An Acute Respiratory Infection Runs Into the Most Common Noncommunicable Epidemic—COVID-19 and Cardiovascular Diseases

The outbreak of novel coronavirus disease 2019 (COVID-19), which emerged in Wuhan, China, in December 2019, has rapidly spread to more than 58 countries and areas. As of March 1, 2020, about 79,968 cases in mainland China have been confirmed and 2,873 deaths have occurred. The Chinese government is mustering medical personnel around the country to treat patients in Hubei province and preventing further spread of COVID-19 in every region of the country.

At the same time, several studies concerning the epidemiological and clinical features of COVID-19 have been published in a timely manner, which has greatly helped health care workers and policy makers to understand COVID-19. Reading these reports, we noticed that many patients with COVID-19 had comorbid chronic cardiovascular diseases (CVDs), which collectively represent the most common noncommunicable epidemic in China currently. Among 44,672 individuals confirmed to have COVID-19 as of February 11, 2020, 26,833 patients (12.8%) had hypertension and 8,733 (4.2%) had CVDs, which were the most common coexisting conditions in patients hospitalized with COVID-19. Furthermore, patients with comorbid CVDs were more likely to have severe illness associated with COVID-19 and had a much higher fatality rate: 10.5% for those with CVDs and 6.0% for hypertension, while the fatality rate was 0.9% in patients who reported no comorbid conditions. Notably, severe acute respiratory syndrome coronavirus (SARS-CoV), which caused a global epidemic in 2003, is recognized as a sister to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, which emerged in Wuhan, China, in December 2019, has rapidly spread to more than 58 countries and areas. In a study of 46 patients with SARS, Li et al found subclinical diastolic impairment, which may be reversible on clinical recovery, but there was no interstitial lymphocytic infiltrate or myocyte necrosis in the heart of a patient with the lowest left ventricular ejection fraction. It was postulated that the temporary diastolic impairment detected by echocardiography might be attributable to systemic inflammatory storm resulting from an

Corresponding Authors: Zening Jin, MD, PhD (jin.zening@hsc.pku.edu.cn), and Chengzhi Yang, MD, PhD (yangcz@hsc.pku.edu.cn), Department of Cardiology and Macrovascular Disease, Beijing Tiantan Hospital, Capital Medical University, Beijing, China.
overaggressive host immune response to viral infection, given that several studies suggested several cytokines, such as tumor necrosis factor and the interleukin-6 family, have clinically significant negative inotropic influence. Further detection by whole-genome sequencing, direct polymerase chain reaction, and culture with biopsy samples may help to identify whether SARS-CoV-2 is present in myocardium. Cardiac magnetic resonance imaging may be helpful to detect cardiac injury noninvasively.

Of note, several investigations have demonstrated that SARS-CoV-2 and SARS-CoV share the same host receptor, angiotensin-converting enzyme 2 (ACE2), which may have many protective effects on CVDs. A recent study revealed that affinity of SARS-CoV-2 binding to ACE2 was 10-fold to 20-fold higher than that of SARS-CoV, which suggests SARS-CoV-2 could spread from human to human easily. Therefore, ACE2 may play 2 contrary roles in patients with COVID-19, especially those with comorbid CVDs. On the one hand, ACE2 may provide protection against hypertension, myocardial fibrosis, myocardial hypertrophy, arrhythmia, atherosclerosis, and sodium-water retention. On the other hand, ACE2 acts as the gate for SARS-CoV-2 infection. Previous studies have shown that angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) could upregulate the expression or prevent the loss of ACE2, which is one of the mechanisms of ACEI/ARB activity. At this point in our knowledge, it is conceivable that ACEI/ARB therapy might increase the risk of SARS-CoV-2 infection. However, experimental evidence indicates that the ARB losartan and recombinant human ACE2 can protect mice from severe acute lung injury induced by acid aspiration or sepsis. Considering that ACEI/ARB therapy is widely prescribed to patients with hypertension, heart failure, and ischemia heart disease, additional attention should be paid to patients with COVID-19 and close contacts who are using ACEI/ARB therapy.

To date, many patients with COVID-19 are still hospitalized in China and other countries, such Italy and Iran. Therefore, continued observations of the cardiovascular complications of the disease are needed. In addition, further assessment is needed to identify risk factors for poor prognosis.

Emerging as an acute infectious disease, COVID-19 may become a chronic epidemic similar to influenza because of genetic recombination. Therefore, we should be ready for the reemergence of COVID-19 or other coronaviruses. Considering that CVDs represent the leading noncommunicable epidemic around the world and numerous patients with CVDs are using ACEI/ARB, clinical studies may be necessary to explore the potential associations of ACEI/ARB with susceptibility and prognosis of COVID-19.