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P312 A CASE OF TAKOTSUBO CARDIOMYOPATHY IN SARS-COV-2 INFECTION

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Cardiovascular complications are a frequent occurrence in Sars-CoV-2 infection. Takotsubo cardiomyopathy is a possible cause of transient ST segment elevation (1-3% of total cases) and is characterized by a reversible left ventricular dysfunction with coronary arteries free from critical stenosis. The predominant echocardiographic pattern is an akinesia of the mid-apical myocardial segments with preserved kinetics of the basal segments. The incidence of Takotsubo cardiomyopathy showed a marked increase during the COVID-19 pandemic. Three factors mainly contribute to the onset of cardiomyopathy in patients with COVID-19: cytokine storm, adrenergic hyperactivation and microvascular dysfunction. A 79-year-old woman was hospitalized in the Pneumology department of our hospital for bilateral interstitial pneumonia Sars-CoV-2 related. In remote medical history: in 2010 diagnosis of breast cancer with negative follow-up for disease recovery, arterial hypertension, hypercholesterolemia and syndrome of reduced mobility as a result of a previous ischemic stroke. Non-invasive mechanical ventilation with c-PAP was started and, for the detection of anterolateral electrocardiographic anomalies (diffuse symmetrical negative T waves) and an increase in myocardionecrosis markers, echocardiographic evaluation was performed, which revealed an akinesia of the mid-apical segments of the left ventricle with preserved kinetics of the basal segments and severely depressed global systolic function (ejection fraction: 30%). BNP (18.100 pg /mL), interleukin-6 (339 pg $\mbox{/mL}$) and CRP (136 mg $\mbox{/L}$) were elevated on blood chemistry tests. In the suspicion of Takotsubo cardiomyopathy (InterTAK Score: 67, probability: 79.8%), cardioprotective therapy with ACE inhibitor, beta-blocker, diuretics, low molecular weight heparin, double antiaggregation and statin was started, deferring coronary angiography to improved clinical conditions. In the following days, due to the respiratory and haemodynamic worsening, the patient was transferred to an intensive setting, where she was intubated and treated with levosimendan and noradrenaline. Serial echocardiograms showed a progressive improvement of the global systolic function (ejection fraction at the last checkup: 55%), with complete regression of the kinetic anomalies, confirming of the diagnostic hypothesis of stress cardiomyopathy. For a pulmonary mycotic superinfection, complicated by iatrogenic pneumothorax, in the following days the patient died.

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