No cardiac sequelae after COVID-19: results of the one year follow-up with echocardiography and biomarkers

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Objective: To evaluate the need for cardiac monitoring of unselected patients recovered from COVID-19 and to estimate the risk of heart failure development after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)

Methods: During March 2020 and January 2021, 106 patients recovered from SARS-CoV-2 (alfa and beta variants) were enrolled in the prospective observational cohort study, CoSuBr (Covid Survivals in Brno). The diagnosis was established by reverse transcription polymerase chain reaction on swab test of the upper respiratory tract.

Demographic parameters, patient history, clinical evaluation, cardiac biomarkers, ECG and echocardiography were recorded within three visits (V1 at least six weeks after infection, V2 three months later and V3 one year after V1).

Results: The study group (n=106) included 58% women, mean age was 46 years (range 18–77 years). Mean time interval between the onset of the infection and the follow-up visit was 107 days. One quarter (24.5%) of the patients required hospitalisation during the acute phase of the disease, the rest recovered at home. 74% suffered from mild form, 3.8% moderate, 18.3% severe and 2.9% of critical form of the disease. At the time of enrolment 63.5% of the patients were referring the ongoing symptoms, while

more than half of the whole group (50.9%) mentioned at least one symptom of possible cardiac origin (breathing problems, palpitations, exercise intolerance, fatigue).

During one year follow up after COVID-19 infection there was no decrease of left ventricle ejection fraction (V1: $62.1\pm5.26\%$ to V3: $60.8\pm6.24\%$, p=0,150), no changes of troponin (4.8 ± 3.41 to 4.7 ± 3.31 ng/L; p=0.417) and NT-proBNP (74.3 ± 75.15 to 95.3 ± 137.28 pg/mL; p=0,315). There was a mild decrease of right ventricle end diastolic diameter (28.9 ± 5.59 to 26.8 ± 3.83 mm, p<0.001), while no right ventricle dysfunction was detected. There was a very mild progress in a left ventricle diastolic diameter (47.9 ± 6.34 to 49.3 ± 4.21 mm; p<0.001) between V1 a V3, a mild enlargement of left atrium (37.1 ± 5.85 to 38.2 ± 5.65 mm; p=0,021) and non-significant trend to impairment of left ventricle diastolic dysfunction. There was a mild change of pulmonary artery systolic pressure (24.7 ± 7.92 to 27.5 ± 6.97 mmHg; p=0,038).

Conclusion: Despite many information regarding cardiac impairment of SARS-CoV-2 our study does not suggest increased risk of development of heart failure during the one year follow-up. Based on our results the routine echocardiography and biomarkers collection is currently not recommended after COVID-19 recovery.