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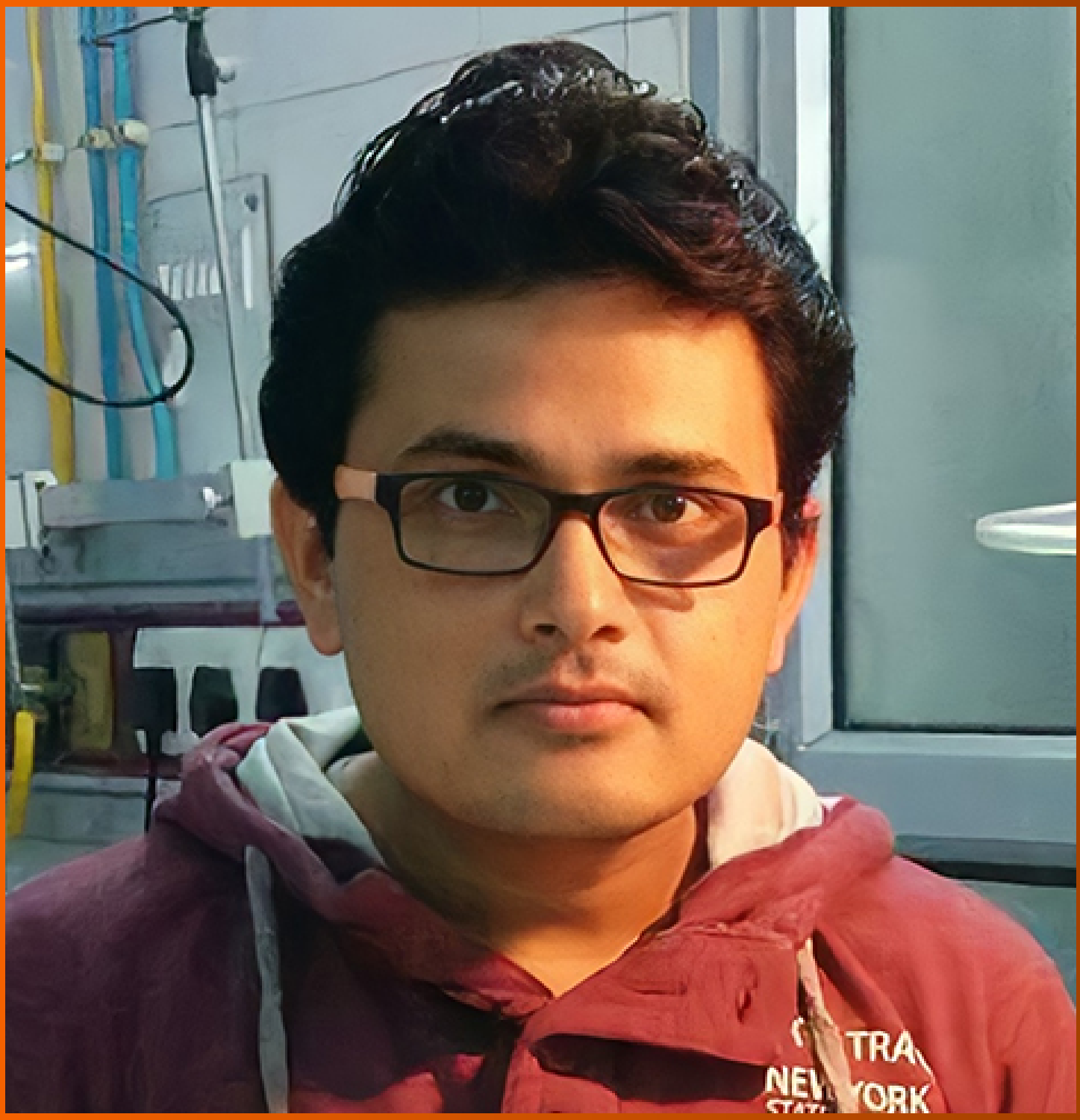

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LETTER TO THE EDITOR

"Heart failure in COVID-19 patients: Critical care experience": A letter to the editor

Vasiliki Tsigkou, Gerasimos Siasos, Evangelos Oikonomou, Evanthia Bletsa, Manolis Vavuranakis, Dimitris Tousoulis

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Abstract

Coronavirus disease 2019 (COVID-19) is associated with poor cardiovascular outcomes in patients with heart failure (HF) of all categories of ejection fraction (EF), but mainly in patients with HF with reduced EF. Moreover, cardiac transplant patients exhibit worse cardiovascular prognosis, high mortality, and more admissions to the intensive care unit. In general, COVID-19 seems to deteriorate the clinical status of HF and favors the development of acute respiratory distress syndrome and multiorgan failure, especially in the presence of cardiovascular comorbidities such as diabetes mellitus, kidney dysfunction, and older age. COVID-19 may induce new-onset HF with complex mechanisms that involve myocardial injury. Indeed, myocardial injury comprises a large category of detrimental effects for the myocardium, such as myocardial infarction type 1 or type 2, Takotsubo cardiomyopathy, microvascular dysfunction and myocarditis, which are not easily distinguished by HF. The pathophysiologic mechanisms mainly involve direct myocardial damage by severe acute respiratory syndrome coronavirus 2, cytokine storm, hypercoagulation, inflammation, and endothelial dysfunction. The proper management of patients with COVID-19 involves careful patient evaluation and ongoing monitoring for complications such as HF.

Key Words: Heart failure; COVID-19; Prognosis; Intensive care unit; New onset heart failure; Ejection fraction

Core Tip: Coronavirus disease 2019 poses a serious threat to patients with pre-existing heart failure (HF) and might induce new-onset HF in hospitalized patients, with complex mechanisms that involve myocardial injury. Cytokine storm, described as excessive inflammation and coagulation, results in microvascular dysfunction, myocardial ischemia and myocarditis, which might not be easily distinguishable from HF. Patients with advanced HF, such as those with reduced ejection fraction, exhibit worse cardiovascular outcomes. Treatment should take into consideration patient-specific characteristics and includes a thorough cardiologic assessment along with obtainment of evidence following published guidelines.

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TO THE EDITOR

We read with interest the systematic review of John *et al*[1], who presented the interaction between coronavirus disease 2019 (COVID-19) and heart failure (HF) from a critical care perspective. After discussing evidence from 26 observational studies, the authors concluded that patients with HF have higher mortality during hospitalization for COVID-19, as well as more complications and admissions to the intensive care unit (ICU)[1]. Furthermore, they found that patients with HF with reduced ejection fraction (HFrEF) exhibited worse outcomes in comparison to patients with HF with mildly reduced ejection fraction (HFmrEF) and with preserved EF (HFpEF)[1].

Patients with HF and COVID-19 develop serious complications, according to the literature; these include severe hypotension, acute respiratory distress syndrome (ARDS), and death[2]. This comes in accordance with the authors' conclusions that HF is a risk factor for COVID-19 and that patients with HF might require hospitalization or develop more complications post hospitalization in ICU, possibly due to an additional organ injury[1]. Patients with HF often need mechanical ventilation and develop venous thromboembolism, sepsis, acute kidney injury, and stroke[3]. In clinically unstable patients with COVID-19 recommendations suggest the discontinuation of chronic cardioprotective medications, such as angiotensin-converting enzyme (ACE) inhibitors or the angiotensin receptor-neprilysin inhibitor due to hypotension[4]. Among the literature there is uncertainty about the safety of these drugs in patients with HF since severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) binds to the ACE2 receptor and administration of these regimens increase the expression of ACE2 in the heart[5,6]. Several clinical trials are under progress, nevertheless, the current recommendation is to continue these drugs in clinically stable patients and in infected patients at risk of complications[6].

Heart transplant patients with comorbidities exhibit poorer cardiovascular outcomes and a need for ICU therapeutic modalities[7]. John *et al*[1] have also resulted in this conclusion, although the prognosis of critically ill heart transplant patients was, according to them, somewhat similar to the critically ill non-heart transplant patients. As mentioned by the authors, patients with HFrEF and COVID-19 have a poorer overall prognosis[1]. Indeed, COVID-19 is linked to poor prognosis of patients with advanced HFrEF, which is reflected by the need for inotropes and/or an intra-aortic balloon pump, increased incidence of lethal arrhythmias and/or cardiogenic or septic shock, and the need for transplantation[8]. However, evidence from the literature indicates that HFpEF might also be a risk factor for adverse complications, as well as a consequence of COVID-19 due to direct myocardial damage, which highlights the need for proper follow-up care of the infected patients[9,10].

COVID-19 may worsen myocardial injury in patients with HF due to the release of pro-inflammatory cytokines, the so-called 'cytokine storm'[11]. On the other hand, COVID-19 might ignite *de novo* left ventricular dysfunction posthospital admission[2]. Indeed, the risk of *de novo* HF post hospital admission, according to the authors, is greater, especially for patients who have been admitted to the ICU[1]. The diagnosis of *de novo* HF is challenging, since patients might suffer from subclinical myocarditis, sepsis-induced cardiomyopathy, Takotsubo cardiomyopathy, or subclinical ischemia[12,13]. According to the authors, in most of the studies cardiac injury was defined as the increase in cardiac troponin I > the 99th percentile upper reference limit or new electrocardiography/echocardiography findings; however, not all the studies reported strict definitions about chronic and *de novo* HF[1]. Actually, symptoms of COVID-19 might be similar to HF, and pneumonia and pulmonary edema might coexist, thereby complicating the diagnosis of both entities[14]. Interestingly, this comes in accordance with the authors conclusions about the diagnostic difficulties among patients with severe ARDS due to

COVID-19 and acute decompensation of HF[1].

COVID-19 induces direct and indirect injury in the myocardium *via* various mechanisms that involve excessive inflammation, hypercoagulation, endothelial dysfunction, and sympathetic system activation [15]. Myocardial injury in patients with COVID-19 is mediated by ischemic and non-ischemic mechanisms, which lead to different clinical consequences and therapeutic implications[16]. SARS-CoV-2 binds to human cells on the ACE2 receptor, which is overexpressed in patients with cardiovascular diseases and exerts harmful effects through direct inoculation of the myocardium[17]. Moreover, the virus stimulates an immune response, which involves T lymphocytes and cell-mediated cytotoxicity; these mechanisms may be associated with the induction of myocarditis post-infection[18]. Myocarditis might present as acute HF in serious cases and diagnosis must be carried out with considerations of findings from medical history-taking, laboratory examinations, electrocardiograms, echocardiography, and cardiovascular magnetic resonance studies; however, a definite diagnosis also involves endomyocardial biopsy, which is not routinely performed[13].

On the other hand, SARS-CoV-2 has been implicated in cardiac ischemia of several types[19]. The imbalance between oxygen supply and demand is reflected by the increase in cardiac troponins and reflects type 2 myocardial infarction (MI) ischemia, which is a common characteristic of pneumonia due to hypotension and blood hypoxemia, especially in patients with pre-existing coronary heart disease [19]. Also, type 1 MI might be the result of pre-existing coronary plaques that become unstable due to the proinflammatory and procoagulant states of the infection[20]. Additionally, the virus induces microvascular dysfunction in patients through endothelial dysfunction; in fact, proinflammatory biomarkers and the development of microthrombi may induce endothelial dysfunction at the level of microcirculation[21]. Lastly, there is evidence that acute coronary microvascular dysfunction may result in Takotsubo syndrome in patients with COVID-19 and especially among those with pre-existing comorbidities, but the specific mechanisms are under investigation[22].

Great effort is needed in order to improve our understanding of the therapeutic needs of patients with HF and COVID-19[23]. Lockdown policies might have reduced visits to general practitioners and have led to lower rates of diagnosis of heart disease, which could then result in more *de novo* HF diagnoses[24]. Targeting the cytokine storm with anti-inflammatory medications such as corticosteroids has been linked to decreased morbidity and mortality from virus infection[25]. On-going inflammation is also present in survivors of COVID-19 infection and poses a great risk for the development of HF, indicating the need for novel therapeutic advances[25]. The development of myocardial injury following COVID-19 infection and specifically of *de novo* HF might result in more hospitalizations and higher mortality; therefore, understanding the pathophysiology of COVID-19 is the cornerstone for therapeutic success[26]. This comes in accordance with the authors conclusions about the need of future studies in order to elucidate the pathophysiology of the complex effects of COVID-19 in the heart[1]. The management of patients with COVID-19 and prior or *de novo* acute HF should be similar and identify at an early stage possible complications, along with the treatment of oxygenation abnormalities, bleeding events and arrhythmias[27]. A detailed cardiac assessment of the structural and functional characteristics of the infected patients should be performed in order to identify the acute or worsening function of the heart[27]. Moreover, guideline-directed treatment should be continued in patients with HF according to their clinical status, irrespectively of COVID-19[26]. The increase of our knowledge from the on-going studies as well as the course of the pandemic might provide a more robust evidence for the management of the patients[26].

FOOTNOTES

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