

COVID-19: Notes for Cardiologist

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ABSTRACT

The emerging novel coronavirus outbreak (COVID-19) highlights several interesting notes for cardiologist. Research has confirmed Angiotensin-converting enzyme 2 (ACE2) as the key SARS-CoV-2 (the cause of COVID-19) cell entry receptor. Currently, patients with clinical COVID-19 have often mild symptoms but severe patients quickly develop complications especially in presence of comorbid conditions such as diabetes or hypertension, underlying cardiovascular disease including CHD, and in the elderly patients. COVID-19 can affect the cardiovascular system also leading to severe myocardial damage closely related to the severity of the disease and even the prognosis and increased levels of biomarkers were found. Research has called attention to several mechanisms related to cardiovascular involvement and cardiovascular treatment could offer additional protection to CVD patients.

Key words: ACE2; COVID-19; SARS-CoV-2; Protease transmembrane protease serine 2 (TMPRSS2)

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EDITORIAL

The emerging novel coronavirus outbreak (COVID-19) highlights several interesting notes for cardiologist. Research has confirmed Angiotensin-converting enzyme 2 (ACE2) as the key SARS-CoV-2 (the COVID-19 causative agent) cell entry receptor^[1] with ACE2 affinity 10-20 times higher than SARS-CoV^[2]. Research has reported that the imbalance between ACE/Ang II/ ACE2 type 1 receptor pathway and ACE2/Ang (1-7)/Mas receptor pathway leads to multi-system inflammation^[1] and has called attention to different human ACE gene polymorphism and to different ACE2 cellular, individual, gender and age expression^[1]. Currently, patients with clinical infection of COVID-19 have often symptoms such as fever, shortness of breath, myalgias, headache nasal congestion, runny nose, sore throat, dysgeusia, anosmia and diarrhea. Severe patients quickly develop complications such as severe pneumonia, coagulation dysfunction, acute respiratory distress syndrome, septic shock, systemic multiple organ insufficiency^[1,2] especially in presence of comorbid conditions such as diabetes or hypertension^[1], underlying cardiovascular disease^[2] including CHD^[5], and in the elderly patients^[2]. COVID-19 can affect the cardiovascular system also leading to severe myocardial damage^[2] closely related to the severity of the disease and even the prognosis^[2]. COVID-19-associated arrhythmias, acute cardiac injury and myocarditis have been reported^[2,3]. The patients complain palpitations as the first symptom^[2] and often present with sinus tachycardia, especially overnight and incommensurate with the increase in body temperature (> 10 beats / °C)^[2]. Research has found increased levels of High-sensitivity cardiac troponin I (hs-cTnI), creatine kinase (CK), creatine kinase isoenzymes (CK-MB), lactate dehydrogenase^[2], NT-proBNP^[3], hs-CRP, serum creatinine and has shown that age, male, elevated NT-proBNP, elevated cTnI, elevated hs-CRP, elevated serum creatinine, hypertension, and CHD were significantly correlated with critical disease status at univariate logistic

regression analysis^[3]. Multivariate logistic regression analysis has shown that elevated cTnI and CHD were the independent risk factors of critical disease status^[3]. Research has also called attention to several mechanisms related to cardiovascular involvement such as direct virus induced cardiomyocyte damage, insufficient myocardial oxygen supply and demand imbalances, cytokine storms induced myocardial damage and severe organ failure^[2]. Statins, beta blockers, ACE inhibitors, ASA could offer additional protection to CVD patients as reported in recent ACC clinical bulletin focuses on cardiac implications of coronavirus (COVID-19)^[4]. Research has recommended the monitoring of cardiac function and myocardial injury and providing to consequential other cardiovascular drugs and supports^[2]. Research has also demonstrated that SARS-CoV-2 uses the cellular protease transmembrane protease, serine 2 (TMPRSS2) for COVID-19 priming^[2]. A nuclear factor erythroid 2-related factor 2(Nrf2)- secretory leukocyte protease inhibitor (SLPI)-Dependent Inhibition of TMPRSS2 Activity has shown as protective against virus infection^[6] and multiple lines of evidence support the potential of dialing up the Nrf2 pathway for cardiac protection^[7]. At present, there is no definite and effective treatment for COVID-19^[1] and further investigation are needed.

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