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Cardiovascular Manifestations of COVID-19

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Although novel coronavirus disease 2019 (COVID-19) mainly affects lung, it also affects the cardiovascular system. Cardiac manifestations of COVID-19 include myocardial injury, myocarditis, heart failure, acute coronary syndrome, thromboembolism, and arrhythmias. Cardiovascular involvement in COVID-19 is related to poor prognosis and increased mortality. Therefore, the recognition of cardiac complications and prompt treatment of cardiac injury in patients with suspected COVID-19 is essential. In this review article, we discuss the cardiovascular manifestations of COVID-19 and their related imaging findings.

Key words COVID-19 · Cardiovascular abnormalities · CT · MRI.

INTRODUCTION

Since severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) was first identified in Wuhan, China, it has spread rapidly and the World Health Organization declared a global pandemic on March 11, 2020 [1]. As of June 30, 2020, more than 10 million people have been infected and more than 500000 have died [2]. Although the main clinical manifestations of novel coronavirus disease 2019 (COVID-19) are pneumonia and respiratory symptoms, serious cardiovascular complications have also been reported [3-5]. Cardiovascular involvement in COVID-19 is associated with increased mortality risk [6]. Cardiac manifestations of COVID-19 include myocarditis, heart failure, stress-induced cardiomyopathy, arrhythmia, and thromboembolism [3,7-9]. Patients with underlying cardiovascular disease are particularly vulnerable to cardiovascular complications caused by COVID-19 and have a poor prognosis [10,11]. Therefore, cardiovascular radiologists and clinicians must be aware of the cardiovascular complications of COVID-19.

UNDERLYING CARDIOVASCULAR DISEASE IN COVID-19 PATIENTS

A considerable proportion of patients have preexisting cardiovascular diseases, such as coronary artery disease, heart failure, arrhythmias, which are related to poor prognosis after COVID-19 infection. A recent meta-analysis reported that the pooled prevalence of cardiovascular disease in COVID-19 patients was 12.1% [12]. Pre-existing cardiovascular disease significantly increases the risk of in-hospital death and fatal outcomes [10]. Recent meta-analysis also reported that preexisting cardiovascular disease increased the risk of severe COVID-19 [odds ratio (OR), 3.14; 95% confidence interval (CI), 2.32−4.24] and of all-cause mortality (OR, 11.08; 95% CI, 2.59−47.32) [13]. Severe COVID-19 in this meta-analysis was defined as a composite of: 1) respiratory distress, defined as a respiratory rate ≥30 per minute; 2) oxygen saturation on room air at rest ≤93%; 3) partial pressure of oxygen in arterial blood/fraction of inspired oxygen ≤300 mm Hg; 4) requirement for mechanical ventilation vital life support, or intensive care unit (ICU) admission; 5) and death [13]. This is thought to be because patients with underlying cardiovascular disease tend to be older and are more likely to have other comorbidities and an impaired immune system than the younger patients [14].

CARDIOVASCULAR INVOLVEMENT OF COVID-19

Pathophysiology

Several mechanisms that explain the pathophysiology of cardiac injury in COVID-19 have been proposed (Fig. 1) [14]. The

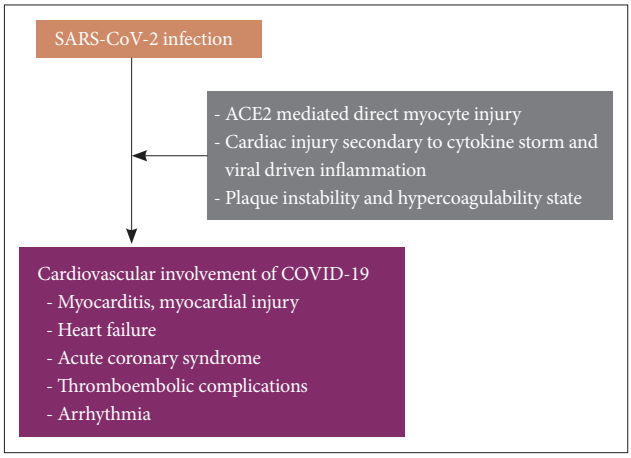

Fig. 1. Pathophysiology of cardiovascular involvement in COVID-19. SARS-CoV-2: severe acute respiratory syndrome-coronavirus 2, ACE: angiotensin converting enzyme, COVID-19: novel coronavirus disease 2019.

first is direct cardiac damage caused by the virus. SARS-CoV-2 is a single-stranded RNA virus that enters cells after binding to the angiotensin-converting enzyme 2 (ACE2) protein [15,16]. ACE2 is highly expressed in type 2 lung alveolar cells, as well as in the heart and blood vessels [17]. ACE2 mediates direct entry of the virus into cells which subsequently causes cardiotoxicity. Another mechanism is myocardial injury secondary to viral driven inflammation and cytokine storm syndrome. COVID-19 has been demonstrated to cause severe inflammation by increasing the expression of plasma cytokines and chemokines such as interleukin (IL)-6, IL-2, IL-7, IL-8, granulocyte colony-stimulating factor, and tumor necrosis factor [18]. This systemic cytokine storm can lead to multi-organ dysfunction and cardiac overload that progresses to cardiac damage due to a mismatch between myocardial oxygen supply and demand. Moreover, the severe inflammation caused by SARS-CoV-2 leads to a hypercoagulable state and increased plaque vulnerability which predisposes patients acute coronary syndrome (ACS) and venous thromboembolism. Further research is required to improve our understanding of the multifactorial etiology of cardiovascular complications in COVID-19 patients.

Myocardial injury

Myocardial injury is defined as an increase of the cardiac biomarkers such as troponin I and troponin T above the 99th percentile of the upper normal reference [19,20]. As cardiac troponin level cannot distinguish the cause of myocardial injury, considering them together with electrocardiogram changes, or global or regional left ventricular (LV) wall motion abnormalities can help to determine the extent and potential cause of myocardial injury. The reported prevalence of myocardial injury in COVID-19 patients ranges from 7−28% [6,21]. Patients with severe COVID-19 requiring mechanical ventilation, ICU admission, and those who ultimately die have more myocardial injury than those with milder forms [22]. Furthermore, patients with underlying cardiovascular disease are more likely to have myocardial injury than those without [23]. Myocardial injury significantly increases the risk of poor outcome and death compared to patients without myocardial injury [23-25]. A recent study reported that even mildly increased troponin I (troponin I 0.03−0.09 ng/mL) was significantly associated with death [adjusted hazard ratio (HR) 1.75, 95% CI 1.37−2.24; p<0.001], and this association was even stronger in patients with troponin I >0.09 ng/dL (adjusted HR 3.03, 95% CI 2.42−3.80; p<0.001) [23].

Myocarditis

COVID-19 related myocarditis has been reported in various countries, but the exact incidence is unclear [3,26-30]. It commonly affects children and young adults [3,30,31]. The clinical presentation of myocarditis is variable and nonspecific and can include fatigue, chest pain, or dyspnea [32]. Myocarditis should be suspected in patients with a history of exposure to SARS-CoV-2 or those with a diagnosis of COVID-19 that have symptoms of myocarditis and elevated levels of cardiac enzymes and inflammatory markers [32]. Distinguishing COVID-19-related myocarditis from other etiologies requires a positive SARS-CoV-2 polymerase chain reaction result from cardiac tissue. However, performing such an invasive procedure with the accompanying risk of contamination should be carefully considered. Although, several reported cases of COVID-19-related myocarditis exhibited SARS-CoV-2 in the cardiac tissue [30,33], most were diagnosed based on the clinical and radiological findings [27,29,34,35]. Cardiac imaging study may help to diagnose COVID-19 related myocarditis in these patients. The European Society of Cardiology (ESC) recommends the use of cardiac magnetic resonance (CMR) imaging for suspected acute myocarditis [36]. CMR is the best imaging modality to detect myocardial edema and myocardial injury in acute myocarditis. Regional and globally increased T2 signal intensity on short-tau inversion recovery sequence indicates myocardial edema, and regional late gadolinium enhancement (LGE) indicates myocardial injury. Emerging CMR techniques, T1 and T2 mapping, and ECV were added to the Revised 2018 Lake Louise Criteria [37]. Myocardial edema prolongs T1 and T2 relaxation time of the myocardium. Extracellular volume fraction can also detect expanded extracellular space caused by myocardial necrosis or fibrosis. Cardiac CT angiography is an alternative non-invasive imaging modality to distinguish myocarditis from coronary artery disease [36]. Fig. 2 illustrates a case of COVID-19 related myocarditis in a 21-year-old female patient. Extensive LGE with myocardial edema was noted on CMR and coronary artery disease was excluded on coronary CT angiography.

Pericardial effusion

Few cases of pericardial effusion complicated by COVID-19 have been reported [38,39]. Pericardial effusion might develop as a result of pericarditis or myo-pericarditis [40]. Farina et al. [39] reported the detection of SARS-CoV-2 in pericardial fluid. Another paper reported that pericardial fluid was exudative in COVID-19 patient with elevated lactate dehydrogenase and albumin levels [41]. At our institution, there was one case of pericardial effusion complicated by COVID-19 in an 86-year-old female patient (Fig. 3). A large pericardial effusion was noted on CT at the time of diagnosis of COVID-19 that completely resolved after treatment of COVID-19. This was presumed to be caused by myo-pericarditis. There is still little known about clinical significance of pericardial effusion in COVID-19 patients. As in our case, pericardial effusion in COVID-19 can demonstrate a self-limiting course, but there are also reported cases of life-threatening cardiac tamponade. Further research on the mechanism of development of pericardial effusion and its clin-

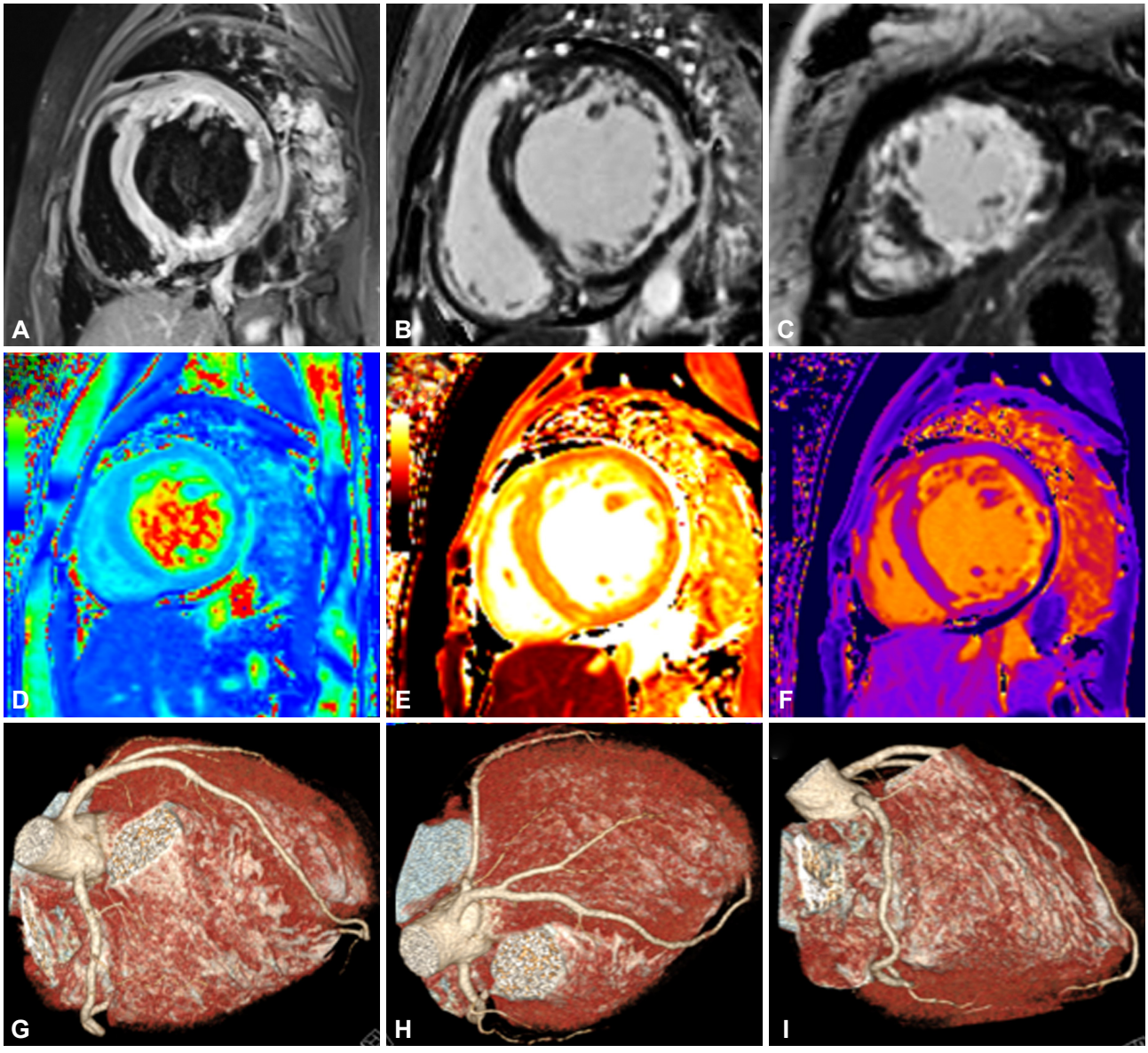

Fig. 2. COVID-19-related myocarditis in a 21-year-old female patient. Myocarditis was suspected during hospitalization because of an increase in the cardiac biomarkers Troponin I (1.26 ng/mL, reference value <0.3 ng/mL) and NT-proBNP (1929 pg/mL, reference <125 pg/mL) with worsening of shortness of breath. Cardiac MRI (3T system, Vida, Siemens) showed diffuse high signal intensity of the LV myocardium on T2 short tau inversion recovery image (A, signal intensity ratio of myocardium over skeletal muscle=2.16), reflecting myocardial edema. Late gadolinium enhanced images (B and C) showed extensive transmural enhancement of the LV myocardium. T2 mapping (D, septal wall 45 ms, reference value ~37 ms), native T1 mapping (E, septal wall 1431 ms, lateral wall 1453 ms, reference value ~1200 ms) values and extracellular volume fraction (F, septal wall 30%, lateral wall 60%, reference ~25%) were also increased. Coronary CT angiography (G, H, and I) excluded coronary artery disease. Finally, an endomyocardial biopsy revealed myocarditis. COVID-19: novel coronavirus disease 2019, NT-proBNP: N-terminal pro brain natriuretic peptide, LV: left ventricular.

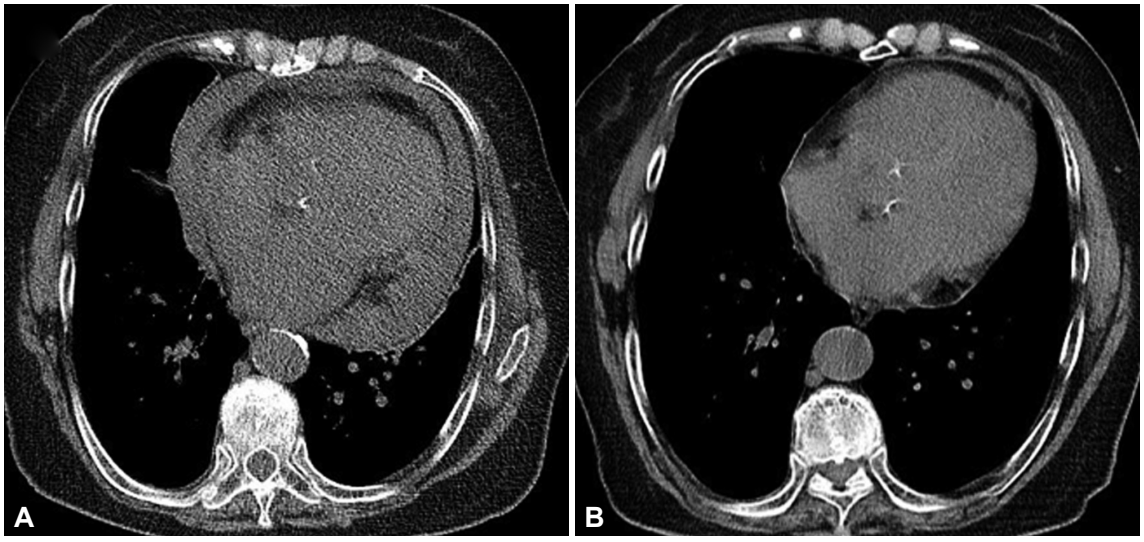

Fig. 3. Pericardial effusion in an 86-year-old female COVID-19 patient. A large pericardial effusion was noted on chest CT at the time of admission (A). Two months after discharge, pericardial effusion was completely resolved (B). COVID-19: novel coronavirus disease 2019.

ical significance in COVID-19 patient is needed.

Heart failure

Several studies have reported that heart failure is a complication of COVID-19, but its exact prevalence is unknown [42]. A cohort study from Wuhan, China reported that 23% (44/191) of COVID-19 patients had heart failure [42]. The prevalence of heart failure was significantly higher in non-survivors than survivors (52% vs. 12%, p<0.001, respectively) [42]. Although exact mechanism of acute heart failure in COVID-19 has not been identified some possible cause have been proposed [43]. Older patients with underlying cardiovascular disease, such as coronary artery disease, hypertension, diabetes, or subclinical heart disease, may progress to heart failure while being in the inflammatory state caused by COVID-19. Myocardial injury, myocarditis, stress-induced cardiomyopathy, acute illness due to systemic inflammation, cytokine storm induced by COVID-19, and sepsis may lead acute cardiac dysfunction [43]. An increased natriuretic peptide level may help to diagnose heart failure in COVID-19 patients. Additionally, transthoracic echocardiography with appropriate personal protective equipment could be considered to evaluate LV dysfunction in COVID-19 patients with symptoms of heart failure [3]. Heart failure has been observed after extensive COVID-19 related myocarditis (Fig. 2) at our institution. Globally decreased LV wall motion (LV ejection fraction 28%) and dilated LV have been seen on transthoracic echocardiography (Supplemental Video 1 in the online-only Data Supplement).

Thromboembolism

The severe inflammatory response and hypoxia caused by COVID-19 may lead to endothelial dysfunction and an increase in circulating thrombotic factors [9,44,45]. One study demonstrated that the levels of D-dimer and fibrin degradation products, fibrin, and fibrinogen were significantly higher in COVID-19 patients than in normal subjects [45]. In another study, venous thromboembolism was confirmed in 58% (7/12) of patients at autopsy and pulmonary embolism was the direct cause of death in 4 patients [46]. Among 100 COVID-19 patients who underwent contrast-enhanced CT, pulmonary thromboembolism was found in 23%, and the incidence of pulmonary embolism was significantly higher in severely unwell patients requiring mechanical ventilation and critical care admission [47]. In another study of 184 patients with severe COVID-19, the cumulative incidence of thrombotic events including pulmonary embolism, deep vein thrombosis, ischemic stroke, and myocardial infarction was 49% [48]. Pulmonary thromboembolism was the most common thrombotic event in COVID-19 patients (87%). Pulmonary embolism should be suspected in COVID-19 patients with worsening respiratory symptoms and increased D-dimer levels. Pulmonary CT angiography is the diagnostic modality of choice in these patients; however, physicians must weigh the potential risks of contamination by intravenous injection of contrast media and the expected benefits of performing CT in this population. The World Health Organization recommends that contrast-enhanced CT is considered when thromboembolic complications are suspected [49]. Fig. 4 illustrates the imaging findings of an 87-year old female with COVID-19 and pulmonary thromboembolism treated in our institution. Contrast-enhanced chest CT showed pulmonary embolism and venous thrombosis in the left internal jugular vein.

Acute coronary syndrome

The pathophysiology of ACS in COVID-19 involves plaque

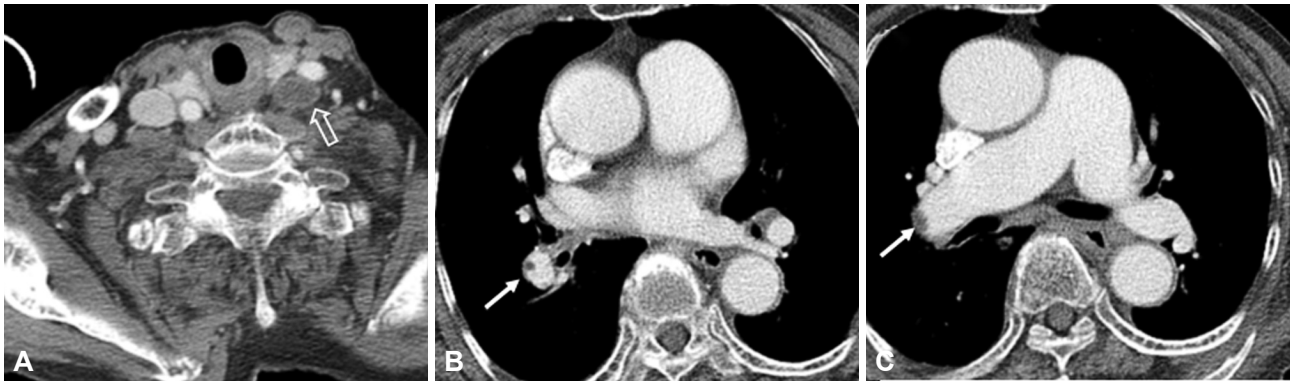

Fig. 4. Thromboembolic complications in an 87-year-old female COVID-19 patient. Contrast enhanced CT showed a thrombus in the left internal jugular vein (A, arrow) and pulmonary thromboembolism at the right main and lower lobar pulmonary arteries (B and C, arrows). COVID-19: novel coronavirus disease 2019.

instability, hypercoagulable state, microvascular dysfunction, hemodynamic change, and hypoxemia caused by severe inflammation [50,51]. The majority of ACS cases caused by COVID-19 are type 2 which result from a mismatch between oxygen supply and demand [20]. Type 1 myocardial infarction caused by plaque disruption may also be precipitated by COVID-19. Since chest pain is common in COVID-19 patients, this can lead to a delay in the diagnosis of ACS. Therefore, clinicians should maintain a high index of suspicion and rule out ACS by assessing cardiac biomarker levels and electrocardiogram findings. In case of ST-elevation myocardial infarction, primary percutaneous coronary intervention remains the therapy of choice, to be performed in a dedicated COVID-19 catheterization laboratory. In patients with non-ST-segment elevation ACS with low to intermediate risk, coronary CT angiography is the preferred non-invasive diagnostic imaging modality because it minimizes the exposure time of patients [36,52].

ARRHYTHMIA

Although the incidence and the exact mechanism of arrhythmia in COVID-19 are uncertain, various types of arrhythmias have been reported in recent studies of COVID-19 patients [53]. Myocardial injury and antiviral therapies are possible drivers of arrhythmias in COVID-19 patients [54]. Among a cohort of 137 patients in Hubei, China, 10 patients (7.3%) had palpitations [55]. In another study of 187 Chinese patients, 11 (5.9%) had malignant arrhythmias such as ventricular tachycardia and ventricular fibrillation [6]. In this study, patients with elevated troponin I level were at a significantly higher risk of developing malignant arrhythmias [6]. In a cohort of 393 patients in New York, Goyal et al. [56] found that the risk of atrial arrhythmia was significantly higher in patients who received invasive mechanical ventilation compared to those who did not (17.7% vs. 1.9%, respectively). Cardiac CT with delayed phase scanning (1 minute after contrast administration) is preferred to transesophageal echocardiography in the evaluation of thrombi in the left atrial appendage and assessment of complications of cardiac implantable devices inserted for the management of arrhythmias [36]. Moreover, since arrhythmia in COVID-19 patients may be caused by structural heart disease such as myocardial injury or myocarditis, imaging studies such as CMR can be considered to identify the cause of the arrhythmia, while weighing the risks and benefits to each patient [57,58].

ROLE OF CARDIOVASCULAR IMAGING IN COVID-19

Imaging COVID-19 patients is difficult because of the risk of transmission of the virus. Therefore, routine cardiac imaging is not recommended for COVID-19 patients and should be reserved for patients with suspected cardiovascular injury. The Society of Cardiovascular Computed Tomography and the ESC have published updated guidance on the use of cardiac CT and MRI in COVID-19 patients [36,59]. The risk of contamination during CT and CMR is considered lower than that during echocardiography. Cardiac CT angiography is the preferred noninvasive imaging tool for patients with suspected coronary artery disease, acute symptomatic valvular heart disease, pulmonary embolism, intracardiac thrombus, and for those requiring urgent structural interventions and urgent management of complications of intracardiac devices. CMR is recommended for patients with suspected acute myocarditis. Prevention of virus transmission from COVID-19 patients to healthcare workers and other patients is the foremost priority. To prevent disease dissemination during CT or MR scans, radiographers are required to wear personal protective equipment [60,61], which consists of goggles, face masks, fluid-resistant gowns, and gloves. Patients should wear a surgical mask in the scanning room, and in the case of MR scanning, the metal strip in the mask should be removed prior to arrival at the imaging suite or MR-safe masks should be used [62]. Non-contrast

enhanced CT is usually recommended in COVID-19 patient because of the increased risk of virus transmission through intravenous contrast injection. After the imaging study is completed, the CT or MR scanner and the rooms are sanitized [60]. High frequency contact surfaces should be wiped with an alcohol-based disinfectant or other environmental protection agency-approved disinfectants [61,63]. Depending on the air exchange rate, the imaging suite may need to be closed for 60 minutes to ventilate and exchange the room after sanitation [60,61]. Some hospitals use negative pressure individual isolation systems of sizes suitable for a CT scanner's gantry [64]. Given the risk of transmission, the appropriate use of diagnostic imaging in COVID-19 patients with suspected cardiovascular complications is essential.

CONCLUSION

Cardiovascular injury is a serious complication of COVID-19 that is related to fatal outcomes. Current studies are ongoing to try and elucidate the exact incidence and mechanism of cardiovascular complications in COVID-19. To improve outcomes, it is essential that clinicians are aware of the various cardiovascular manifestations COVID-19 and that they are familiar with the latest guidance on the appropriate use of cardiac biomarkers and cardiovascular imaging in suspected patients.

Supplementary Video Legends

Video 1. Transthoracic echocardiography shows globally decreased left ventricular wall motion (ejection fraction 28%) because of COVID-19 related myocarditis.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.22468/cvia.2020.00066.

Conflicts of Interest

The author has no potential conflicts of interest to disclose.

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