Doppler Echocardiography

The evaluation of myocardial performance index in patients with COVID-19: An echocardiographic follow-up study

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Introduction: The epidemic of pneumonia caused by a new coronavirus rapidly spread all over the world. World Health Organization called the condition as coronavirus disease 2019 (COVID-19). COVID-19 has become a life-threatening public health emergency internationally. COVID-19 mostly presents by respiratory tract symptoms including fever, dry cough, and dyspnea. The disease progression causes pneumonia and acute respiratory distress syndrome. Pathophisyology of cardiovascular effects of COVID-19 have not been well known yet. Myocardial dysfunction may occur in cytokine-originated immune reactions. Myocardial performance index (MPI) is a feasible parameter that reflects systolic and diastolic cardiac functions.

Purpose: We aimed to evaluate the MPI in patients with COVID-19.

Methods: The study consisted of 40 patients diagnosed with COVID-19 who had mild pneumonia and had not needed intensive care treatment. Transthoracic echocardiographic examination was performed in all patients at the acute stage of infection and after clinical recovery. The average time interval between the baseline and recovery echocardiography exam was about 28 ± 3,4 days. Blood samples were studied on day 0 and on days 7, 14, 21, and 28. Immunofluorescence assay was used for COVID-19 antibody titers. Respiratory secretions were sent for RT-PCR tests.

Results: The mean age was 54 ± 11 years (male 26 (65%)). Statistically significant higher MPI (0.56 ± 0.09 versus 0.44 ± 0.07, $p < 0.001$), longer isovolumic relaxation time (112.3 ± 13.4 versus 91.8 ± 12.1ms, $p < 0.001$), longer deceleration time (182.1 ± 30.6 versus 161.5 ± 43.5ms, $p = 0.003$), shorter ejection time (279.6 ± 20.3 versus 298.8 ± 36.8ms, $p < 0.001$) and higher E/A ratio (1.53 ± 0.7 versus 1.22 ± 0.4, $p < 0.001$), were observed during acute period of infection compared to ones after clinical recovery. Compared with basal values, no significant change in left ventricular systolic ejection fraction was observed after clinical recovery (60.3± 3.2% versus 61.7 ± 2.4%, $p > 0.05$). Isovolumic contraction time was similar at acute infection and after clinical recovery (44.3 ± 7.8 versus 40.6 ± 9.7ms, $p > 0.05$)

Conclusion: In conclusion, our study suggests global reversible LV dysfunction in COVID-19 patients with preserved LV systolic function based on tissue Doppler derived MPI. This could be due to isolated subclinical diastolic dysfunction. To our knowledge, this study is the first echocardiographic follow-up study that evaluated the systolic and diastolic function of the left ventricle in COVID -19 patients. The underlying mechanism and its clinical significance can be established by further studies.