Tissue Doppler, Speckle Tracking and Strain Imaging

Association of COVID-19 with impaired endothelial glycocalyx, vascular function and myocardial efficiency four months after infection

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Aims: SARS-CoV-2 infection may lead to endothelial and vascular dysfunction. We

investigated alterations of arterial stiffness, endothelial coronary and myocardial

function markers four months after COVID-19 infection.

Methods: In a case-control prospective study, we included 100 patients four months after COVID-19 infection, 50 age- and sex-matched healthy individuals. We measured a) pulse wave velocity (PWV), b) flow-mediated dilation (FMD) of brachial artery, c) coronary Flow Reserve (CFR) by Doppler echocardiography d) left ventricular (LV) global longitudinal strain (GLS), e) left ventricular myocardial work index, constructive work, wasted work and work efficiency and e) von-Willenbrand factor and thrombomodulin as endothelial biomarkers.

Results: COVID-19 patients had lower CFR and FMD values than controls (2.39 ± 0.39 vs 3.31 ± 0.59, p = 0.0122, 5.12 ± 2.95% vs 8.12 ± 2.23%, p = 0.006 respectively). Compared to controls, COVID-19 patients had higher PWV (PWVc-f 12.32 ± 2.44 vs 10.11 ± 1.85 m/sec, p = 0.033) and impaired LV GLS (-19.11 ± 2.14% vs -20.41 ± 1.61%, p = 0.001). Compared to controls, COVID-19 patients had higher myocardial work index, and wasted work (2067.7 ± 325.9 mmHg% vs 1929.4 ± 312.7 mmHg%, p = 0.026, 104.6 ± 58.9 mmHg% vs 75.1 ± 52.6 mmHg%, p = 0.008, respectively), while myocardial efficiency was lower (94.8 ± 2.5% vs 96.06 ± 2.3%, p = 0.008). and thrombomodulin were higher in COVID-19 patients than controls (3716.63 ± 188.36 vs 2590.02 ± 156.51pg/ml, p < 0.001). MDA was higher in COVID-19 patients than controls (10.55 ± 2.45 vs 1.01 ± 0.50 nmole/L, p = 0.001). Residual cardiovascular symptoms at 4 months were associated with oxidative stress markers. Myocardial work efficiency was related with PWV (F=-0.309, p = 0.016) and vWillenbrand (F=-0.541, p = 0.037). Myocardial wasted work was related with PWV (F = 0.255, p = 0.047) and vWillenbrand (F = 0.610, p = 0.016).

Conclusions: SARS-CoV-2 may cause vascular dysfunction, followed by a waste of cardiac work, in order to compensate for increased arterial stiffness 4 months after infection.