P302 ENDOTHELIALITIS AS A POSSIBLE CAUSE OF MYOCARDIAL INJURY DURING COVID-19 INFECTION

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A 53-year-old woman was admitted for chest pain for over two weeks, fever, dry cough, fatigue and myalgia. Chest pain increased in supine position and inspiration. Nasopharyngeal swab was positive for SARS-CoV-2 RT-PCR assay. Admission ECG displayed mild ST-segment elevation (0,5 mm) in inferolateral leads and echocardiogram showed normal left ventricular volume, mildly decreased left ventricular EF (52%) and no regional wall motion abnormalities. Blood tests exhibited elevated values of high-sensitivity troponin I (818 pg/mL), proBNP (19.190 pg/mL), CRP (42 mg/dL), D-Dimer test (1090 ng/mL) and WBC (13000/uL, N 89%, L 7%). IL-6 resulted slightly increased (18 ng/mL). ECG after 24 hours showed T wave inversion in inferolateral leads. Coronary CT angiography revealed normal epicardial arteries. A new echocardiogram, after five days, showed infero-basal hypokinesia, improved global EF (57%) and minimal pericardial effusion. Aspirin and fondaparinoux were started. Myopericarditis was suspected and high dose steroids were administered. After a few days, symptoms improved and ECG, echocardiographic abnormalities and troponin levels were normalized. At 6 weeks follow-up the patient was asymptomatic, ECG and echocardiogram were both normal. Instead cardiac MRI showed signs of endothelial injury: presence of interstitial oedema in the inferoapical and anteroseptal wall (STIR-T2-weighted), without late gadolinium enhancement. We measured blood levels of endothelial dysfunction markers: soluble ICAM-1, VCAM-1 and von Willebrand factor (vWF). They all resulted elevated as compared to a laboratory pool of normal serum (535 ng/ml, 265 ng/ml, and 1659 ng/ml, respectively). Our case was suggestive of myopericarditis related to Sars-Cov-2, but echocardiogram and MRI did not show signs of myocarditis permanent damage. However MRI showed interstitial oedema and hyperemia, typical signs of endothelial injury. Moreover we found high levels of soluble endothelial adhesion molecules, vWF, ICAM-1 and VCAM-1, likely expression of the microcirculatory endothelial dysfunction due to myocardial endothelialitis.

In conclusion, our case report suggests that, in COVID-19, acute myocardial injury associated with clinical and investigational signs of myocarditis can be due to myocardial endothelialitis rather than true myocarditis and that an early management with anti-inflammatory drugs (steroids) can be particularly beneficial in patients with unexplained elevated cardiac troponin.