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## Complex Clinical Cases

### CARDIOGENIC SHOCK SECONDARY TO COVID 19 MYOCARDITIS: PECULIARITIES ON DIAGNOSIS, HISTOLOGY AND TREATMENT

Poster Contributions

Saturday, May 15, 2021, 1:15 p.m.-2:00 p.m.

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Session Title: Complex Clinical Cases: FIT COVID-19 1

Abstract Category: FIT: Coronavirus Disease (COVID-19)

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**Background:** The current year was dramatically characterized by SARS-Cov 2 outbreak. COVID-19 related heart diseases have been reported.

**Case:** a 45-year-old healthy male, with no medical history, presented with fever, shortness of breath and severe asthenia. EKG showed new onset of left bundle branch block, Troponin T was above the upper limit and fast echocardiography revealed severely impaired biventricular function (EF: 25%, TAPSE 12mm). The patient had spent a few days working with a colleague from Lodi, one of the most affected cities by the Italian February 2020 epidemic outbreak of SARS-Cov 2. The colleague's parents had had fever some days before. Nasopharyngeal swab for SARS-Cov 2 resulted negative.

**Decision-making:** the patient needed mechanical circulatory support with intra-aortic balloon pump and inotropes. Because of the high suspect of COVID-19, based on thoracic CT and epidemiological history, a bronchoalveolar lavage was done and SARS-Cov 2 RNA was then detected. Cardiac MRI confirmed the suspect of myocarditis, revealing higher values of T1 and T2 mapping, with no late gadolinium enhancement. Cardiac biopsy showed mild lympho-histiocytic inflammatory infiltrate, without myocardial necrosis, with unusual diffuse platelets microclots; SARS-Cov 2 RNA was not detected. Continuous infusion of levosimendan was administered for 24h and a subcutaneous IL-1 inhibitor was started to counteract the disease-related cytokine storm. A dramatic improvement in biventricular function was observed few days later and 6-month therapy with IL-1 inhibitor was prescribed. At three-month follow-up cardiac MRI confirmed the stable improvement of biventricular function with normalization of T1 and T2 mapping.

**Conclusion:** the severe impairment of cardiac function was the result of the massive cytokine storm caused by the SARS-Cov 2 infection. Platelet microclots were observed in myocardial tissue, related to the "Microvascular Covid-19 vessels obstructive thrombo-inflammatory syndrome" contributing to myocardial distress. A dramatic and stable improvement from 25% to 45-50% of left ventricular ejection fraction was achieved counteracting the cytokine burden with a IL-1 inhibitor.