COVID-19 patients with hypertension under potential risk of worsened organ injuries

Fei Xia (53042940@qq.com)
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University) https://orcid.org/0000-0001-5469-6785
Mingwei Zhang
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University) https://orcid.org/0000-0001-7673-6418
Bo Cui
Renmin Hospital of Wuhan University https://orcid.org/0000-0002-4510-030X
Wei An
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Min Chen
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University) https://orcid.org/0000-0002-2149-5933
Ping Yang
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Tao Qin
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Xiaoyang Zhou
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Yaling Liao
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Xin Xu
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Shiguo Liu
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Kuangyu Li
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Qin Zhou
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Keke Wang
the First Affiliated Hospital of China Medical University
Guangxu Hu
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Ming Du
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Songrui Chen
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Jianjun Zhang
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Yafang Zhang
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Wei Wei
the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University)
Ming Xiang

Huazhong University of Science and Technology https://orcid.org/0000-0002-3048-3897

Research Article

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Abstract

COVID -19 has rapidly spread from Wuhan to worldwide, and now has become a global health concern. Hypertension is the most common chronic illness in COVID-19, while the influence on those patients have not been well described. In this retrospective study, 82 confirmed patients with COVID-19 were enrolled, with epidemiological, demographic, clinical, laboratory, radiological, and therapies data analyzed and compared between COVID-19 patients with (29 cases) or without (53 cases) hypertension. Of all 82 patients with COVID-19, the median age of all patients was 60.5 years, including 49 females (59.8%) and 33 (40.2%) males. Hypertension (31[28.2%]) was the most chronic illness, followed by diabetes (16 [19.5%]) and cardiovascular disease (15 [18.3%]). Common symptoms included fatigue (55[67.1%]), dry cough (46 [56.1%]) and fever (≥37.3℃ (46 [56.1%]). The median time from illness onset to positive outcomes of RT-PCR analysis were 13.0 days, ranging from 3-25 days. In hypertension group, 6 (20.7%) patients died compared to 5 (9.4%) died in non-hypertension group. More hypertension patients with COVID-19 (8 [27.6%]) had at least one coexisting disease than those of non-hypertension patients (2 [3.8%]) (P=0.002). Compared with non-hypertension patients, higher levels of neutrophil counts, serum amyloid A, C-reactive protein, and NT-proBNP were observed in hypertension group, whereas levels of lymphocyte count and eGFR were decreased. Dynamic observations displayed more significant and worsened outcomes in hypertension group after hospital admission. COVID-19 patients with hypertension take more risks of severe inflammatory reactions, worsened internal organ injuries, and deteriorated progress.

Introduction

In December 2019, several cases of acute pneumonia illness have been reported in Wuhan, Hubei province, China [1]. The pneumonia cases of unknown aetiology were firstly ascribed to originate from a large seafood and animal market in Wuhan [2]. The disease has rapidly and widely spread in China and become pandemic, causing an outbreak of acute infectious pneumonia [3]. By Jan 7, 2020, a novel coronavirus was isolated by Chinese scientists from the throat swab sample of a patient, and was finally named SARS-CoV–2, from these patients with pneumonia, which was later designated coronavirus disease 2019 (COVID–19) by WHO in Feb 12. SARS-CoV–2 was categorized as the beta coronavirus 2β lineage, sharing 79.5% sequence identity to SARS-Co and 96% identical at the whole genome level to a bat coronavirus [4]. As of May 14, 2020, there have been more than 80,000 cases confirmed and 4633 deaths have resulted from COVID–19 in mainland china. Globally, the virus has spread in almost 200 countries and areas with more than 4,250,000 infected cases reported.

To date, the person-to-person transmission is robustly supported by more evidences on COVID–19 [5, 6]. SARS-CoV–2 is evidenced to infect multiple systems and organs through spike binding angiotensin converting enzyme II (ACE2) with higher affinity than SARS-CoV spike. As the functional receptor, ACE2 protein is reported to abundantly expressed in humans in the epithelia of the lung and small intestine[7]. Thereby, clinical spectrum of SARS-CoV–2 infection appears to be wide, including fever, cough, dyspnea, decreased leukocyte counts or white blood cell counts, mild upper respiratory tract illness, and severe viral pneumonia[8]. System or organ malfunction, including shock, acute respiratory distress syndrome (ARDS), acute cardiac injury, and acute kidney injury and death can also occur in severe cases[9]. Notably, hypertension is the most coexisting chronic illness in Covid–19 patients as reported[2, 8, 10], implying population with hypertension under risks of SARS-CoV–2 infection due to the imbalance of ACE system. In this study, we aim to describe the epidemiology, clinical features, and medicine therapies of hospitalized patients with COVID–19, and to further compare the available data between hypertension and non-hypertension patients admitted to the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University).

Methods

Study design and participants

This retrospective, single-center study included patients from Dec 31, 2019 to Feb 01, 2020, and the final date of follow-up was February 08, 2020, at the third people's hospital of Hubei province, in Wuhan, China. The third people's hospital of Hubei province is a hospital with over 1,500-bed accommodation, designated for COVID–19 treatment. Diagnosis of COVID–19 was based on current New Coronavirus Pneumonia Prevention and Control Program (6th edition, in Chinese) released by the National Health Commission of China [11] and was indicated by suspected symptoms, chest CT results and SARS-CoV–2 positivity by use of quantitative RT-PCR. On admission, throat-swab specimens from all patients were collected and kept in viral transport medium. Briefly, the Chinese Center for Disease Control and Prevention, the Chinese Academy of Medical Science, the Academy of Military Medical Sciences, and the Wuhan Institute of Virology, Chinese Academy of Sciences conducted SARS-CoV–2 detection in respiratory specimens by real-time RT-PCR methods or next-generation sequencing. Other respiratory viruses including influenza A virus (H1N1, H3N2, H7N9), influenza B virus, respiratory syncytial virus,

parainfluenza virus, adenovirus, SARS coronavirus (SARS-CoV), and MERS coronavirus (MERS-CoV) were also examined with real-time RT-PCR. Sputum or endotracheal aspirates were also inspected for identification of potential causative bacteria or fungi. Chest computed tomographic (CT) scans were carried out twice at least for each patient.

The clinical classifications of COVID−19 patients admitted to hospital compromised mild cases, moderate cases, and sever cases. These definitions are elucidated as follows: Mild cases: the clinical symptoms are mild and no pneumonia manifestation can be found in imaging; Moderate cases: Patients have symptoms like fever and respiratory tract symptoms, etc. and pneumonia manifestation can be seen in imaging; Severe cases: Meeting any of the following: Respiratory distress, respiratory rates ≥30 breaths/min; The SpO2 ≤93% at a rest state; PaO2/FIO2 ratio ≤300; Patients with >50% lesions progression within 24 to 48 hours in pulmonary imaging should be treated as severe cases. The research protocol was reviewed and approved by the Ethics Committee at the third people's hospital of Hubei province (202004). All research procedures meet the tenets of the Declaration of Helsinki.

Data collection

82 hospitalized patients with COVID−19 were included in this retrospective study, and 29 of them were hypertension individuals. Epidemiological, demographic, clinical, laboratory, X ray and chest computed tomographic (CT) scans, treatments, and outcome data were extracted from electronic medical records with data collection forms. Eight researchers, including physicians and clinical pharmacist, reviewed the data collection forms to triple check the data independently. COVID−19 patients were categorized into two groups according to the presence and absence of hypertension. Hypertension is defined as a clinic systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg without the use of anti-hypertensive medications. Subjects with a BP <140/90 mmHg but having hypertensive history and currently are taking anti-hypertensive medication should also be diagnosed as hypertensives[12].

Statistical Analysis

Categorical variables were described with frequencies and percentages. Continuous variables were described with the mean (SD), median, and interquartile range (IQR) values. Comparisons of quantitative variables between groups were performed using Wilcoxon rank sum test. Categorical variables were expressed as numbers (%) and compared by χ^2 test or Fisher's exact test between the first group and the second group. A two-sided p-value less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS (version 21.0).

Results

Baseline characteristics of hospitalized COVID−19 patients on admission

In this retrospective study, we included 82 patients hospitalized in the third people's hospital of Hubei province before Feb 08, 2020. In total, the median age was 60.5 years, and 49 (59.8%) patients were women (Table 1). The median time from first symptom to hospital admission was 7.0 days. Hypertension (29 [35.4%]) was the most coexisting chronic illness, followed by diabetes (16 [19.5%]) and cardiovascular disease (15 [18.3%]). Fatigue (55 [67.1%]), dry cough (46 [56.1%]), and fever (≥37.3°C) (46 [56.1%]) were the most common symptoms. The median time from illness onset to positive outcomes of RT-PCR analysis were 13.0 days, ranging from 3−25 days. A significant difference in clinical types on admission was observed (P<0.001) between the two groups. There were 6 (20.7%) death cases in hypertension group and 5 (9.4%) in non-hypertension group. Of 29 COVID−19 patients with hypertension, 8 (27.6%) had more than one coexisting disease, which was significantly higher than that in non-hypertension group (P = 0.002).

Laboratory outcomes in patients with or without hypertension

Major laboratory markers were recorded from hospital admission for all patients (Table 2). In blood routine analysis, there were no significant differences between hypertension and non-hypertension group. However, higher levels of white blood cell (4.9×10^9/L), neutrophil counts (3.9×10^9/L), and neutrophil percentage (79.3%) were simultaneously found in hypertension group, whereas lower levels of lymphocyte counts (0.72×10^9/L) and lymphocyte percentage (12.5%) were observed. Moreover, the median of SAA reached 630.1mg/L, which was approximately double as that (373.5 mg/L) in non-hypertension group. Similarly, the median of CRP (79.2 mg/L) also exceeded that of non-hypertension group (57.8mg/L). In blood chemistry assays, no significant changes were found in AST (32.4U/L vs 34.1U/L), ALT (23.0U/L vs 26.3 U/L), and GGT (29.0 U/L vs 30.3U/L) between the two groups, and all medians were within the normal

ranges. As for renal injury, eGFR in hypertension group with COVID−19 was significantly decreased compared with that of non-hypertension group (77.0 mL/min/1.73m^2 vs 113.0 mL/min/1.73m^2) (P = 0.017), while higher levels of urea nitrogen (4.7 mmol/L vs 4.2 mmol/L) and serum creatinine (70.5 μmol/L vs 57.0 μmol/L) were observed in hypertension group. There were also elevated levels of NT-proBNP (166 ng/L vs 26 ng/L), lactate dehydrogenase (263.0U/L vs 240.0mU/L), and creatine kinase (143.0 U/L vs 64.0 U/L), with a significant increase in NT-proBNP in hypertension group with COVID−19.

Outcomes of image manifestations

Of all 82 non-medical COVID−19 patients on admission (Table 3), 14 (17.1%) patients showed unilateral pneumonia and 64 (78.3%) ones developed bilateral pneumonia. 28 (34.2%) patients showed patchy shadows while 22 (26.8%) patients showed multiple patchy shadows. 22 (26.8%) patients also displayed ground glass opacity. In addition, hydrothorax occurred in 9 (11.0%) patients.

Organ injuries and main treatment

On admission, common complications among 82 patients contained ARDS (9 [11.0%]), sepsis (3 [3.7%]), acute renal injury (1 [1.2%]), and acute respiratory injury (1 [1.2%]) (Table 4). Though the differences between the two group failed to reach statistical significance on hospital admission, the lab outcomes suggest that patients with hypertension took more risks of organ injuries in kidney, heart, and lung. For COVID−19 treatment, 63 (76.8%) patients received antiviral therapy, including oseltamivir (56 [50.9%]), arbidol (46 [41.8%]), lopinavir/ritonavir (42 [38.2%]), and ganciclovir (28 [25.4%]). More hypertension cases (6 [20.7%]) were applied with three kinds of antiviral chemicals (P = 0.040). Many patients applied glucocorticoid therapy (73 [89.0%]) and antibacterial therapy (55 [67.1]), including carbapenems (22 [20.0%]), quinolones (33 [30.0%]), and cephalosporin (14 [12.7%]). 55(67.1%) patients received both antiviral and antibacterial therapies. Oxygen therapy (24 [29.3%]) and immune globulin γ treatment (42 [51.3%]) were also applied.

Dynamic characteristics of laboratory parameters in COVID−19 patients with or without hypertension

The dynamic laboratory features of COVID−19 patients, including 8 clinical parameters related to hematology, infection, coagulation function, and internal organ injury, were traced from hospital admission to day 20 in hospital at a 2-day intervals, on the basis of no significant difference observed at hospital admission between the two groups (Table.1). As shown in Figure 1A and B, white blood cell and neutrophil counts were higher in almost whole duration of hospitalization in hypertension group than those in non-hypertension group. Most patients had notable lymphopenia, and there was a 2-day delayed of an increase in lymphocyte count to normal range compared to non-hypertension group (Figure 1C). Both of CRP and SAA were higher in hypertension group till day 10 after admission (Figure 1D and E). eGFR of hypertension patients were markedly lower than that in non-hypertension, keeping a slowly increase but a slight decrease from its peak on day 8; by contrast, there was a about 30 mL/min/1.73m^2 reduction in eGFR of non-hypertension group from day8 to 14 (Figure 1F). Creatine kinase of hypertension group was higher on admission, increasing to approximately 400U/L on day 2, and then decreased (Figure 1H). Similarly, the level of D-Dimer kept an increase till day 4 after admission or day 11 from the onset of illness in hypertension group, and then went declining after day 6 (Figure 1G).

Discussion

Nowadays, there is a globally confirmation that SARS-CoV−2 has the stronger capacity to transmit from human to human. With its spike of 10- to 20-fold higher affinity binding to ACE2 than that of SARS-CoV[13], COVID−19 rapidly spread from a single city to a pandemic health challenge in just 70 days. In this study, we try to gain more useful understandings about the epidemiology, clinical characteristics, and treatment of COVID−19 patients, particularly the potentially different changings in hypertension population.

In this study, of 82 cases of COVID−19 patients, there were 11 death cases (13.4%), which was similar to contemporary one in Jinyintan Hospital in Wuhan in January[10]. By contrast, other two highly pathogenic coronaviruses, SARS-CoV (severe acute respiratory syndrome) and MERS-CoV (Middle East respiratory syndrome) result in severe respiratory syndrome, with reported mortality at more than 10% and more than 35% in humans[14, 15]. Nevertheless, national data showed that the case-fatality had dropped to 2.3% by Feb 11[5], suggesting that SARS-CoV−2 seemed more insidious at early phage of outbreak in Wuhan[16]. Notably, median time from illness onset to RT-PCR positive outcomes was 13 days inall infected patients, with the longest duration of 25 days, suggesting more detections needed for suspected patients. This study included 49 females and 33males, differing from other local reports[8−10]. This difference in sex ratio could

be attributed to varying population around hospital. It was noticed that sex ratio was 51.4: 48.6 on national scale of 44672 cases by Feb 11; of note, sex mortality was 63.8: 36.2[17], which may be attributable to X chromosome and sex hormones, acting as an important role in immune reactions[18].

Clearly, approximately 30% COVID–19 patients were hypertension population as the most common chronic illness reported[8, 9, 19]. Renin-angiotensin II-aldosterone axis has been traditionally recognized as a key regulator of blood pressure and fluid homeostasis, with angII levels regulated by angiotensin-converting-enzyme (ACE). The balance between ACE1 and ACE2 is crucial for controlling level of AngII. In present studies, ACE2 was identified as the receptor of SARS-CoV–2 for virus entry, identical to SARS-CoV transmission via the spike (S) glycoproteins–ACE2 binding pathway[20–23]. Importantly, SARS-CoV infections and the spike protein of the SARS-CoV were reported to reduce ACE2 expression[24]. Thereby, there could be a possibility that SARS-CoV–2 infection also decreases the level of ACE2 in potential target organs, and disrupts the balance between ACE1 and ACE2, which further put hypertension population into a deteriorated progress with worsen organ injuries. In this study, we found higher percentages of severe degree on admission and augmented mortality during treatment in COVID–19 patients with hypertension. Laboratory outcomes also provided evidences. For dynamic outcomes, in hypertension group, white blood cell and neutrophil counts continued to increase till 18 days at least; SAA and CRP remained to increase till approximately 10 days after admission. By contrast, lymphocyte counts continued to decrease till 10 days after admission, while an increase to normal range was delayed by two day compared to that of non-hypertension group.

More laboratory evidences were found in COVID–19 patients with hypertension. There were elevated levels of serum urea nitrogen, serum creatinine, lactate dehydrogenase, creatine kinase, NT-proBNP, and markedly reduced eGFR observed on hospital admission. These changing parameters suggest that internal organs expressing abundant ACE2 protein, such as lung, kidney, and heart[7, 25], are more susceptible to be under attack from SARS-CoV–2. As a consequence of those worsened changings, 6 (20.7%) cases of hypertension group died during treatment, compared to 5 (9.4%) of patients without hypertension. Moreover, if there were prolonged duration from illness onset to hospital admission, more organ failures or death would be found in COVID–19 patients with hypertension. These varying findings indicated that patients with hypertension had to face with higher risks of worsened outcomes not only from changed blood routine related to cytokine storm, but also from a dwindled protection of decreased ACE2 level[26, 27].

In this study, individualized treatments were applied depending on disease severity. Though there are still no specific drugs or vaccines for prevention and treatment to COVID–19[28], exploring therapies based on marketed chemical drugs still remains huge benefits to the clinical treatment of infected patients.

This study has several limitations. First, 110 patients with confirmed COVID–19 were included, and 82 of them were applied for further analysis. It would be better accumulate more patients to deeply understand the role of hypertension in the progress of COVID–19. Second, due to the retrospective study design, some detailed information was unavailable, particularly regarding time from illness onset to signs and symptoms in electronic medical records. Third, positive rate of SARS-CoV–2 RNA detection in throatswabs was relative low, and antibody assay not available to assist the diagnose by the end of January, leading to suspected but unconfirmed cases ruled out in the analyses. However, this investigation revealed the hypertension factor influencing the progress of COVID–19, and more work are needed to describe the clinical features of this special sub-population in detail.

Declarations

This study was carried out in the third people's hospital of Hubei province (Hubei No. 3 People's Hospital of Jianghan University), located in 26 Zhongshan Road, Qiaokou area, Wuhan city, Hubei province. Fei Xia was responsible for this study. This study was aim to describe the epidemiological and clinical characteristics of COVID-19 patients. The research protocol was reviewed and approved by the Ethics Committee at the third people's hospital of Hubei province (202004). In this this retrospective, single-center study, all data were extracted from electronic medical records with data collection forms. All cases had been finished at the beginning of this study. This study did not interfere in any diagnosis or treatment of cases. Therefore, there were no information about the informed consent for the included cases in the third people's hospital of Hubei province. Further, all privacy of patients was respected and protected.

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Author contributions

Fei Xia and Ming Xiang 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.

Fei Xia, Mingwei Zhang, and Bo Cui contributed equally and share first authorship.

Concept and design: Fei Xia, Mingwei Zhang, Ming Xiang.

Acquisition, analysis, or interpretation of data: Fei Xia, Bo Cui, Wei An, Min Chen, Ping Yang, Xiaoyang Zhou, Yaling Liao, Xin Xu, Kuangyu Liu, Qin Zhou, Keke Wang, Guangxu Hu, Ming Du, Songrui Chen.

Drafting of the manuscript: Fei Xia.

Critical revision of the manuscript for important intellectual content: Fei Xia, Mingwei Zhang, Bo Cui, Xiaoyang Zhou, Tao Qin, Ming Xiang.

Statistical analysis: Fei Xia, Bo Cui, Wei An, Min Chen.

Administrative, technical, or material support: Mingwei Zhang, Shiguo Liu, Yafang Zhang. Supervision: Jianjun Zhang, Wei Wei, Ming Xiang.

All authors: read, revised, and approved the final manuscript. All authors agree to this study and to this version of the manuscript as submitted to Scientific Reports, agree to their contributions and order of attribution to this study.

Disclosure statements

The authors have declared that no conflict of interest exists.

OCRID

Fei Xia: http://orcid.org/ 0000–0001–5469–6785

Mingwei Zhang: http://orcid.org/ 0000–0001–7673–6418

Bo Cui: http://orcid.org/ 0000–0002–4510–030X

Ming Xiang: http://orcid.org/ 0000–0002–3048–3897

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Tables

Table1 Demographics and clinical characteristics of patients with COVID-19 on admission

	All Patients (n=82)	Hypertension group (n=29)	non-hypertension group (n=53)	*χ2/Z*	*P*
Age, years	60.5 (46.8-69.0)	66.0 (56.5-69.0)	57.0 (40-68.5)	-10.61	<0.001
Female age	60.0 (44.0-69.0)	66.5 (57.8-71.5)	63.5 (56.8-70.3)	-14.798	<0.001
male age	61.0 (46.5-69)	63.0 (53.0-69.0)	57.5 (44.0-71.5)	-0.967	0.334
Sex					
Female	49 (59.8)	14 (48.3)	19 (35.8)	1.204	0.273
Male	33 (40.2)	15 (51.7)	34 (64.2)		
Clinical types on admission					
Mild	3 (3.7)	0 (0.0)	3 (5.7)	43.931	<0.001
Moderate	68 (82.9)	23 (79.3)	45 (84.9)		
Severe	11 (13.4)	6 (20.1)	5 (9.4)		
Illness onset to hospital admission, days	7.0 (4.0-10.0)	7 (5.5-10)	7 (4-10)	-1.129	0.259
Chronic medical illness					
Hypertension	29 (35.4)	29 (100.0)	0 (0)	–	–
Cardiovascular disease	15 (18.3)	9 (31.0)	6 (11.3)	4.814	0.028
Diabetes	16 (19.5)	8 (27.6)	8 (15.1)	1.862	0.172
COPD	7 (8.5)	4 (13.8)	3 (5.7)	1.568	0.210
Malignancy	1 (1.2)	0 (0.0)	1 (1.9)	0.547	0.459
Digestive system disease	3 (3.7)	2 (6.9)	1 (1.9)	1.318	0.251
Cerebrovascular disease	2 (2.4)	2 (6.9)	0 (0.0)	3.747	0.054
Nervous system disease	3 (3.7)	1 (3.4)	2 (3.8)	0.006	0.941
Chronic liver disease	3 (3.7)	3 (10.3)	0 (0.0)	5.622	0.018
More than one disesase	10 (12.2)	8 (27.6)	2 (3.8)	9.926	0.002
Signs and symptoms					
Fever (≥37.3℃)	46 (56.1)	13 (44.8)	33 (62.2)	2.314	0.128
≥38℃	28 (34.1)	6 (20.7)	22 (41.5)	3.361	0.057
Fatigue	55 (67.1)	16 (55.2)	39 (73.6)	2.877	0.090
Dry cough	46 (56.1)	13 (44.8)	33 (62.3)	2.314	0.128
Shortness of breath	23 (28.0)	7 (24.1)	16 (30.2)	0.34	0.560
Diarrhoea	15 (18.3)	7 (24.1)	8 (15.1)	1.026	0.311
Anorexia	10 (12.2)	1 (3.4)	9 (17.0)	3.167	0.075

Myalgia	9 (15.1)	3 (10.3)	6 (11.3)	0.018	0.893
Expectoration	20 (24.3)	10 (34.4)	10 (18.9)	2.478	0.115
Pharyngalgia	4 (4.9)	2 (6.9)	2 (3.8)	0.389	0.533
Nausea or vomiting	7 (8.5)	2 (6.9)	5 (9.4)	0.155	0.694
Dyspnea	5 (6.1)	2 (6.9)	3 (5.7)	0.049	0.824
More than three signs and symptoms	40 (48.9)	17 (58.6)	23 (434)	1.739	0.187
Time from illness onset to RT-PCR positive outcomes, days	13 (6.0-19.0)	13.0 (6.0-20.0)	12.5 (6.0-15.5)	-0.372	0.743
Death outcomes	11 (13.4)	6 (20.7)	5 (9.4)	2.044	0.153
Female	5 (4.5)	3 (10.3)	2 (3.8)	0.100	0.752
Male	6 (5.4)	3 (10.3)	3 (5.7)		
Time from onset to hospital admission, days	7.0 (5.0-10.0)	7.0 (5.0-8.0)	10 (5.5-20)	-0.746	0.456
Time from admission to death, days	6.0 (4.0-12.0)	7.5 (3.0-12.5)	4.0 (2.5-9.5)	-0.737	0.461
Time from onset to death, days	13.0(10.5-19.5)	13.5 (10.5-19.5)	11.0 (10.5-28.5)	-0.184	0.854

Abbreviations: COVID-19, Coronavirus Disease 2019; IQR, interquartile range; COPD, Chronic Obstructive Pulmonary Disease. Data are median (IQR) or n/N (%), where N is the total number of patients with available data. *p* values comparing between patient with or without hypertension cases are from χ^2, Fisher's exact test, or Wilcoxon rank sum test.

Table 2 Laboratory findings of patients infected with COVID-19 on admission

	Normal range	All patients (n=82)	Hypertension group (n=29)	Non-hypertension group (n=53)	*Z*	*P*
Blood routine						
WBC, $\times 10^9$/L	3.5-9.5	4.7 (3.5-6.7)	4.9 (3.7-7.4)	4.5 (3.3-6.6)	-1.093	0.274
Neutrophil counts, $\times 10^9$/L	1.8-6.3	3.2 (2.2-5.2)	3.9 (2.7-6.3)	3.1 (2.2-4.9)	-1.544	0.122
N (%),	40-75	76.0 (63.8-84.3)	79.3 (69.2-85.7)	71.6 (62.9-83.4)	-1.852	0.064
Lymphocyte counts, $\times 10^9$/L	1.1-3.2	0.8 (0.6-1.1)	0.72 (0.59-0.10)	0.81 (0.56-1.26)	-0.652	0.515
L (%)	20-50	14.1 (9.1-19.2)	12.5 (8.6-17.3)	16.8 (12.2-21.3)	-1.625	0.098
Platelets, $\times 10^9$/L	125–350	192.0 (127.0-225.0)	195 (165-293)	188 (130-225)	-0.492	0.623
Haemoglobin, g/L	130·0–175	127.0 (119.0-135.0)	129.5 (114.8-137)	126.5 (120-134.8)	-0.067	0.947
Infection-related biomarkers						
PCT, ng/mL	0.04-0.25	0.1 (0.0-0.2)	0.07 (0.04-0.18)	0.05(0.04-0.1)	-1.679	0.093
SAA, mg/L	0.1-10	501.0 (111.3-102.0)	630.1 (185.3-1010.5)	373.5 (48.8-777.5)	-1.509	0.131
CRP, mg/L	0-5	67.5 (27.2-102.0)	79.2 (33.5-129.1)	57.8 (20.4-83.1)	-1.802	0.072
ESR, mm/h	0-20	47.8 (23.3-80.5)	50.5 (24.9-88.0)	47.8 (19-70)	-0.723	0.470
Blood biochemistry						
AST, U/L	8-40	33.6 (26.2-51.2)	32.4 (24.2-49.8)	34.1 (26.8-57.0)	-0.982	0.326
ALT, U/L	5-35	26.1(17.0-40.0)	23.0 (18.1-42.0)	26.3 (14.7-39.4)	-0.630	0.529
GGT, U/L	0-50	30.2 (18.0-50.6)	29.0 (22.6-48.3)	30.3 (16.2-55.4)	-0.583	0.560
Serum urea nitrogen, mmol/L	3.5-7.2	4.4 (3.2-4.5)	4.7 (3.4-5.9)	4.2 (3.2-4.9)	-1.324	0.185
Serum creatinine, μmol/L	44-120	60.0 (49.0-79.3)	70.5 (51.8-80.8)	57.0 (49.0-79)	-1.473	0.142
eGFR, mL/min/1.73m^2	>90	102.0 (84.0-119.3)	77.0 (35.3-100.7)	113.0 (91.9-125.9)	-2.387	0.017
Lactate dehydrogenase, U/L	120–250	249.5 (200.8-353.8)	263.0 (235-384.5)	240.0 (193.0-318.5)	-1.042	0.297
Creatine kinase, U/L	50–310	97.5 (55.7-179.8)	143.0 (84.0-220.0)	64.0 (50.0-154.0)	-1.732	0.083
NT-proBNP,ng/L	25-500	115 (26-372)	166 (70-1293)	26 (20-120.5)	-10.252	<0.001
K, mmol/L	3.5-5.5	3.7 (3.4-4.0)	3.6 (3.5-4.1)	3.7 (3.5-3.9)	-0.635	0.525
Ca, mmol/L	2.0-2.6	2.1 (2.0-2.2)	2.1 (2.0-2.1)	2.1 (2.0-2.2)	-0.112	0.911
Albumin, g/L;	40-55	39.0 (35.8-43.3)	36.4 (34.6-39.9)	40.0 (36.6-44.6)	-2.218	0.026

Glucose, mmol/L	3.9-6.1	7.54 (6.26-9.23)	7.8 (7.4-9.4)	6.5 (5.5-10.0)	-1.583	0.113
Total bilirubin,μmol/L	3.4-20.5	9.9 (8.4-13.5)	9.8 (8.6-12.2)	10.0 (8.0-13.8)	-0.159	0.874
Direct bilirubin,μmol/L	0-6	3.7 (3.2-4.7)	3.5 (3.2-4.7)	3.9 (3.1-4.8)	-0.817	0.413
Total bile acid, μmol/L	0-12	4.4 (2.5-6.9)	3.8 (2.5-6.3)	5.3 (2.5-9.1)	-1.141	0.254
Coagulation function						
D-Dimer, μg/mL	0·0–1.5	0.5 (0.3-1.1)	0.5 (0.3-1.0)	0.5 (0.3-1.4)	-0.157	0.875
Prothrombin time, S	9-14	10.4 (7.9-11.1)	10.6 (9.7-11.1)	10.8 (10.3-11.5)	-1.109	0.267
Activated partial thromboplastin time, S	20-40	27.4 (11.7-31.9)	29.7 (24.7-39.2)	28.2 (26.9-32.0)	-0.802	0.422
Fibrin(-ogen) degradation products, mg/L	0-5	3.7 (2.5-5.7)	3.7 (3.0-5.5)	3.7 (1.7-8.7)	-0.217	0.828

Abbreviations: COVID-19, Coronavirus Disease 2019; IQR, interquartile range; WBC , While blood cell; PCT, procalcitonin; SAA, serum amyloid A; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; GGT, Glutamine transpeptidase; eGFR, Estimated glomerular filtration rate; NT-proBNP, N-terminal pro-brain natriuretic peptide. Data are expressed as median (IQR) or n/N (%), where N is the total number of patients with available data. *p* values comparing between patient with or without hypertension cases are from χ^2, Fisher's exact test, or Wilcoxon rank sum test.

Table 3 Chest X-ray and CT findings of COVID-19 patients on admission

	All patients (n=82)	Hypertension group (n=29)	Non-hypertension group (n=53)	*χ2*	*P*
Bilateral pneumonia	64 (78.3)	23 (79.3)	41 (77.4)	0.042	0.838
Unilateral pneumonia	14 (17.1)	4 (13.8)	10 (18.9)	0.341	0.559
Patchy shadows	28 (34.2)	9 (31.0)	19 (35.8)	0.249	0.618
Multiple patchy shadows	22 (26.8)	11 (37.9)	11 (20.7)	2.871	0.093
Ground glass opacity	4 (4.9)	1 (3.4)	3 (5.7)	0.198	0.657
Hydrothorax	9 (11.0)	3 (10.3)	6 (11.3)	0.018	0.892

Abbreviations: COVID-19, Coronavirus Disease 2019; CT□computed tomography. Data ar expressed as median (IQR) or n/N (%), where N is the total number of patients with available data. *p* values comparing between patient with or without hypertension cases are from χ^2, or Fisher's exact tes

Table 4 Complications and treatments of COVID-19 patients

	All patients (n=82)	Hypertension group (n=29)	Non-hypertension group (n=53)	*χ2*	*P*
Complications					
ARDS	9 (11.0)	5 (17.2)	4 (7.5)	1.803	0.179
Sepsis	3 (3.7)	2 (6.9)	1 (1.9)	1.402	0.236
Acute renal injury	1 (1.2)	1 (3.4)	0	1.893	0.169
Acute respiratory injury	1 (1.2)	1 (3.4)	0	1.893	0.169
Treatment					
Antiviral therapy	63 (76.8)	22 (75.9)	41 (77.4)	0.024	0.878
Two antiviral chemicals	28 (34.1)	11 (37.9)	17 (32.1)	0.286	0.593
Three antiviral chemicals	16 (19.5)	6 (20.7)	10 (18.9)	0.040	0.842
Glucocorticoid therapy	73 (89.0)	26 (89.7)	47 (88.7)	0.018	0.892
Antibacterial therapy	55 (67.1)	18 (62.7)	37 (69.8)	0.509	0.476
Anti-virus and antibacterial treatment	34 (41.5)	14 (48.3)	20 (37.7)	0.858	0.354
Oxygen therapy	24 (29.3)	11 37.9)	13 (24.5)	1.626	0.202
Immune globulin γ treatment	42 (51.3)	15 (51.7)	27 (50.9)	0.005	0.946

Abbreviations: COVID-19, Coronavirus Disease 2019; ARDS, Acute respiratory distress syndrome; Data are expressed as n/N (%), where N is the total number of patients with available data. *p* values comparing between patient with or without hypertension cases are from χ^2, or Fisher's exact test.

Figures

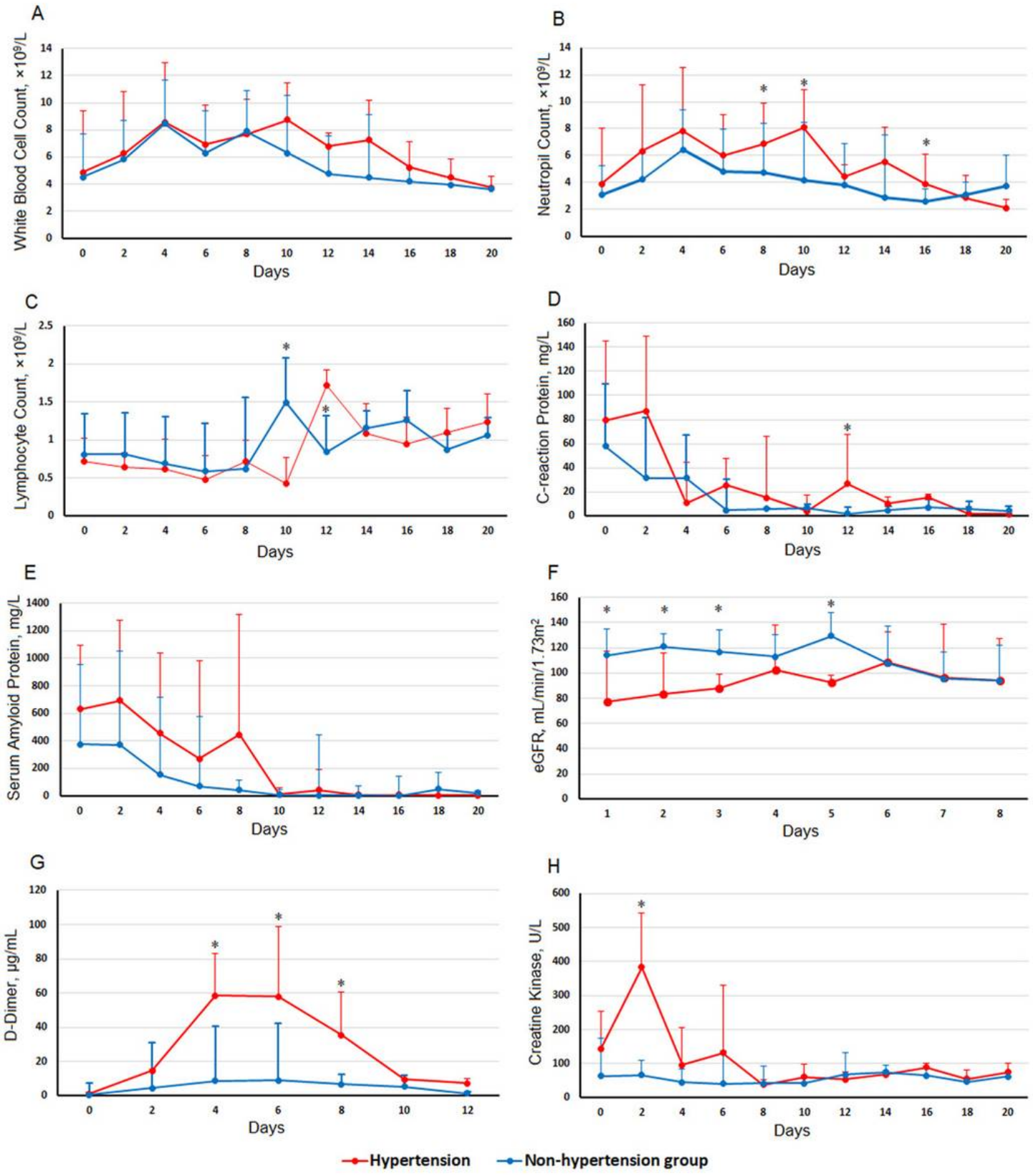

Figure 1

Timeline charts in laboratory markers from hospital admission with COVID-19. Figure shows dynamic change in white blood cell counts (A), neutrophil counts (B), lymphocyte counts (C), C-creation protein (D), serum amyloid A (E), eGFR (F), D-Dimer (G), and creatine kinase (H). For eGFR and D-Dimer, the continuous data with 2-day intervals available were 14 days and 12 days, respectively. *P < 0.05 for hypertension group vs non-hypertension group