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Widespread myocardial dysfunction in COVID-19 patients, detected by myocardial strain imaging using 2-D speckle-tracking echocardiography.

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Commentary

Two-dimensional speckle tracking echocardiography (2-D STE) is a quantitative method to assess global and regional myocardial function and has been proved to be a sensitive tool to detect subclinical cardiac dysfunction in pathological conditions as hypertension, diabetes and coronary artery disease [1,2]. By using this method, Li et al. evaluated the cardiac function in patients with COVID-19.

Since December 2019, coronavirus disease 2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has spread swiftly and been a worldwide pandemic now. Other than respiratory distress, accumulative evidences suggest that cardiac injury is a common condition and is associated with poor short-term prognosis in COVID-19 patients [3-5]. The incidence of myocardial injury in general COVID-19 patients was up to 20%-30% as reported by multiple studies [3-8]. However, cardiac injury in these studies was primarily proved by increased biomarkers such as troponin I. The population-based imaging data of cardiac involvement is lacking due to the limitation of accessing to echocardiography and/or MRI during the pandemic situation of COVID-19.

In this issue, Li et al. provided the first echocardiographic evidence of cardiac abnormalities in a cohort of consecutive COVID-19 patients [9]. Of great interests and clinical significance, this report found the prevalence of cardiac dysfunction as evidenced by strain analysis using 2-D speckle tracking echocardiography (2-D STE) was much higher than that reported in previous studies. Strain analysis by 2-D STE has been proved to be a sensitive tool to detect subclinical cardiac dysfunction in pathological conditions as hypertension, diabetes and coronary artery disease. The results revealed a reduction of global longitudinal strains (GLS) in 78.3% of general patients and 98% of severe patients, which was much higher than that detected by troponin I [9]. A recent study retrospectively performed strain analysis in 40 patients who were from 60 patients with available echocardiographic scans out of 589 patients and found 32 (80%) had reduced GLS [10]. Two recent MRI studies showed that 58%-78% of patients had abnormal CMR findings in patients recently recovered from COVID-19 illness [11,12]. Therefore, cardiac involvement is more likely a widespread phenomenon in COVID-19 patients, although many may only present as subclinical cardiac dysfunction. Given the large number of COVID-19 survivors and the high prevalence of cardiac injury, ongoing investigation of the long-term cardiovascular consequences of COVID-19 should be a priority of future research.

Currently, the pathogenesis of cardiac injury in COVID-19 remains controversial. The proposed mechanisms include myocarditis, systemic inflammation, interferon mediated immune response, coronary plaque destabilization, and hypoxia [13,14]. The traditional TTE features are nonspecific to myocarditis. The study by Li et al. demonstrated that the reduction of GLS was predominantly in the subepicardium than subendocardium, a feature consistent with imaging pattern of myocarditis, suggesting probable involvement of myocarditis in COVID-19 [9]. Moreover, changes in indices of myocardial strain were correlated with indices of inflammatory markers and hypoxia. It suggests partly secondary nature of myocardial dysfunction in COVID-19 to systemic inflammation and hypoxia [9]. The underlying mechanism of myocardial injury in COVID-19 is another subject of ongoing investigation.

Conclusion

In conclusion, this commentary highlights the high prevalence of subclinical cardiac dysfunction in COVID-19 and future research is required to reveal the long-term outcome of COVID-19 patients with overt or subclinical cardiac dysfunction and the underlying mechanisms, which is the rationale to initiate appropriate cardio-protective treatments.

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