COVID-19 patients present impaired endothelial glycocalyx, vascular dysfunction and myocardial deformation resembling those observed in hypertensives four months after infection

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Background/Introduction: COVID-19 infection has been associated with increase arterial stiffness, endothelial dysfunction, and impairment in coronary and cardiac performance. Inflammation and oxidative stress have been suggested as possible pathophysiological mechanisms leading to vascular and endothelial deregulation after COVID-19 infection.

Purpose: The objective of our study is to evaluate premature alterations in arterial stiffness, endothelial, coronary, and myocardial function markers four months after SARS-CoV-2 infection.

Methods: We conducted a case-control prospective study, including 70 patients four months after COVID-19 infection, 70 age- and sex-matched untreated hypertensive patients (positive control) and 70 healthy individuals. We measured a) perfused boundary region (PBR) of the sublingual arterial microvessels (increased PBR indicates reduced endothelial glycocalyx thickness b) flow-mediated dilation (FMD), c) coronary Flow Reserve (CFR) by Doppler echocardiography d) pulse wave velocity (PWV) and central systolic (SBP) e) global LV longitudinal strain (GLS) by speckle tracking imaging and f) malondialdehyde (MDA) as oxidative stress marker.

Results: COVID-19 patients had similar CFR and FMD with hypertensives (2.48±0.41 vs 2.58±0.88, p=0.562, 5.86±2.82% vs 5.80±2.07%, p=0.872 respectively), but lower CFR and FMD than controls (3.42±0.65, p=0.0135 9.06±2.11%, p=0.002 respectively) Both COVID-19 and hypertensive group had greater PBR than controls (PBR5–25: 2.07±0.15 μm and 2.07±0.26 μm p=0.8 vs 1.89±0.17 μm, p=0.001). COVID-19 patients and hypertensives had higher PWV and central SBP than controls (PWVcf 12.09±2.50 and 11.92±2.94, p=0.7 vs 10.04±1.80 m/sec, p=0.036). COVID-19 patients and hypertensives had impaired values of GLS compared to controls (–19.50±2.56% and –19.23±2.67%, p=0.864 vs –21.98±1.51%, p=0.020). Increased PBR5–25 was associated with increased SBP central which in turn was related with impaired GLS ($p<0.05$). MDA was found increased in COVID-19 patients compared to both hypertensives and controls (10.67±2.75 vs 1.76±0.30, p=0.003 vs 1.01±0.50 nmole/L, p=0.001).

Conclusions: SARS-CoV-2 may cause impaired coronary microcirculatory, endothelial and vascular deregulation which remain four months after initial infection and are associated with reduced cardiac performance. The 10-fold increase of MDA compared to healthy individuals four months after COVID-19 infection indicate oxidative stress as possible pathophysiological mechanism.