ABSTRACT
COVID-19 has posed an extraordinary burden on health and the economy worldwide. Patients with cardiovascular diseases are more likely to have severe illness due to COVID-19 and are at increased risk for complications and mortality. We performed a narrative literature review to assess the burden of COVID-19 and cardiovascular morbidity and mortality. Myocardial injury has been reported in 20%–30% of patients hospitalized due to COVID-19 and is associated with a worse prognosis and high mortality (~50%–60%). Proposed mechanisms of myocardial injury include inflammation within the myocardium (due to direct viral infection or cytokine storm), endotheliitis, coronary vasculitis, myocarditis, demand ischemia, plaque destabilization and right ventricular failure. The right ventricle is particularly vulnerable to injury and failure in COVID-19-infected patients, given the hypoxic pulmonary vasoconstriction, pulmonary microthrombi or pulmonary embolism. Echocardiography is an effective and accessible tool to evaluate left and right ventricular functions and risk stratify patients with COVID-19 infection. Cardiac MRI has detected and characterized myocardial injury, with changes compatible with other inflammatory cardiomyopathies. The long-term consequences of these inflammatory changes are unknown, but accumulating data will provide insight regarding the longitudinal impact of COVID-19 infection on cardiovascular morbidity and mortality.

INTRODUCTION
The COVID-19 pandemic caused by infection with SARS-CoV-2 has posed an unprecedented burden on health and the economy worldwide. Infection with SARS-CoV-2 results in a hyperactive inflammatory response accompanied by innate immune cell infiltration and hyperinflammation, especially in patients whose host immune system may not successfully eliminate the virus at disease onset.1,2 SARS-CoV-2 infection usually presents in 3 phases: first, an asymptomatic incubation period with or without detectable virus; second, a non-severe symptomatic period with detectable virus; and third, a severe respiratory symptomatic phase with high viral load.3 In the first 2 phases, the predominant immune response is native and mediated through interferons, macrophages and granulocytes and is considered a ‘protective’ phase. Once progression to the final phase happens, the adaptive immune response mediated by leucocytes other than T cells (given the lymphocytopenia often seen in severe COVID-19) and interleukins may lead to a cytokine storm and a ‘damaging’ phase.2

Similar to other viral epidemics like influenza or SARS, patients with cardiovascular diseases (CVD) are more likely to develop severe viral illness and are at increased risk for complications and mortality.3,4 A series of 44,672 confirmed patients with COVID-19 (including mild cases) reported 4.2% had underlying CVD,5 but they accounted for 22.7% of all fatalities, with a case fatality rate of 10.5%, compared with a 2.3% in the overall population. An increased risk for hospitalization, severe disease and up to 5-fold to 10-fold increase in mortality in patients with underlying CVD have been reported.1,6 which is concerning given the high prevalence of these comorbidities. Furthermore, patients with COVID-19 without underlying heart disease have subsequently developed heart failure, myocardiitis, pericarditis, vasculitis, and cardiac arhythmias.7,8 Given the public health impact of these cardiovascular complications of COVID-19 infection, we performed a narrative literature review on the topic. We searched the PubMed, Embase, Cochrane Central Register of Controlled Trials, and Cumulative Index to Nursing and Allied Health Literature databases through August 10, 2020, with no restrictions on language. Keywords of COVID-19, SARS-CoV-2, coronavirus, cardiac involvement, cardiac injury, myocarditis, endotheliitis, and right ventricle (RV) were used. Randomized clinical trials with cardiovascular data, prospective studies, systematic reviews and meta-analysis were included, and reference lists of included articles and relevant reviews were manually searched. Given the nature of this review and no direct patient contact, Institutional Review Board approval was not necessary.

COVID-19 PHENOTYPE
SARS-CoV-2 is an RNA beta coronavirus which has multiple spike glycoproteins (S) that protrude far from the viral surface. These spikes bind to ACE2 receptors on cells to facilitate virus internalization.9,10 In the renin-angiotensin
aldosterone system (RAAS), ACE2 catalyzes the conversion of angiotensin II to angiotensin 1–7, which acts as a vasodilator and exerts protective effects in the cardiovascular system. On viral entry, the ACE2 receptor may be downregulated, leading to a predominant angiotensin II effect that, in conjunction with loss of the cardioprotective effects from angiotensin 1–7, promotes inflammation, vasoconstriction (hypertension) and thrombosis. Loss of ACE2 increases the risk of heart failure and increasing ACE2 levels may prevent and even reverse the heart failure phenotype. Despite the theoretical increase in ACE2 expression among patients receiving ACE inhibitors or angiotensin receptor blockers, RAAS inhibitors do not result in increased plasma ACE2 concentrations. In a case population study from Spain, RAAS inhibitors did not increase the incidence of hospitalization, intensive care unit utilization or mortality in patients with COVID-19. In fact, their use was associated with a protective effect for hospitalization in patients with diabetes.

Multiple specialty organizations recommend continuing or starting inhibition of RAAS if clinically indicated in patients with COVID-19. Specifically, in patients with heart failure, appropriate standard heart failure medications, including RAAS inhibitors, should be considered. The overall imbalance between innate and acquired immunity, favoring macrophages and neutrophils, inappropriate type 1 interferon and lymphopenia promotes uncontrolled self-amplification of cytokine production, which drives most of the systemic inflammatory effects observed with COVID-19. It has been proposed that SARS-CoV-2 could directly infect T-lymphocytes and induce their death which may eventually lead to lymphopenia and impeded antiviral responses, favoring uncontrolled innate immune responses.

**MYOCARDIAL INJURY**

Myocardial injury—defined as elevation in troponin levels to 3 times higher of normal serum concentrations or above their 99th percentile upper reference limit—has been reported in 20%–30% of patients hospitalized due to COVID-19, with increased risk noted in patients with hypertension, coronary artery disease, heart failure or diabetes. Cardiac injury is associated with higher in-hospital mortality compared with patients without myocardial injury (50%–60% vs <9%, respectively). The prevalence of myocardial injury is particularly high among severe, critically ill and deteriorating patients with COVID-19, and patients with myocardial injury are more likely than those without to have acute respiratory distress syndrome (ARDS), need for mechanical ventilation, arrhythmias, renal failure and thrombotic events.

Elevation of plasma troponin levels does not necessarily reflect heart failure or acute myocardial infarction and has been linked to high levels of C-reactive protein, procalcitonin, natriuretic peptides and lower lymphocyte counts suggesting that inflammation severity and ventricular wall stress are linked to myocardial injury. In the setting of COVID-19, marked elevation of biomarkers informs clinicians regarding cardiovascular system involvement and overall prognosis, but does not necessarily identify acute coronary syndrome or heart failure. Meta-regression including 4189 confirmed patients with COVID-19 suggested that cardiac injury biomarker differences of severity could be explained by the presence of hypertension (p=0.030). Concordant with cohort studies, COVID-19-related cardiac injury was associated with increased mortality (summary risk ratio 3.85, 2.13–6.96; p<0.001). Both pooled high-sensitivity troponin and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels increased progressively during hospitalization in those with mortality, but these incremental changes were not seen in survivors.

Several mechanisms have been proposed for the myocardial injury. Given the studies showing higher levels of inflammatory and myocardial wall stress (C-reactive protein and NT-proBNP) in patients with myocardial injury, one of the proposed mechanisms is *interstitial inflammation within the myocardium*, either as a direct complication of COVID-19 or due to systemic response with the intense release of cytokines. *Direct viral infection* and replication in the myocytes has been proposed as another mechanism. Pathologic specimens show a spectrum of myocardial involvement, ranging from few interstitial mononuclear inflammatory infiltrates without substantial myonecrosis to viral presence within the myocytes. A postmortem study of patients who died from COVID-19 documented SARS-CoV-2 in 61.5% of the hearts, with a high viral load (>1000 copies/μg RNA) in 41% of patients. Interestingly, viral presence in the myocardium was not associated with influx of inflammatory cells in the interstitium.

COVID-19 involvement of endothelium in multiple vascular beds, including the heart, has been reported, likely because of the expression of ACE2 receptors in endothelial cells. An autopsy report has documented *endotheliitis and vasculitis*, diffusely involving small cardiac vessels and even extending into epicardial fat and interstitial spaces, without lymphocytic infiltration of the myocardium. It is suspected that pericytes are the site of SARS-CoV-2 infection, which may lead to capillary endothelial cell and microvascular dysfunction. Direct viral entry and replication in myocardium and blood vessels may enhance the risk for myocardial injury, and subsequent inflammatory response leading to *myocarditis*. It is uncertain whether this manifestation triggers a prominent immune response with acute myocarditis leading to acute decompensated heart failure, or if a more indolent response may lead to a chronic, persistent, inflammatory-driven, dilated cardiomyopathy, as has been reported for other viruses. Fulminant myocarditis has been suspected in 7% of deceased patients.

*Demand ischemia or plaque destabilization* could be another mechanism of cardiac injury, promoted by the intense systemic inflammatory response, increased sympathetic stimulation and tachyarrhythmias, all of which can destabilize underlying atherosclerotic plaques. A significant increase in inflammatory cells in atherosclerotic coronary plaques has been described during an acute systemic infection. Additionally, the increase in inflammatory cytokines activates the inflammatory cells present in atherosclerotic plaques, leading to coronary plaque destabilization and subsequent myocardial ischemia. It has been proposed that the thin-walled RV is particularly susceptible to ischemia and dysfunction in response to sudden increases...
in right-sided afterload and coronary supply/demand mismatch by microthrombi, explaining the higher prevalence of right-sided dysfunction. Hypoxia and respiratory dysfunction leading to right ventricular (RV) strain likely play a major role in the COVID-19 cardiac pathophysiology. RV dysfunction is associated with moderate to severe ARDS in more than 50% of patients and is a well-established determinant of mortality. RV failure in the setting of ARDS is thought to be related to the increased pulmonary vascular resistance (PVR) from vasoactive agents, adverse vascular remodeling, hypoxia-driven pulmonary vasoconstriction, intravascular thrombosis and vascular compression from atelectasis and edema. Moreover, the uncoupling between the RV and pulmonary circulation under positive pressure ventilation may also contribute to RV failure. The endothelial and microvascular injury seen in COVID-19 leads to potentiation of inflammation that results in disruption of vascular homeostasis by increasing coronary capillary hyper-permeability, vasospasm, myocardial perfusion defects, and subsequent ischemia, exacerbating the oxygen supply/demand, a well-described precipitant of RV dysfunction. Finally, acute myocarditis can directly involve RV myocardium. Overall, the proinflammatory cytokines can have negative inotropic effects on the RV, and in combination with the acutely elevated PVR, increase the likelihood of RV failure physiology.

MICROVASCULAR AND COAGULATION DYSFUNCTION
Pathologic specimens of lung tissue and other organs have shown evidence of microvascular thrombi and release of proinflammatory cytokines by endothelial cells. The presence of microangiopathy and thrombus predisposes to microinfarcts in the heart as well as other organs, further worsening the multiorgan dysfunction. Multiple reports document profound dysregulation of coagulation pathways evidenced by elevated plasma D-dimer and fibrin degradation products, prolonged prothrombin time, and thrombocytopenia in patients with COVID-19. Pulmonary vasculitis with microangiopathic thrombi formation, together with the overall prothrombotic state, promotes pulmonary embolism, which worsens hypoxemia due to right to left intrapulmonary shunting. The resultant increased afterload to the RV due to microthrombi and hypoxia, together with hyperactive coagulation and inflammatory pathways, likely contributes to RV failure physiology.

The endothelial disease, either by direct virus involvement or secondary to the cytokine hyperactivity, favors a prothrombotic state in different organs. The impaired endothelial function, manifested as decreased barrier function (basement membrane degradation and sloughing), antifibrinolytic properties, increased expression of leucocyte adhesion molecules, vasoconstriction and pro-oxidant molecule expression have been proposed as potential mechanisms for the multiorgan affection seen in COVID-19. There are multiple reports of worsening outcomes secondary to microvascular involvement in different organs, manifesting as ischemic strokes, deep vein thrombosis and pulmonary embolism, arterial and placental thromboembolism.

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Atrial and ventricular wall inflammation is a substrate for arrhythmias, and atrial fibrillation or ventricular arrhythmias have been reported frequently in COVID-19 cardio-myopathies. In a cohort of hospitalized Chinese patients with COVID-19, shock or arrhythmias (unspecified) were present in 8.7% and 16.7% of cases, respectively. Not surprisingly, arrhythmias were more common in critically ill patients. Other reports have shown ventricular tachycardia or ventricular fibrillation in 5.9% of hospitalized patients with COVID-19, all in patients with documented cardiac injury. In a cohort of 700 hospitalized patients with COVID-19 in the USA, 9 (1.3% of the cohort) cardiac arrests—8 with non-shockable rhythms and 1 with torsades de pointes—were documented. Also noted were 25 incident atrial fibrillation events, 9 clinically significant bradyarrhythmias and 10 non-sustained ventricular tachycardia events. No association was seen between bradyarrhythmias or non-sustained ventricular tachycardia and in-hospital mortality.

Potential mechanisms for arrhythmia include inflammation, ischemia, hypoxia and decompensated heart failure, all of which promote myocardial injury. Although not recommended, treatment with hydroxychloroquine and azithromycin may result in QT prolongation and increase the potential risk of arrhythmias. Table 1 summarizes the cardiac manifestations of COVID-19.

**Table 1 Cardiac manifestations of COVID-19**

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Proposed mechanisms</th>
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<tbody>
<tr>
<td>Myocardial injury (troponin elevation)</td>
<td>► Intestinal myocardial inflammation (direct or due to systemic intense cytokine storm).</td>
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<tr>
<td></td>
<td>► Direct viral infection and replication.</td>
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<tr>
<td></td>
<td>► Endothelitis and coronary small vessel vasculitis.</td>
</tr>
<tr>
<td></td>
<td>► Myocarditis.</td>
</tr>
<tr>
<td></td>
<td>► Demand ischemia/plaque rupture (ie, acute coronary syndrome).</td>
</tr>
<tr>
<td></td>
<td>► Right ventricular ischemia.</td>
</tr>
<tr>
<td>Heart failure (predominantly right ventricular failure)</td>
<td>► Myocarditis.</td>
</tr>
<tr>
<td></td>
<td>► Right ventricular failure.</td>
</tr>
<tr>
<td></td>
<td>► Hypoxia.</td>
</tr>
<tr>
<td></td>
<td>► Cytokine storm, negative inotropic effect.</td>
</tr>
<tr>
<td></td>
<td>► Pulmonary vasculitis and microthrombi.</td>
</tr>
<tr>
<td></td>
<td>► Pulmonary thromboembolism.</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>► Inflammation, ischemia, hypoxia, decompensated heart failure.</td>
</tr>
</tbody>
</table>
ROLE OF IMAGING
Systematic echocardiographic evaluation of hospitalized patients with COVID-19 showed that RV dilatation and dysfunction was the most common abnormality (observed in 39% of patients), followed by left ventricular diastolic dysfunction (16%) and systolic dysfunction (10%); a normal echocardiogram was present in only 32% of patients. In the 20% of patients who experienced clinical deterioration, follow-up echocardiography showed RV function worsening, followed by left ventricular systolic and diastolic dysfunctions. In another cohort of hospitalized patients with COVID-19, RV dilatation by echocardiography was present in 31%. Not surprisingly, patients with RV dilatation had higher incidence of RV hypokinesis and moderate to severe tricuspid regurgitation than those without RV dilatation and higher mortality (41% vs 11%, p=0.001). The mechanism of RV dilatation is likely multifactorial, including pulmonary thrombotic events (macroangiopathic or microangiopathic), hypoxemic pulmonary vasoconstriction, cytokine damage, and possible direct viral damage.

Routine echocardiographic measures of RV function, such as tricuspid annular plane excursion and RV fractional area change, are significant predictors of mortality in patients with COVID-19.41,63 with reduced RV longitudinal strain—measured using two-dimensional echocardiography with speckle tracking—shown to be a better predictor of mortality.42

MRI is feasible and safe to perform to detect and characterize cardiac injury non-invasively in patients with suspected myocarditis. In a small cohort of recovered patients with COVID-19 with persistent cardiac symptoms, late gadolinium enhancement (LGE) showed myocardial edema and fibrosis in 58%.66 Moreover, in another cohort of recovered patients with COVID-19 without active cardiac symptoms,64 cardiac MRI showed abnormalities in 78% of patients, including lower left ventricular and RV ejection fraction, higher left ventricle volume and mass, and elevated native T1 and T2 measures. The most prevalent abnormalities were myocardial inflammation (defined as abnormal native T1 and T2 relaxation times), detected in 60% of patients, followed by regional scar and pericardial enhancement. Elevated high-sensitivity troponin plasma levels were detectable in 71% of patients of this series, which is much higher than reported in other series. Patients with severe abnormalities (markedly elevated high-sensitivity troponin levels, native T1 and T2 relaxation times in the upper tertile, LGE, and left ventricular ejection fraction less than 50%) underwent endomyocardial biopsy, revealing active lymphocytic inflammation with no evidence of any viral genome. Patterns of non-ischemic myocardial involvement of LGE, which can be observed in patients with acute or healed myocarditis, have a strong association to adverse outcomes.65,69

Increased native T2 relaxation times are specific for edema, whereas increased native T1 relaxation times may be due to myocardial fibrosis and/or edema. Hence, patients with an increase in both have an active inflammatory process, whereas those with only increased native T1 relaxation times have healed with residual myocardial injury.71 Importantly, ventricular volumes and function—by MRI or echocardiography—are less sensitive and less predictive markers of myocardial involvement compared with tissue characterization with LGE.61,71 Table 2 displays a summary of the imaging findings on echocardiographic and MRI evaluation of patients with COVID-19.

Similar to other disease processes, the optimal imaging modality will depend on the availability and the clinical setting. Appropriate use criteria still prevail, and per guidelines, echocardiography is recommended as first-line imaging in many of the described COVID-19 cardiac manifestation.72 Furthermore, echocardiography is widely available, it allows evaluation, quantification and longitudinal trends of chamber sizes, function and pericardial effusion.

The intrinsic limitations of echocardiography, related to poor acoustic windows and suboptimal tissue characterization, are likely exacerbated in critically ill patients. It has been suggested that echocardiography carries a higher risk of transmission of COVID-19 during the examination compared with cardiac MRI.73

Cardiac MRI is the only non-invasive modality that allows for tissue characterization and quantification of the injury to provide prognosis. Widespread use is limited by the low availability, high cost, long scan times, use of contrast and intrinsic patient’s factors (ability to hold breath while hypoxic, claustrophobia, metallic implants, arrhythmias, mechanical ventilation). It has been proposed that an appropriate timing for MRI would be at the time of transfer to a subintensive ward, for prognostication based on LGE.72

Table 2  Echocardiographic and MRI findings in COVID-19

<table>
<thead>
<tr>
<th>Imaging modality</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiogram</td>
<td>▶ RV dilatation and dysfunction.60</td>
</tr>
<tr>
<td></td>
<td>▶ Reduced tricuspid annular plane excursion.60</td>
</tr>
<tr>
<td></td>
<td>▶ Reduced RV fractional area change.60</td>
</tr>
<tr>
<td></td>
<td>▶ LV diastolic dysfunction.39</td>
</tr>
<tr>
<td></td>
<td>▶ LV systolic dysfunction.24</td>
</tr>
<tr>
<td></td>
<td>▶ Tricuspid regurgitation (associated to RV dilatation).59</td>
</tr>
<tr>
<td></td>
<td>▶ Reduced RV longitudinal strain, &lt;-23.60</td>
</tr>
<tr>
<td>MRI</td>
<td>▶ Late gadolinium enhancement and increased extracellular volumes.51,64,66</td>
</tr>
<tr>
<td></td>
<td>▶ Lower LV and RV ejection fractions.50,64</td>
</tr>
<tr>
<td></td>
<td>▶ Regional scoring and other patterns of non-ischemic myocardial disease.64,67</td>
</tr>
<tr>
<td></td>
<td>▶ Pericardial and pericardial enhancement.66</td>
</tr>
<tr>
<td></td>
<td>▶ Increased global and septal native T1.61,64</td>
</tr>
<tr>
<td></td>
<td>▶ Increased global and septal native T2.61,64</td>
</tr>
</tbody>
</table>

LV, left ventricle; RV, right ventricle;
An important consideration in the setting of COVID-19 is the risk of exposure of healthcare workers. The protection of nurses, technicians and doctors with personal protection equipment involved in the imaging of patients with COVID-19 is of utmost importance, as well as sanitization of all the related machines, probes and rooms after each examination.72

CARDIAC TRANSPLANT RECIPIENTS
COVID-19 infection has been reported in a heart and kidney recipient, 25 days after transplants, manifesting with atrial flutter and allograft dysfunction without documented rejection. Corticosteroid and tacrolimus therapy were continued, and the patient recovered cardiac function a week after mechanical ventilation.73 Other case reports67 77 and a survey78 from China have not shown a worse outcome of COVID-19 in heart transplant recipients.

A single-center cohort of 28 heart transplant recipients in New York with confirmed diagnosis of COVID-19 was published, with a median time to transplant of 8.6 years and a high proportion of comorbidities (diabetes in 61%, hypertension in 71%, allograft vasculopathy in 25% of patients).79 A quarter of patients required mechanical ventilation, three-quarters of them had evidence of myocardial injury and a quarter of them died. Immunosuppression was reduced in most patients, mostly discontinuation of mycophenolate mofetil and reduction of calcineurin inhibitor dose. Of note, asymptomatic heart transplant recipients were not screened, so these outcomes reflect the incident outcomes among symptomatic transplant patients. To date, the effect of immunosuppression on the natural history of COVID-19 remains to be determined and a close monitoring of this patient population is warranted, particularly when reintroducing preinfarction regimens.

Finally, in accordance with International Society for Heart and Lung Transplantation guidance, cardiac transplant candidates with active COVID-19 infection are made inactive on the waitlist.80 For cardiac transplant candidates who recover from COVID-19, criteria for heart transplantation currently include waiting at least 14 days after initial COVID-19 diagnosis and documenting 2 successive negative PCR-based tests at least 48 hours apart.

LONG-TERM CARDIAC CONSEQUENCES OF COVID-19
As the pandemic continues, there will be an expanding population of ‘recovered’ patients. There have been reports of up to 60% of persistent systemic symptoms months after recovery,81 and whether local inflammation is diminished is uncertain. Long-term complications and sequelae can have a major impact on public health if the immune hyperactivation leads to tissue fibrosis and microangiopathy, resulting in cardiomyopathy for those with heart involvement.1 During the original SARS epidemic, increased cardiometabolic risk was reported up to 12 years later in recovered patients, postulated to be related to steroid treatment and RAAS imbalance.82 Furthermore, the development of pulmonary fibrosis in patients who develop ARDS could lead to pulmonary hypertension and cor pulmonale in the subsequent years.

It is too early to determine the long-term health consequences of the inflammatory changes seen on cardiac MRI studies, even in asymptomatic patients. It is particularly worrisome that several of the abnormalities described in MRI studies are features that have been related to worse outcome in inflammatory cardiomyopathies.61 83 84

FUTURE DIRECTIONS
There are major differences in the populations included in the COVID-19 studies, ranging from asymptomatic to severely decompensated patients. Inclusion of COVID-19 status on existing cardiac registries, as well as further details on biomarkers that reflect cardiac damage, will provide more information of the effect on COVID-19 on CVD, its therapy and the most effective way to stratify patients. Further detection of COVID-19 by whole-genome sequencing, PCR and pathologic specimens from myocardium may help identify SARS-CoV-2 presence in the myocardium. Longitudinal studies will elucidate if chronic cardiovascular damage is a feature of COVID-19 infection, similar to what has been described in SARS infection.11

COVID-19-endotheliitis may explain the systemic impaired microcirculatory function in various vascular beds, including the heart, and their clinical consequences. This provides the rationale for randomized trials evaluating therapies that promote endothelium stabilization such as anti-inflammatory or anticytokine drugs, ACE inhibitors, and statins, particularly in patients with underlying endothelial dysfunction.29

Reverting the immune response imbalance to favor adaptive mechanisms as opposed to self-perpetuating cytokine storm may be the holy grail of COVID-19 therapy. Potential therapies appropriately timed in the natural history of the disease could prevent rapid cardiac decompensation as well as the long-term adverse consequences.

SUMMARY
There are extensive clinical and epidemiological data suggesting that COVID-19 is associated with myocardial injury. The pathophysiology of COVID-19 places the RV at higher risk of involvement and complications and has prognostic implications. Accumulating data will provide additional insight regarding long-term consequences of COVID-19 infection on cardiovascular morbidity and mortality.

Correction notice Dr. Mukherjee’s ORCID ID has now been added.

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