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

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Introduction

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. The incidence of myocardial injury in general COVID-19 patients was up to 20-30% as reported by multiple studies. 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.

Description

In this issue, Li et al. provided the first echocardiographic evidence of cardiac abnormalities in a cohort of consecutive COVID-19 patients. 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. 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. Two recent MRI studies showed that 58-78% of patients had abnormal CMR findings in patients recently recovered from COVID-19 illness. 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. The traditional TTE features are nonspecific to myocarditis. The study by Li. et al. demonstrated that the reduction of GLS was predominantly in the sub epicardium than sub endocardium, a feature consistent with imaging pattern of myocarditis, suggesting probable involvement of myocarditis in COVID-19. 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. The underlying mechanism of myocardial injury in COVID-19 is another subject of ongoing investigation.

Conclusion

In conclusion, this study 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. The preferential alteration of strains in the sub epicardium supports possible myocarditis as the underpinning cardiac pathology in COVID-19. Likewise, inflammatory storms and hypoxemia may be important mechanisms leading to cardiac injury in COVID-19 patients.

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