A systematic review of etiology, epidemiology, clinical manifestations, image findings, and medication of 2019 Corona Virus Disease-19 in Wuhan, China

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Abstract

Background: Unknown origin pneumonia has been furiously spreading since the late of December 2019, subsequently spread to approximately all provinces and areas in China and many countries, which was announced as a Public Health Emergency of International Concern by World Health Organization (WHO). The studies on 2019 Corona Virus Disease-19 (COVID-19) conducted from various fields around the world. Herein, the objective of the present study is to summarize the etiology, epidemiology, clinical manifestations, image findings, traceability analysis, and drug development of COVID-19.

Methods: The following electronic databases were searched: Cochrane Central Register of Controlled Trials (CENTRAL), PubMed, EMBASE, Cochrane Library, China National Knowledge Infrastructure (CNKI), Chinese Biomedical Literature Database, VIP Chinese Science and Technology Periodical Database, and Wanfang Data. Other relevant literature will be manually searched as a compliment. We have reviewed etiology, epidemiology, clinical manifestations, image findings, and medication from case reports and retrospective clinical studies relating to COVID-19 published since the outbreak.

Results: The coronavirus is closely related to bat coronavirus and pangolin coronavirus. Besides, the infection pathway is confirmed to be the respiratory and digestive systems. The virus indicates person-to-person transmission and some patients present asymptomatic. The elderly have a higher mortality rate. Rapid and sensitive nucleic acid testing is usually used as a basis for diagnosis. Currently, there is no specific vaccine and antiviral drug. Intervention actions such as travel bans and quarantine adopted have effectively reduced the spread of the epidemic.

Conclusion: This systemic review will provide high-quality evidence to summarize etiology, epidemiology, clinical manifestations, image findings, traceability analysis, drug development in patients with COVID-19.

Abbreviations: ACE2 = angiotensin-converting enzyme II, AAK1 = AP2 related protein kinase 1, ACEI = angiotensin-converting enzyme inhibitor, ARDS = acute respiratory distress syndrome, COVID-19 = 2019 Corona Virus Disease-19, CSS = cytokine storm syndrome, CT = computed tomography, HIV-1 = human immunodeficiency virus 1, IL-2R = interleukin-2 receptor, IL-6 = interleukin-6, MERS-CoV = middle east respiratory syndrome coronavirus, MOF = multiple organ failure, NCP = novel coronavirus pneumonia, RAS = renin-angiotensin system, SARS-CoV = severe acute respiratory syndrome coronavirus, SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2, SIRS = systemic inflammatory response syndrome, WHO = World Health Organization.

Keywords: 2019 Corona Virus Disease-19, clinical manifestations, drug development, epidemic characteristics, pathogenic mechanism.
1. Introduction

Unknown origin pneumonia has been furiously spreading since the late of December 2019, which has been epidemiologically and etiologically investigated by the Chinese Center for Disease Control and Prevention immediately.[1-4] Subsequently, a novel coronavirus, as the pathogen, was identified,[5] which was termed as 2019 novel coronavirus (2019-nCoV) by the WHO. Currently, the virus boasts a new nomenclature: severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), and the disease triggered by it is entitled 2019 Corona Virus Disease-19 (COVID-19); those infected patients mostly involved their lungs, the National Health Commission of the People’s Republic of China, therefore, named pneumonia induced by the virus as novel coronavirus pneumonia (NCP), and listed COVID-19 as a Class B infectious disease in the Law of the People’s Republic of China on the Prevention and Treatment of Infectious Diseases, indicting the prevention and control measures for Class A infectious diseases should be taken. By 18:00 on April 9, 2020, a total of 83,264 confirmed cases and 3344 cumulative deaths have been reported nationwide. Additionally, confirmed patients had been reported in several countries, most involving those people living or visiting Wuhan; and the virus has been well-established that person-to-person transmission is a truth.[2,3,6,7] This article summarizes the etiology, epidemiology, clinical manifestations, imaging findings, and medication on COVID-19 and analyzes and reviews the etiology, epidemiology, clinical manifestations, image findings, and medication of COVID-19 to help comprehensively understand and correctly cope with COVID-19.

2. Ethics

This study was approved by the ethics committee of the West China Hospital, Sichuan University, Chengdu, Sichuan, China. The information of patients involved in the present study has been anonymized.

3. Etiology

Coronaviruses refer to single positive-stranded RNA viruses that roundly or ovaly shaped (usually polymorphous) with envelope, which is characterized by the corolla shaped periphery protrusion on the viral envelope and is frequently associated with acute respiratory infections in humans that belong to a more sophisticated class of pathogens.[3,8-11] Zoonotic pathogens,[12] namely severe acute respiratory syndrome coronavirus (SARS-CoV) and middle east respiratory syndrome coronavirus (MERS-CoV) that initiate severe respiratory disease in humans, have spread worldwide and attracted much international attention. The Gao’s Group disclosed that SARS-CoV-2, different from genetic characteristics of MERS-CoV and SARS-CoV, is a novel coronavirus.[13] The physicochemical characteristics of SARS-CoV-2 have not yet been clarified, and it is generally accepted that SARS-CoV-2 is sensitive to ultraviolet light and heat and can be effectively killed by lipid solvents such as diethyl ether, while chlorhexidine is with poor effects.[11-16] SARS-CoV-2 is an RNA virus that may mutate during the occurrence and development of the disease, thereby making it more difficult for epidemic prevention and control.[2,5,17-20]

4. Epidemiology

Transmission is the central principle in biology and epidemiology of infectious diseases, and for many viruses, the transmission from animals to humans is one of the critical steps in the pathogenesis. SARS-CoV-2 owns 96% ribonucleic acid similarity to the coronavirus isolated from Rhinolophus sinicus in Yunnan province, China,[18] with origins traceable to a local seafood wholesale market in Wuhan, China. Bats may be the original reservoir of the virus with animals sold on the market by the intermediate hosts,[19] it is human and animal comorbidity ability remains exploration. The prime source of infection understood presently is patients with SARS-CoV-2 infection, and those asymptomatic infected individuals may also become the source of infection. Respiratory droplet and contact transmission are the main routes of transmission. SARS-CoV-2 has an S protein structure similar to SARS-CoV, which can bind to angiotensin-converting enzyme II (ACE2),[18,21-23] a cellular receptor in humans, to infect cells, it has been shown that ACE2 expression is especially abundant in lung and small intestinal tissues.[20] On February 13, 2020, personnel from the Chinese Center for Disease Control and Prevention isolated 2 novel coronaviruses from the feces of confirmed patients, demonstrating that there was indeed a live virus in the feces, which further suggested that we had the possibility of oral transmission of SARS-CoV-2. Whether aerosol or mother-to-child transmission is possible also needs to be further clarified. From the current dissemination rate to its interpersonal transmission ability, the population is generally susceptible. By further studying the affinity of SARS-CoV-2 to ACE2, it may be possible to verify its transmission capacity. The first survey by the Chinese Center for Disease Control and Prevention found that the average incubation period of this virus was about 3.2 days, with a maximum of fewer than 14 days, and the basic reproduction number was estimated to be 2.2,[1] which predicted the infectivity of SARS-CoV-2, which provided first-hand data for human understanding of the epidemiological characteristics of SARS-CoV-2, but epidemiological data may be continuously updated based on the increase of patients and a series of national prevention and control measures. Continue to monitor the epidemiological characteristics of more patients, closely investigate the source of the virus and intermediate hosts, and pay attention to its future evolutionary direction, adaptability, transmission ability, and route, all of which play a considerable role in guiding the prevention and control of the epidemic.

5. Clinical Manifestations

The Gao Group reported 3 patients with pneumonia of unknown cause found in the first time, with clinical manifestations of fever and cough with chest discomfort.[19] Huang et al.[24] reported the clinical features of the first 41 patients diagnosed with COVID-19, with infectious symptoms including fever, cough, and generalized weakness, but their upper respiratory symptoms were not prominent; however, gastrointestinal symptoms such as diarrhea, which are common in SARS-CoV infected patients, were not significant in COVID-19 patients. Chen et al.[25] retrospectively analyzed the clinical manifestations of 99 patients with COVID-19 and found that among 99 patients, fever and cough accounted for >50% of the total patients, shortness of breath accounted for 31%, muscle pain for 11%, and there were also few patients with confusion, headache, sore throat, runny nose, chest pain, diarrhea, nausea, and vomiting (Table 1). As we can see above, common symptoms are fever, cough, dyspnea, and fatigue, which may be accompanied by upper respiratory tract symptoms. COVID-19 Quick Advice for Diagnosis and
TNF-α cytokine storm syndrome (CSS).

Reason for its strong infectivity, infectivity, and pathogenicity.

10 to 20 times stronger than that of SARS, which may be the main reason for its strong infectivity.

Gastrointestinal symptoms may exist as the first manifestation of the virus due to the expression of ACE2 in the intestine[72]; cough with nausea, vomiting, and abnormal stools was reported in the first diagnosed patient in the United States.[28]

With the spread of the epidemic and the increase in the number of cases, COVID-19 patients may have atypical symptoms as the first manifestation.[29,30] For such patients, “early detection, early diagnosis, and early treatment” should be achieved to avoid missed diagnosis. In the early stage of the disease, the total white blood cell count in the peripheral blood of most patients may be normal or reduced, the lymphocyte counts reduced, the high-sensitivity C-reactive protein increased, and normal procalcitonin.[17,31-33]

6. Pathogenesis

6.1. The effect of cytokine storm on COVID-19

SARS-CoV-2 is an RNA virus of β-coronavirus. The S protein on its capsule membrane is a crucial protein for invading target cells. It binds to the target cell receptor ACE2 through the S protein and then begins to replicate and drift in large numbers after entering the target cells. The human body can eliminate SARS-CoV-2 through the occurrence of an immune response. However, the affinity between S protein of SARS-CoV-2 and target cell ACE2 is 10 to 20 times stronger than that of SARS, which may be the main reason for its strong infectivity, infectivity, and pathogenicity.[34]

After preliminary clinical observation, critically ill patients with COVID-19 may exhibit a significant increase in inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-γ (IFN-γ),[35] characterized by a cytokine storm syndrome (CSS).

CSS is an over-immune response induced by SARS-CoV-2 in the body, resulting in an imbalance of cytokine levels, such as TNF-α, IL-1, IFN-γ, IFN-α, and IFN-β.[35] Consequently, it is easy to make the immune system out of control and cause systemic inflammatory response syndrome (SIRS), ARDS, septic shock, multiple organ failure (MOF), and even death. de Wit et al.[46] reported that the characteristics of CSS caused by SARS-CoV, SARS-CoV-2, or MERS-CoV are different. After SARS-CoV-2 infection, TNF-α, granulocyte colony stimulating factor (G-CSF), and interferon-inducible protein-10 (IP10) cytokines were highly expressed, and the expression levels of TNF-α and G-CSF were firmly related to the degree of illness, which was different from MERS-CoV and SARS-CoV infection. After MERS-CoV infection, lung inflammation was caused by high expression of IFN-γ, TNF-α, IL-17, and other pro-inflammatory cytokines. After SARS-CoV infection, high expression of IL-6, IL-12, IFN-γ other cytokines resulted in the extensive pulmonary inflammatory response.

Moreover, SARS-CoV-2 infection mainly leads to diffuse alveolar injury and transparent membrane formation in the lungs. Although the degree of pulmonary fibrosis is not apparent, the inflammatory response in the lung tissue is severe. Therefore, it is necessary to monitor the patient’s inflammatory indicators to assess changes in the condition.

The lungs of patients who died of COVID-19 had the following manifestations:[37], diffuse alveolar injury with fibrosus mucus exudation in both lungs; the epithelial cells of alveoli were exfoliated, the hyaline membrane was formed, and pulmonary edema was observed; the inflammatory infiltration of monocytes in the lung stroma was mainly lymphocytes; multinucleated giant cells and atypical enlarged alveolar cells were found in the alveoli. Besides, after SARS-CoV-2 infection, peripheral blood CD4⁺ T and CD8⁺ T cells had low expression numbers, but higher activation degree.

6.2. The role of ACE2 in COVID-19

SARS-CoV-2 binds to the ACE2 receptor through S protein on the capsule, then enters the cell through endocytosis, and infecting the ciliated bronchial epithelial cells and type II alveolar cells in the lung. AP2 related protein kinase 1 (AAK1) is the main regulatory factor of endocytosis. The damage of AAK1 can prevent SARS-CoV-2 from entering the cell and inhibit the replication of the virus. Therefore, AAK1 inhibitor agents may become a potential target drug for anti-COVID-19 therapy in the future. The SARS-CoV-2 infection causes the renin-angiotensin system (RAS) imbalances, and serum Ang II expression level increased. Angiotensin-converting enzyme inhibitor (ACEI) therapy can significantly reduce the high levels of cytokines and lung inflammation caused by SARS-CoV-2 infection.[38] We believe that the regulation of ACEI levels or the treatment of COVID-19 with angiotensin receptor blockers (ARB) may be future research hotspots.

7. Diagnosis

7.1. Radiological findings

Computed tomography (CT) imaging is a strongly recommended auxiliary diagnostic modality.[28,39-48] In the early stage, due to pulmonary edema and hyaline membrane formation, alveolar sepal telangiectasia and congestion, CT mostly shows unilateral or scattered ground-glass opacity, which may be patchy and clumped, in which bronchial inflammation sign is observed. After alveolar fluid exudation and interlobular sepal interstitial edema, it may also disclose consolidation, nodular shadow, intralobular sepal thickening, and interstitial changes and other forms. The lesions mostly involve the outer lung field and subpleural area (Fig. 1).[47] Wang et al.[48] analyzed 14 cases in early-stage and found that there was a time difference in clinical symptoms and CT findings in some patients (Fig. 2). In some patients, the density of the lesion is pale, and the extent is small in the early stage, and chest radiography shows that it is easy to miss the diagnosis. Chest CT is recommended to replace chest radiography in those with conditions.
In the advanced stage, alveolar and interstitial edema are further aggravated due to the accumulation of a large amount of exudate in the alveolar space, and fibrinoid exudation causes alveolar fusion. CT findings develop from an early small shadow to a large consolidation of fusion, and a single lesion develops into multiple lesions (Fig. 3), in which the pneumatic bronchus sign is seen. However, the lesions around the nodules may have “anti-fainting sign,” fine grid shadows (fine vascular network) can be seen in the lesions, some lesions have “anti-fainting sign,” the lesions are mostly located under the pleura of the middle and lower lobes of both lungs, and pleural effusion is rare.

In the severe stage, diffuse ground-glass opacities or consolidation in critically ill patients can rapidly progress to “pulmonary fibrosis seen in the lungs” (Fig. 4). Clinical manifestations may be acute respiratory distress syndrome (ARDS), while secondary bacterial, fungal, and other pathogen infections. In this stage, the lesion progresses rapidly and is in critical condition, and a small proportion of patients can absorb and dissipate, which may leave fibrous streaks.

In summary, the characteristic changes of chest CT in new-coronary pneumonia are subpleural or peripheral ground-glass opacities or fainting signs, mostly with bronchial ventilation signs (Table 2).

7.2. Nuclear acid assays

The genome sequence of SARS-CoV-2 was published instantly after the start of the outbreak in Wuhan, China, on January 10, 2020. Reverse transcriptase-polymerase chain reaction (RT-PCR) was utilized to identify SARS-CoV-2 RNA as the main criterion for the diagnosis of COVID-19. Nevertheless, it has the disadvantage of a high false-negative rate which may facilitate the epidemic. Consequently, a combination of disease history, clinical features, laboratory examination, and CT image is vital for making an accuracy diagnosis.

8. Treatment

8.1. Medication

At present, there is no specific vaccine and antiviral drug, and research and development of a vaccine have its cycle and rule, which cannot be applied in clinical practice in the short term. Guidelines recommend that interferon-alpha, Lopinavir/Ritonavir, can be tried. Interferon-alpha enhances immunity, Lopinavir is a human immunodeficiency virus 1 (HIV-1) protease inhibitor with broad-spectrum antiviral activity, and Ritonavir can inhibit Lopinavir metabolism thereby increasing its plasma concentration, so Lopinavir is usually used in combination with...
Ritonavir. During SARS in 2003, Hong Kong scholars found that Lopinavir combined with Ritonavir reduced the risk of ARDS and death compared with ribavirin monotherapy alone.[19] Early application of Lopinavir/Ritonavir against coronavirus can reduce patient mortality and glucocorticoid dosage, and late application has no significant effect. Therefore, early use of Lopinavir/Ritonavir in patients with confirmed COVID-19 may be beneficial in clinical practice, but its efficacy and safety are still controversial and need to be further evaluated.

Holshue et al.[28] reported the trial administration of intravenous Remdesivir after the first confirmed patient’s deterioration in the United States and found that raloxifene may have a good effect on inhibiting SARS-COV-2. Remdesivir is a novel ribonucleic acid analog under development that is superior to Lopinavir/Ritonavir in combination with interferon-beta in the treatment of MERS-CoV.[11]

At present, clinical trials have been conducted in China to evaluate its effect on the treatment of COVID-19. On February 4, 2020, Li Lanjuan’s team released the latest research results in Wuhan: Abidol and Darunavir can effectively inhibit SARS-COV-2, which is a significant finding in treating COVID-19. To date, most of the broad-spectrum antiviral drugs used in clinical practice, and the therapeutic effect mostly needs practical evaluation. Blind use of antibacterial drugs should be avoided,
Table 2
CT findings of COVID-19 patients at different stages.

| Stage       | CT features                                                                                                                                                                                                 |
|-------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Early stage | (a) Unilateral or scattered ground-glass opacity, which may be patchy and clumped, in which bronchial inflation sign is observed  
(b) After alveolar fluid exudation and interlobular septal interstitial edema, it may also disclose consolidation, nodular shadow, intralobular septal thickening, and interstitial changes and other forms  
(c) The lesions mostly involve the outer lung field and subpleural area                                                                |
| Advanced stage | (a) Large consolidation of fusion, multiple lesions, in which the pulmonary bronchus sign is seen  
(b) The lesions around the nodules may have “anti-fainting sign,” fine grid shadows (fine vascular network) can be seen in the lesions, some lesions have “anti-fainting sign”  
(c) The lesions are mostly located under the pleura of the middle and lower lobes of both lungs, and pleural effusion is rare                                                                 |
| Severe stage | (a) Diffuse ground-glass opacities or consolidation in critically ill patients can rapidly progress to “pulmonary fibrosis seen in the lungs”  
(b) The lesion progresses rapidly and is in critical condition, and a small proportion of patients can absorb and dissipate, which may leave fibrous streaks |

COVID-19 = 2019 Corona Virus Disease-19; CT = computed tomography.

Table 3
Treatment options for COVID-19 patients.

| Author                  | Management                                                                 | Efficacy                                                                                           |
|-------------------------|----------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------|
| Jennifer[52]            | Respiratory support  
Supplemental oxygen therapy  
High flow nasal cannula  
Noninvasive positive pressure ventilation  
Invasive positive pressure ventilation  
Extracorporeal membrane oxygenation | To ensure the respiratory status of the patient does not deteriorate                                |
| Wang et al.[53]         | Cope with organs injuries  
Mechanical ventilation  
Symptomatic treatment  
Glucocorticoids  
antivirals  
Antishock therapy  
Drug treatment | To prevent multiple organ and systems injuries                                                   |
| Authors[2,24,30,36,51,54,55] | Antiviral treatment  
Antibiotic therapy  
Corticosteroids  
Antihistamines  
Antiplatelets  
Anticoagulants  
Chlorpromazine  
Cyclosporin A  
Mycophenolic acid  
Plasma | Currently, there is no evidence from a randomized controlled trial (RCT) to support specific drug treatment against the COVID-19 in suspected or confirmed cases. |
| Wang et al.[53]         | Control complications  
Monitoring clinical deterioration signs  
Assessing risk to recognize the deteriorating patient  
Timely and appropriate underlying disease treatment  
Providing general supportive care interventions timely | To proactively prevent potential complications, such as acute renal injury, sepsis, shock, thrombotic, ventilator-associated pneumonia, catheter-related bloodstream infection, pressure ulcers, stress ulcers, and gastrointestinal bleeding, ICU-related weakness and rhabdomyolysis |
| Wang et al.[53]         | Control comorbidities  
Monitor changes in comorbidities  
Assess all comorbidities of the individual  
Monitor for drug–drug interactions  
Develop individually tailored therapeutic approaches | To proactively prevent potential complications comorbidities (underlying diseases) such as hypertension, diabetes, cardio-cerebrovascular disease, kidney disease, liver disease, obesity, malignant tumors, chronic obstructive pulmonary disease |
| General Office of National Health Committee (China)[56] | Traditional Chinese medicine | Some drugs and herbal formulae also were recommended for treating COVID-19 in the latest version of China guideline for diagnosis and treatment of COVID-19. For the patients who can oral intake, a diet rich in protein, carbohydrates, and vitamins is recommended. Patients who cannot oral intake but are compatible with enteral nutrition should be given enteral nutrition as soon as possible. For the patients incompatible with enteral nutrition, parenteral nutrition should be given in time to meet the energy needs. |
| Jin et al.[54]          | Nutrition support | Relieve stress and improve mental health. The mental health interventions include formal psychotherapy for psychiatric disorders and a series of wellness and resilience-based psychosocial interventions for emotional distress and social problems. |
| North et al.[57]        | Mental health interventions |                                                                                                   |

COVID-19 = 2019 Corona Virus Disease-19.
especially in combination with broad-spectrum antibacterial drugs. The use of glucocorticoids is controversial. It should be used according to the patient’s condition. Appropriate use of glucocorticoids can reduce the inflammatory response and promote the absorption of pulmonary lesions. However, it should also be noted that high-dose glucocorticoids will delay the clearance of SARS-COV-2 and have a certain incidence of adverse reactions.

Immunoglobulins are purified blood products from the human body, containing a large number of antibodies, which interact with antigens to neutralize and kill bacteria and viruses. Under the action of large doses, they can also remove immune complexes in the human body and be used in combination with antiviral drugs, which can improve the efficacy for the infection of some serious viral diseases. Therefore, the application of immunoglobulins in treating COVID-19 is worthy of clinical promotion. Patients with COVID-19 can have natural antibodies, and it is necessary to consider convalescent plasma therapy when available. The S protein of SARS-COV-2 has a unique Fyn-like cleavage site (RRAR), which has potential significance in studying its pathogenicity and developing drugs such as vaccines.

We note that SARS-COV-2 can bind to ACE2, providing direction for drug development. For patients with hypertension, ACEI/ARB drugs should be avoided in the selection of antihypertensive drugs. Patients often have anxiety and fear, and the treatment process strengthens the psychological counseling for patients.

Besides, other treatment options, such as respiratory support, cope with organs injuries, Traditional Chinese Medicine, nutrition support, and mental health interventions, are also very essential for COVID-19 patients and listed in Table 3.

9. Prognostic factors

The prognosis factors of COVID-19 patients include age, sex, obesity, smoking, C-reactive protein (CRP), D-dimer, and lymphocyte count.

About age, it was one of the most critical factors for COVID-19. COVID-19 mainly occurred in humans between 30 and 65, and people older than 65 years have a poor prognosis. As for sex, men are more susceptible to COVID-19 than women. Concerning obesity, it contributes to the increased risk and poorer prognosis of COVID-19. As for smoking, it is very likely to be related to the negative progression and adverse consequences of COVID-19.

Hematology examination includes CRP, D-dimer, lymphocyte count, and so on. CRP level can reflect the severity of inflammation, which has also been reported to be closely associated with the severity and prognosis of COVID-19. Scholars have reported elevated CRP levels in most COVID-19 cases. Wang et al. reported that patients with the severe disease showed an increase in D-dimer concentration and a progressively decrease in lymphocyte count.

10. Conclusions

There has been some understanding of SARS-COV-2, a novel coronavirus, whose epidemiology is not yet fully understood and requires further investigation. Typical symptoms after infection are fever, fatigue, cough, dyspnea, etc., and atypical symptoms may be the first manifestation. Close monitoring of inflammatory markers during treatment avoids excessive inflammatory response. Typical chest CT findings are subpleural or peripheral ground-glass opacities or fainting signs, mostly with bronchial ventilation signs. Its imaging findings change rapidly, and the lesions can increase or decrease in a short time, so the interval between reexaminations should be shorter than that of common pneumonia, and reexamination in 2 to 3 days is recommended in the early stage (when the lesions are unstable). With the gradual increase of confirmed and suspected cases, the lag of nucleic acid detection and the lack of medical resources, whether to use CT instead of nucleic acid detection to diagnose COVID-19, has become a topic of discussion. It should be clear that during the outbreak, when nucleic acid detection is negative, it still needs to be comprehensively evaluated according to the clinical manifestations of patients, relevant examinations and the experience of doctors to avoid missing suspected patients, and causing the spread of the range. Timely and effective therapeutic measures are particularly necessary. At present, there is no specific vaccine and antiviral drug. It is hoped that more effective drugs can be screened on the basis of exerting known drug efficacy, and vaccines and specific drugs can be developed as soon as possible. Timely use of immunoglobulin and antibacterial drugs, rational use of glucocorticoids, and at the same time, psychotherapy should not be forgotten. At present, there are many problems to be discovered and solved urgently for COVID-19. We must recognize that COVID-19 brings great challenges to mankind, and it is urgent to understand and control the epidemic situation.

Author contributions

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