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CHEST

What We Might Find If We Only Looked

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In this issue of *CHEST*, Doyen et al[1] assessed cardiac function in 43 patients with coronavirus disease 2019 (COVID-19). Although it is not the first study to evaluate the heart in patients with COVID-19, it is unique in performing serial assessments in consecutive patients. Prior cohort studies reported cardiac dysfunction in more than one-half of studied patients with COVID-19.[2,3] Although larger, those studies were susceptible to ascertainment bias, because they relied on clinically obtained biomarkers, echocardiograms, and ECGs and often performed these assessments late in the hospital course. Although smaller, the work of Doyen et al[1] is the first study to suggest the true prevalence and time course of cardiac dysfunction among critically ill patients with COVID-19.

Despite confirmation that cardiac injury is common among critically ill patients with COVID-19, it is unknown whether these abnormalities are intrinsic to COVID-19 or if these are simply the manifestations of critical illness. By way of comparison, many clinicians were concerned with reports of thrombosis in up to 30% of critically ill patients with COVID-19.[4] These rates seem high, until one considers that higher rates have been observed in comparably critically ill patients with sepsis or ARDS from H1N1 virus, even while receiving prophylactic anticoagulation.[5,6] Similarly, alarming reports of the high rates of ICU-acquired weakness and post-ICU syndromes that are seen in patients with COVID-19 may not identify novel physiologic conditions when one recognizes that these unfortunate complications are rather common in critically ill patients. Although Doyen et al[1] report that one-half of the critically patients with COVID-19 have cardiac dysfunction, this observation is consistent with observations among patients with sepsis or ARDS from H1N1 virus.[7,8]

FOR RELATED ARTICLE, SEE PAGE 1974

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Abnormalities increasingly are recognized when assessments are more common. We know that tachyarrhythmia is common in critically ill patients because we continually assess heart rhythm. Increasing biomarker testing has resulted in recognition that mild troponin elevations are common in critically ill patients. Echocardiography is performed increasingly in critically ill patients, which may increase detection of cardiac dysfunction. Additionally, as our sophistication with imaging grows, so does our ability to recognize subtle abnormalities. Cardiac abnormalities are recognized more easily on echocardiography compared with pulmonary artery catheter.[7,9] Myocardial strain imaging may sometimes detect subtle cardiac dysfunction that is otherwise missed in conventional echocardiography. However, cardiac assessment in critically ill patients remains challenging, in that many of our sophisticated measurements are affected by loading conditions. Many echocardiographic abnormalities, especially left ventricular diastolic and right ventricular assessments, can change substantially with interventions common in the ICU, including receipt of mechanical ventilation, vasopressors, and fluids.[8,10] An additional challenge in interpreting right ventricular dysfunction in this population is that disease severity is often associated with increased right ventricular afterload through hypoxic vasoconstriction and application of positive pressure to the airways. These changes in preload or afterload may alter echocardiographic parameters without necessarily affecting contractility, thereby complicating assessment of cardiac injury. We should recognize that echocardiographic abnormalities may conflate abnormal loading states with myocardial injury. It is possible that some proportion of the abnormal conditions of the patients studied by Doyen et al[1] may be demonstrating a normal physiologic response to

severe loading conditions. Future investigations of cardiac function would benefit from accounting for loading conditions.

Another challenge with ascertaining cardiac dysfunction in critically ill patients is that many patients lack premorbid information. In those patients, it is unknown whether they had underlying heart dysfunction. However, in the serial assessments performed by Doyen et al[1] at least 20% of patients experienced the development of cardiac dysfunction after initially normal function. One interpretation is that cardiac dysfunction was not an underlying precondition but developed later. It is also possible that the newly observed cardiac abnormalities were attributable to increased vasopressors, worsened lung disease, higher ventilator pressures, or other factors that might affect loading conditions.

Whether cardiac dysfunction causes worse outcomes in critically ill patients or whether severe critical illness causes cardiac dysfunction is unclear. The cytokine and catecholamine surges that accompany critical illness often depress the myocardium and often result in end-organ hypoperfusion, microthrombosis, and hypoxemia, which further injure the heart and create a vicious circle.[11] Trying to untangle causality in a complex system using crude tools is difficult. Consequently, it is premature to opine on whether interventions to improve cardiac function in critically ill patients will yield improved outcomes. Yet, many intensivists are not assessing cardiac function routinely in critically ill patients . In ARDS, we frequently monitor several ventilator parameters, such as tidal volume, compliance, pressures, and flow rates. Conversely, echocardiography is noticeably underutilized in those patients, despite one-third of them having cardiac abnormalities. Further, what assessments are done are often simple qualitative assessments, devoid of hemodynamic quantification that might be of use to the intensivist in guiding therapy. These echocardiographic assessments might be performed serially, much as an intensivist might assess blood gases serially. As demonstrated by Doyen et al,[1] the cardiac function of patients with ARDS changes with a second assessment a few days later. In some severely ill patients, there could be value in multiple assessments in a single day.

It is uncertain whether COVID-19 is a novel cause of cardiac injury. Myocarditis and pericardial effusions presumably could be attributed to inflammation or viremia. Most of the other cardiac abnormalities that were observed are common in critically ill patients, although many intensivists fail to ascertain how common. During this pandemic, much of the medical community has embraced anecdote over data, relied on data plagued with bias, or failed to contextualize findings. Doyen et al[1] are commended for not only providing high-quality data but also interpreting these data in the appropriate context. With the growing utilization of critical care echocardiography, we expect that larger multicenter observations that will incorporate loading conditions will confirm and expand on their initial findings. It is unknown whether such data might guide therapy, but it is remarkable to think what we might find if we only looked.

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