In most patients, coronavirus disease-2019 (COVID-19) causes minor respiratory symptoms or even no symptoms. SARS-CoV-2 infection can also cause extrapulmonary manifestations and complications. COVID-19 is more severe and fatal among patients with pre-existing cardiovascular risk factors or diseases. Increases in cardiac troponin (cTn) are indicative of myocardial injury and frequently found in patients with COVID-19. The Chinese Center for Disease Control and Prevention published a survey demonstrating that among patients diagnosed with COVID-19, 13% had hypertension, 5% had diabetes mellitus, and 4% had a history of cardiovascular disease. However, in this same cohort, among patients who had not survived, 40% had hypertension, 20% had diabetes, and 22% had pre-existing cardiovascular disease. Patients with cardiovascular disease had the highest case fatality rate (10.5%).

In this issue of the International Journal of Cardiovascular Sciences, Rocha et al., studied 192 patients admitted with COVID-19 in a quaternary care cardiac hospital in Rio de Janeiro, Brazil. Mortality rate was 28%. Multivariate analysis demonstrated that elevated cTnI levels (OR=9.504; 95% CI=1.281–70.528; $P=0.028$) upon admission and the need for mechanical ventilation during hospitalization (OR=46.691; 95% CI=2.360–923.706; $P=0.012$) were independent predictors of death during hospitalization. The main limitations of the study are a sample from a single Brazilian metropolitan region and a short follow-up period. However, these limitations do not invalidate the main message of the study: the measurement of cardiac troponin at hospital admission may be useful for identification of high-risk patients infected by SARS-CoV-2.

Cardiac troponins are not specific markers of ischemic injury of the heart. The Fourth Universal Definition of Myocardial Infarction defines myocardial injury (acute or chronic) as cTn concentrations >99th percentile upper reference limit (URL). While dynamic changes in cardiac troponins characterize acute injury, patients without these changes have chronic injury. Patients with COVID-19 have several conditions that may be associated with myocardial injury such as myocarditis, stress cardiomyopathy, acute heart failure, pulmonary embolism, critical illness, and sepsis. Garcia de Guadiana-Romualdo et al., demonstrated that elevated troponin levels were common in patients with COVID-19. These authors found abnormal levels of cTn in 26.9% of the patients and in 30% when sex-specific cut-offs were used to detect myocardial injury. Some factors increase the risk of abnormal cTn levels, including older age and the type of troponin measured (troponin T is associated with higher levels). Nascimento et al., studied 61 patients admitted to intensive care unit with COVID-19 in a Brazilian hospital and found a high incidence of myocardial infarction in patients with severe COVID-19, with impact on in-hospital mortality.

COVID-19 can damage the cardiovascular system in many ways. A myocardial oxygen supply-demand...
mismatch (type 2 myocardial infarction) can result from an imbalance between high metabolic demand and low cardiac reserve, systemic inflammation and thrombogenesis, in addition to direct cardiac damage from the virus. Cardiovascular complications occur mainly in patients with cardiovascular risk factors (advanced age, hypertension and diabetes) or preexisting cardiovascular diseases. There are few reports of COVID-19 patients who presented with acute ST-segment elevation myocardial infarction (STEMI) that the final diagnosis was myocarditis. This diagnosis is supported by elevated cardiac troponins, moderate decrease of left ventricular ejection fraction, and absence of flow-limiting coronary artery disease by invasive coronary angiography. Autopsy findings support the concept that the pathogenesis of severe COVID-19 involves a virus-induced injury of multiple organs, including heart and lungs, coupled with the consequences of a procoagulant state with coagulopathy, although overt myocarditis is very rare. Together, these data suggest that in most cases, myocardial injury detected by increased cardiac troponins are not causes of severe COVID-19, but rather consequences/effects of high risk COVID-19.

Type I myocardial infarction is not commonly associated with COVID-19 and some studies have shown even a decreased incidence of hospitalization for acute myocardial infarction during the Covid-19 pandemic. Solomon et al., reported a decrease by up to 48% in weekly rates of hospitalization for acute myocardial infarction during the COVID-19 period. De Filippo et al., found similar reductions in 15 hospitals in northern Italy. This decrease may also be associated with the anxiety and fear of catching COVID-19 in the emergency department, commonly seen among patients during the initial months of the pandemic. Consequently, parallel to this decrease in myocardial infarction hospitalizations, a transient increase in the incidence of out-of-hospital cardiac arrest (AOHCA) was observed when compared with the equivalent time period in previous years with no pandemic. The same trend was demonstrated in a Brazilian study by Guimarães et al., who found a proportional increase of 33% of home deaths in March 2020, which is when the World Health Organization declared the COVID-19 pandemic. The increase of AOHCA can be linked to COVID-19 infections and to the potential increase of patients with acute cardiac diseases that did not seek emergency care.

We are entering a new phase of the COVID-19 pandemic. The United Kingdom’s Medicines and Healthcare products Regulatory Agency has issued temporary authorization of the antiviral drug molnupiravir for the treatment of mild to moderate COVID-19 in adults with at least one risk factor for severe illness. The approval of effective oral drugs, combined with the use of effective vaccines against COVID-19, can potentially change the scenario and contribute to a consistent decrease in the number of cases and the adverse effects of SARS-COV-2 infections. However, we must remember that there is a long way to go to let our guard down. Science needs to be protected and valued; now more than ever.

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References


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