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CARDIOVASCULAR DISEASE THAT LEADING TO DEATH OF PATIENTS IN THE COVID-19

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Annotation

Acute respiratory viral infections can result in cardiovascular involvement, with such patients having a significantly higher mortality rate than those without cardiovascular involvement. Due to the ongoing coronavirus disease 2019 (COVID-19) pandemic, it is important to determine whether cardiovascular risk factors are associated with the severity of COVID-19. The major cause of death in COVID-19 is acute respiratory distress (ARDS); however, there is also significant other vital organ involvement, including the cardiovascular system and shock.

Keywords: myocardial injury**,** heart failure, hypertension, pulmonary embolism, arrhythmias and sudden cardiac arrest, thromboembolism and coagulation abnormalities, acute coronary syndrome

The myocardial injury appears to be a common feature in COVID-19 and portends a poor prognosis when present. A meta-analysis of six published studies from China found that 8% of patients with COVID-19 had associated cardiac injury.[8] Several case series have examined cardiac enzyme elevations indicating myocardial damage that could be secondary to ischemic or non-ischemic causes. Other studies have reported patients with viral myocarditis, myocardial injury, and inflammation without an ischemic cause.

Heart failure has been well described in patients with pneumonia, and now multiple studies have shown an association between heart failure and COVID-19. In a case series of 21 severely ill patients with COVID-19 in an ICU, one-third developed new-onset cardiomyopathy with globally decreased left ventricular ejection fraction on transthoracic echocardiogram with clinical signs of cardiogenic shock and elevated creatine kinase or troponin I. In a cohort of 191 patients with COVID-19 from Wuhan, 23% were diagnosed with heart failure; of the 54 patients who died, 52% had heart failure. In another study of 799 COVID-19 patients from Wuhan, heart failure was observed in 24% of patients and 49% of those who died. Patients with COVID-19 may develop right-sided heart failure secondary to pulmonary

hypertension due to hypoxia and ARDS. In a cohort of 105 COVID-19 patients hospitalized at one center, right ventricular dilation was present in 31% of intubated patients. Right ventricular hypokinesia was observed in 66% of COVID-19 patients with right ventricular enlargement versus 5% of COVID-19 patients without right ventricular enlargement. A few studies have reported takotsubo (stress-induced) cardiomyopathy as a complication of COVID-19. Most cases occurred in women older than 65 years of age. The association with COVID-19 infection is not clear, but it has been suggested that the emotional distress and anxiety associated with the pandemic may have a role in the onset of this disease. The etiology of heart failure in COVID-19 disease is unclear whether it is a direct effect of SARS-CoV-2 on the myocardium or indirectly caused by hypoxia, cytokine release, volume overload, renal failure, stress, or overwhelming critical illness. Further, some patients with risk factors for heart disease (diabetes, hypertension, hyperlipidemia, coronary artery disease) may have had underlying subclinical heart failure uncovered or exacerbated by COVID-19 infection and associated illness. Acute coronary syndromes triggered by COVID-19 can also result in heart failure or worsen the preexisting disease. Diagnostic workup for suspected heart failure includes brain natriuretic peptide, troponin markers, transthoracic echocardiography, and cardiac MRI. Cardiac MRI may help look for changes induced by COVID-19 in patients with diastolic heart failure. Knowledge of the presence or absence and degree of cardiomyopathy is crucial for managing patients in shock status and determining the need for circulatory support and type of extracorporeal membranous oxygenation. The management of heart failure in patients with COVID-19 should be initiated and continued as per current guidelines.

Arrhythmias and sudden cardiac arrest have been reported with COVID-19. In a report of 138 hospitalized patients with COVID-19 in Wuhan, 17% had arrhythmias although the specific types of arrhythmias were not described. In another study of hospitalized COVID-19 patients in Wuhan, 6% had developed ventricular tachycardia or ventricular fibrillation. More patients with elevated troponin T levels (17%) than normal troponin T levels (2%) had ventricular tachycardia or ventricular fibrillation. In patients with COVID-19, a cardiac injury that induces arrhythmia can be due to various causes, such as hypoxia, a worsening of coronary perfusion, direct tissue damage, hyperacute systemic inflammatory response syndrome, or the effects of medications used to manage the COVID-19. Hypokalemia can occur in patients

with COVID-19 due to the interaction of SARS-CoV-2 with the renin-angiotensin-aldosterone system and increases vulnerability to various kinds of arrhythmia. Recommendations for managing arrhythmias are similar to those for non-COVID patients, including electrolyte optimization, avoidance of triggers, and medication modification. Concomitant ECG monitoring for patients who have long QTc or taking medications known to prolong the QTc interval is mandated. Cardiac arrest was reported in 11% of COVID-19 patients with ECG evidence of ST elevation in a case series from New York.[90] A study from Italy looked at local out-of-hospital cardiac arrests during the first 40 days of the COVID-19 outbreak and compared the rate with that from the same period a year ago. During the study period, there was a 58% increase in out-of-the hospital cardiac arrests (362 cases compared to 229 cases the year before) correlating to the incidence of COVID-19. Of the 362 cases of out-of-hospital cardiac arrest, 28% had, or were suspected of having, COVID-19.

COVID-19 infection has been associated with venous and arterial thromboembolism. Studies have shown abnormalities of the coagulation cascade, with elevated D-dimer, thrombocytopenia, slightly elevated prothrombin time, and higher levels of fibrinogen and von Willebrand factor. The hypercoagulable state in COVID-19 infection is thought to be related to severe inflammatory response, cytokine storm, and endothelial damage, along with underlying patient comorbidities. In a retrospective cohort study from China, a D-dimer >1 mcg/mL was reported in 42% of patients and in 81% of those who died from COVID-19; as such, it was identified as one of the risk factors for death and was associated with 18-times increased risk for mortality.[24] A Wuhan study found elevated D-dimer in 46% of patients, 60% of patients with severe illness, and up to 70% of patients in a composite group with ICU admission, mechanical ventilation, or death. Platelet count was <150,000/mm3 (signifying thrombocytopenia) in 36% of patients and 58% of patients with severe disease. In an autopsy series of COVID-19 patients from Germany, deep venous thrombosis was found in 58% of patients in whom venous thromboembolism was not suspected before death. Pulmonary embolism was found in 4 of the patients and was the direct cause of death. COVID-19 infection has been associated with cerebrovascular accidents. The incidence of acute ischemic stroke in patients with COVID-19 is approximately 1%–3%. A review of 37 studies with 370 patients with COVID-19 who had developed acute ischemic stroke or transient ischemic attack found that most patients had underlying comorbidities predisposing

them to ischemic stroke.[95] However, case reports describe large-vessel strokes in young adults with COVID-19 who did not have any cardiovascular risk factors. In a case series from the United Arab Emirates, 22 patients with confirmed COVID-19 infection presented with ischemic stroke symptoms as the first evidence of their COVID-19 infection; most were male and younger than 55 years of age.[96] Patients with COVID-19 and stroke are reported to have significantly higher mortality than historical controls. In addition to arterial strokes, cerebral venous sinus thrombosis has been reported in 13 patients in 9 studies. Peripheral arterial thromboembolism causing acute limb ischemia also has been described in COVID-19. Two young patients without any known risk factors have been diagnosed with acute thrombosis involving the aorta presenting as acute limb ischemia. A report from Italy described four patients with COVID-19 who developed acute limb ischemia; two had comorbidities, but the other two were young and active, without any comorbidities. In another Italian case series, 20 patients were diagnosed with COVID-19-related pneumonia before acute limb ischemia was detected; revascularization was less successful than expected, possibly secondary to a virus-related hypercoagulable state. A case series from Spain described acute limb ischemia in four patients infected with COVID-19 secondary to a hypercoagulable state. None of these patients had known cardiovascular disease or comorbidities that could have predisposed them to arterial embolisms. There is increasing evidence that anticoagulation is of benefit in COVID-19 illness. A study from New York demonstrated that treatment-dose anticoagulation was associated with reduced mortality. In the study, 786 out of 2773 patients (28%) were administered systemic anticoagulation. In-hospital mortality for patients who received anticoagulation was 23%, and median survival was 21 days, compared with 23% and median survival of 14 days in patients who did not receive treatment-dose anticoagulation. In patients who needed mechanical ventilation, in-hospital mortality was 29% for those who received treatment-dose anticoagulation, and median survival was 21 days, versus 63% and median survival of 9 days in patients who did not receive treatment-dose anticoagulation. A retrospective study from China showed that the use of low molecular weight heparin was associated with better prognosis in severe COVID-19 patients meeting sepsis-induced coagulopathy criteria or with markedly elevated D-dimer. At this time, most centers have incorporated anticoagulation into the treatment protocol for COVID-19 patients, especially if they have elevated D-dimer

levels. However, it is unknown which anticoagulation agent (unfractionated heparin, low-molecular-weight heparin, warfarin, or direct oral anticoagulants) is most efficacious in preventing thromboembolic events in COVID-19 patients. As always, the risks and benefits of anticoagulation treatment must be weighed for each patient. Future clinical trials may shed more light on the benefits of anticoagulation in COVID-19 infection.

Several studies and case reports have established an association between COVID-19 and acute coronary syndrome. In a letter to the editor, investigators from New York reported their experience with COVID-19 patients who showed ST-segment elevation on ECG.[90] Ten patients had ST-segment elevation at the time of presentation, whereas the other eight developed ST changes during hospitalization. Nine patients underwent coronary angiography, and six were found to have an obstructive disease; five of these six patients needed percutaneous coronary intervention. Overall, eight patients were diagnosed as having an acute myocardial infarction, and the other ten were deemed to have a noncoronary myocardial injury. Of the 13 patients (72%) who passed away during hospitalization, four had a myocardial infarction, and the other nine had a noncoronary myocardial injury. In Italy, researchers published a study of 28 patients with confirmed COVID-19 who had undergone with coronary angiogram for ST-elevation myocardial infarction. Of these, 79% had typical chest pain, while 21% presented with dyspnea without any chest pain; 86% had ST-elevation myocardial infarction as the initial presentation of COVID-19. This suggests that COVID-19 caused acute coronary syndrome (ACS) without substantial systemic inflammation.[105] The pathophysiology of ACS in COVID-19 is not clear, but it may be related to direct endothelial injury by the SARS-CoV-2 virus, microthrombi formation, inflammation and cytokine storm resulting in plaque rupture or coronary spasm. Despite the association between COVID-19 and ACS, the reported incidence of ACS has been lower during the pandemic than in the pre-COVID-19 period. Reports indicate a 42%–48 % reduction in ACS hospitalizations and 38%–40% fewer percutaneous coronary interventions performed for ST-elevation myocardial infarction. This could be due to patients being reluctant to present to hospitals and clinics out of fear of contracting COVID-19. Unavailability of beds has also been a reason, as hospitals have been flooded with sick COVID-19 patients precluding many other admissions, particularly non-urgent cases.

All in all, our results showed that those over 60 years, of the male sex, or those with heart failure, cardiovascular risk factors; hypertension, diabetes mellitus, and CKD have an increased risk of severe COVID-19. Further, the risk scoring model showed the significance of multiple risk factors. Those with four risk factors, old age (≥60 years), male sex, hypertension, and diabetes mellitus, had odds ratio more than 100 of severe COVID-19 than those without these risk factors, although it should be taken into account that it can be statistically exaggerated due to relatively small numbers of patients. In addition, dementia and cancer were also found to be related to severe COVID-19.

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