent for publication: All authors agree to publish.
- 3. Availability of data and material: Availability of data and information.
- 4. Competing interests: None of the authors have any competing interests.
- 5. Funding: No financial support.
- 6. Authors' contributions:

Han Zhang did a formal analysis and wrote the original draft.

Lian Lu did a investigation and found resources.

Wei Hu found a methodology and supervised the whole process.

Jian Zhang conducted data planning and performed data analysis using software.

Wei Zhu conceptualized and visualized the subject.

Qi-Qiang He conceptualized the topic and looked for a verification method.

Corresponding Author Cheng-Cao Sun is also responsible for data planning, form analysis, and supervision.

Corresponding Author De-Jia Li was responsible for project management and review and editing of the original draft.

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Tables	;
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	Totaln=80	COVID-19 patientsn=62	Other patientsn=18	P value
Age groupsyears	56.09±13.75	56.26±14.17	55.50±12.57	0.838
≤14	0(0%)	0(0%)	0(0%)	0.135
15-44	16(20%)	10(16%)	6(33%)	
45-64	38(48%)	29(47%)	9(50%)	
≥65	26(32%)	23(37%)	3(17%)	
Sex				
Male	46(58%)	37(60%)	9(50%)	0.59
Female	34(42%)	25(40%)	9(50%)	
Body mass index	23.93±3.226	23.81±3.28	24.32±3.09	0.563
Underweight<18	3(4%)	2(3%)	1(6%)	0.514
.5				
Ideal18.5-24.9)	45(56%)	37(60%)	8(44%)	
Overweight25-	27(34%)	19(31%)	8(44%)	
29.9				
Obesity≥30	5(6%)	4(6%)	1(6%)	
Occupation				
Self-employed	8(10%)	4(6%)	4(22%)	0.142
Employed	31(39%)	22(35%)	9(50%)	
Agricultural	13(16%)	11(18%)	2(11%)	
worker				
Retired	22(28%)	19(31%)	3(17%)	

Unemployed	6(7%)	6(10%)	0(0%)	
Education				
Primary School	17(21%)	13(21%)	4(22%)	0.229
Junior	16(20%)	15(24%)	1(6%)	
High school	16(20%)	10(16%)	6(33%)	
(technical				
secondary				
school)				
Junior College	19(24%)	13(21%)	6(33%)	
University	7(9%)	6(10%)	1(6%)	
Illiteracy	5(6%)	5(8%)	0(0%)	
Smoking history	2(3%)	1(2%)	1(6%)	0.402
Drinking history	7(9%)	4(6%)	3(17%)	0.381
Underlying				
disease				
Hypertension	19(24%)	17(27%)	2(11%)	0.264
Diabetes	13(16%)	10(16%)	3(17%)	1
Exposure history				
Yes	37(46%)	28(45%)	9(50%)	0.327
No	32(40%)	27(44%)	5(28%)	
unknown	11(14%)	7(11%)	4(22%)	
Onset of	8.56.25 - 10	85 - 10	107 - 15	0.014
symptom to				
hospital				
admission				
Signs and				
symptoms				
Fever	63(79%)	49(79%)	14(78%)	1
Dry cough	58(73%)	45(73%)	13(72%)	1
Chest tightness	46(58%)	34(55%)	12(67%)	0.427
Dyspnea	21(26%)	13(21%)	8(44%)	0.091
Expectoration	27(34%)	23(37%)	4(22%)	0.273
Fatigue	28(35%)	20(32%)	8(44%)	0.404
Myalgia	13(16%)	7(11%)	6(33%)	0.062
Palpitation	11(14%)	9(15%)	2(11%)	1
Headache	14(18%)	9(15%)	5(28%)	0.341
Poor appetite	10(13%)	6(10%)	4(22%)	0.312
Nausea	9(11%)	6(10%)	3(17%)	0.687
Vomiting	2(3%)	2(3%)	0(0%)	1
Diarrhea	2(3%)	1(2%)	1(6%)	0.402
Chest CT				
Unilateral	3(4%)	1(2%)	2(11%)	0.125
involvement				
Bilateral	77(96%)	61(98%)	16(89%)	
involvement				

Table 1 Baseline Characteristics of COVID-19 Patients.

Normal Range	All patientsn=80	COVID-19	Oth
		patientsn=62	

White blood cell count (10 ⁹ /L)	3.5-9.5	4.92±1.886	4.94±1.985	4.
Lymphocyte count (10 ⁹ /L)	1.1-3.2	0.810.6-1.07	0.780.5275-0.9725	0.905
Monocyte count (10 ⁹ /L)	0.1-0.6	0.35±0.148	0.36 ± 0.158	0.
-	0.02-0.52	00-0.03	00-0.02	1
Eosinophil count (10 ⁹ /L)	0-0.6	0.0050-0.01	00-0.01	0
Basophil count (10 ⁹ /L)				
Neutrophil count (10 ⁹ /L)	1.8-6.3	3.66±1.810	3.69±1.906	3.
Red cell count (10 ¹² /L)	4.3-5.8/3.8-5.1	4.33±0.519	4.37±0.533	4.
Mean corpuscular volumefL	82-100	88.486,3-91.55	88.586.3-91.225	87.9{
Mean corpuscular hemoglobin	27-34	30.629.7-31.475	30.6529.775-31.4	30.1
contentpg				
Mean corpuscular hemoglobin concentrationg/L	316-354	344340-349	344340-349	344.
Red blood cell volume distribution width-SDfL	35-56	39.6538.1-41.2	39.5538-41.2	39.95
Platelet count (10 ⁹ /L)	125-350	210.03±93.190	194.18±75.912	264.
Platelet distribution widthfL	9-17	16.25±0.328	16.26±0.302	16
Mean platelet volumefL	9-13	10.02 ± 1.155	10.13 ± 1.119	9.
Thrombocytocrit	0.16-0.4	0.21±0.085	$0.19 \pm 0,975$	0.
Large platelet ratio (%)	19.7-46.7	26.00±7.691	26.71±7.420	23
Lymphocyte percentage (%)	20-50	19.84±10.130	19.47±10.450	21
Hemoglobing/L	131-172/113- 151	131.75±17.503	133.13±16.380	127
Hematocrit (%)	40-50/35-45	38.27±4.518	38.63±4.391	37
Prothrombin timesecond	11-13.7/11-	13.212.8-13.7	13.212.7-13.525	13.65
	14.3			
Activated partial thromboplastin timesecond	31.5-43.5/32- 43	39.23±6.667	40.23±6.330	35
Thrombin timesecond	16-18	17.216.125-18.1	17.1516.075-18.1	17.21
Fibrinogeng/L	2-4	5.16 ± 1.251	5.18 ± 1.286	5.
D-dimerµg/mL	0-1	1.381.05-1.7025	1.331.045-1.6725	1.61
High-sensitivity c-reactive proteinmg/L	0-3	21.7812.87-22.9	22.1818.2-23.12	18.58
Low density lipoprotein	≤3.36	1.98 ± 0.581	1.99 ± 0.553	1.
CholesteroImmol/L)				
High-density lipoprotein Cholesterolmmol/L)	0.9-2.19	1.12 ± 0.309	1.13 ± 0.326	1.
Glucosemmol/L)	3.9-5.9	6.59±2.227	6.76±2.454	6.
Procalcitoninng/mL)	5.5 5.5	0.270.1575-0.4225	0.260.14-0.46	0.3
	≤0.5	0.270.2070 0.1220		0.1
Triglyceridemmol/L)	≤1.7	1.24±0.541	1.27 ± 0.557	1.
Total cholesterolmmol/L)	≤5.2	3.60±0.744	3.65±0.746	3.
Creatinineµmol/L	53-123/44-106	76.21±26.342	80.10±26.995	63.
Potassiummmol/L)	3.5-5.3	3.84±0.467	3.88±0.490	3.
Sodiummmol/L)	137-147	136.26±4.715	135.57 ± 4.682	138
Total calciummmol/L)	2.03-2.54	2.06 ± 0.145	2.06 ± 0.151	2.
Magnesiummmol/L)	0.7-1.1	0.86 ± 0.073	0.85 ± 0.072	0.
Phosphorusmmol/L)	0.96-1.62	0.93 ± 0.261	0.91 ± 0.290	0.
Carbondioxidemmol/L)	22-29	25.30±2.509	24.83±2.437	26
Cystatinmg/L	0.63-1.25/0.6- 1.55	0.93±0.220	0.95 ± 0.221	0.
Uric acidµmol/L	208-428/155-	232.24±82.891	246.02±86.101	187

	357			
Ureammol/L)	2.9-8.2	3.753.05-5.725	3.9953.29-5.985	3.2
Total bilirubinµmol/L	≤23	11.659.075-14.75	129.525-14.95	10.
Direct bilirubinµmol/L	≤4	4.253.125-5.55	4.453.2-5.55	3.8
Total proteing/L)	65-85	67.06±6.783	67.87±6.451	64
Albuming/L)	40-55	37.04±5.436	37.94±5.515	34
Globuling/L)	20-40	30.02±4.737	29.93±4.245	30

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Table 2. Laboratory findings of COVID-19 and other patients. Parts of laboratory results are not

available for all patients.

	Normal	Over normal range	Below normal range	Total
White blood cell	44(71%)	2(3%)	16(26%)	62
count (109/L)				
Lymphocyte count	14(23%)	(0%)	48(77%)	62
(109/L)			(22())	
Monocyte count	58(94%)	4(6%)	(0%)	62
(109/L)				
Eosinophil count	16(26%)	(0%)	46(74%)	62
(109/L)		(00)	(
Basophil count	62(100%)	(0%)	(0%)	62
(109/L)			0(1=0()	60
Neutrophil count	47(76%)	6(10%)	9(15%)	62
(109/L)	45(300()	5 (00)		60
Red cell count	45(73%)	1(2%)	16(26%)	62
(1012/L)	F7(000()	1(20/)	4(CO()	60
Mean corpuscular volumefL	57(92%)	1(2%)	4(6%)	62
	$E_{7}(0,20/)$	2(20/)	2(E0/)	62
Mean corpuscular hemoglobin	57(92%)	2(3%)	3(5%)	02
contentpg				
Mean corpuscular	52(84%)	9(15%)	1(2%)	62
hemoglobin	52(0470)	9(1570)	1(270)	02
concentrationg/L				
Red blood cell	61(98%)	0(0%)	1(2%)	62
volume distribution	01(00/0)	0(070)	1(270)	02
width-SDfL				
Platelet count	50(81%)	4(6%)	8(13%)	62
(109/L)	00(02/0)	.(0,0)	0(20,0)	-
Platelet distribution	62(100%)	(0%)	(0%)	62
widthfL		()		
Mean platelet	54(87%)	1(2%)	7(11%)	62
volumefL				
Thrombocytocrit	39(64%)	1(2%)	21(34%)	61
Large platelet ratio	51(84%)	1(2%)	9(15%)	61
(%)				
Lymphocyte	28(45%)	0(0%)	34(55%)	62
percentage (%)				
Hemoglobing/L	48(77%)	2(3%)	12(19%)	62
Hematocrit (%)	37(60%)	0(0%)	25(40%)	62

Prothrombin 52(84%) 10(16%) (0%) 62 timesecond	
Activated partial 43(70%) 16(26%) 2(3%) 61 thromboplastin -	
thromboplastin timesecond Thrombin 35(56%) 17(27%) 10(16%) 62	
timesecond Thrombin 35(56%) 17(27%) 10(16%) 62	
Thrombin35(56%)17(27%)10(16%)62	
Fibrinogeng/L 10(16%) 51(82%) 1(2%) 62	
$D-dimer\mu g/mL 14(23\%) 48(77\%) (0\%) 62$	
High-sensitivity c- 1(2%) 50(98%) (0%) 51	
reactive	
proteinmg/L	
Low density $46(100\%)$ (0%) (0%) 46	
lipoprotein	
cholesterolmmol/L)	
High-density 16(29%) 38(68%) 2(4%) 56	
cholesterolmmol/L)	
Glucosemmol/L) 41(82%) 9(18%) (0%) 50	
Procalcitoninng/mL) 36(78%) 1(2%) 9(20%) 46	
-	
Triglyceridemmol/L 36(78%) 10(22%) (0%) 46	
)	
Total 45(98%) 1(2%) (0%) 46	
cholesteroImmol/L)	
Creatinineµmol/L 45(87%) 5(10%) 2(4%) 52	
Uric acidµmol/L 32(62%) 4(8%) 16(31%) 52	
Potassiummmol/L) 37(76%) (0%) 12(24%) 49	
Sodiummmol/L) 17(35%) (0%) 32(65%) 49	
Total 28(61%) (0%) 18(39%) 46	
calciummmol/L)	
Magnesiummmol/L) 22(100%) (0%) (0%) 22	
Phosphorusmmol/L) 7(32%) 1(5%) 14(64%) 22	
Carbondioxidemmo 45(90%) 1(2%) 4(8%) 50	
I/L)	
Cystatinmg/L 43(86%) 5(10%) 2(4%) 50 Use served (1) 20(70%) 4(0%) 50	
Ureammol/L) 39(78%) 4(8%) 7(14%) 50 Table 47(00%) 5(10%) 50	
Total 47(90%) 5(10%) (0%) 52	
bilirubinµmol/L	
Direct 22(42%) 30(58%) (0%) 52	
bilirubin μ mol/L	
Total proteing/L) 30(58%) (0%) 22(42%) 52 Albuming(l) 21(40%) (0%) 21(60%) 52	
Albuming/L)21(40%)(0%)31(60%)52Globuling/L)50(96%)2(4%)(0%)52	
Globuling/L) 50(96%) 2(4%) (0%) 52	

Table 3 Laboratory findings of COVID-19 patients classified according to normal range.

Treatments and	All patientsn=80	Novel coronavirus	Other patientsn=18
outcomes		infectionn=62	
Treatment			
Antiviral treatment	75(94%)	58(94%)	17(94%)
Antibiotics	70(88%)	54(87%)	16(89%)
Immunoglobulin	21(26%)	16(26%)	5(28%)
Oxygen therapy	14(18%)	8(13%)	6(33%)
Traditional Chinese medicine	11(14%)	9(15%)	2(11%)
Probiotics tablet	5(6%)	4(6%)	1(6%)
Corticosteroid	1(1%)	1(2%)	0(0%)
Prognosis			
Hospital admission	80(100%)	62(100%)	18(100%)
Discharge	28(35%)	22(35%)	6(33%)
Admission to intensive	3(4%)	3(5%)	0(0%)
care unit			
Death	0(0%)	0(0%)	0(0%)

Table 4. Treatments and outcomes in COVID-19 patients.

Variable	Adjusted OR	95%CI	P value
Gender (male vs female)	2.599	(0.520,12.988)	0.245
Time from symptom occurrence to admission (days)	0.779	(0.609,0.995)	0.046
Hypersensitive C-reactive protein (mg/L)	1.133	(1.017,1.264)	0.024
Albumin (g/L)	1.297	(1.060,1.586)	0.011
Age (years)	1.107	(1.030,1.191)	0.006

Table 5. Multivariable logistic regression model for determining risk factors for positive result of the

viral nucleic acid assay.