

lactates 10 mmol/dL) and hypoxaemia. Situation evolved towards an end-organ hypoperfusion associated with multisystem organ failure (MOF). High resolution chest CT demonstrated bilateral ground glass opacities consisting of COVID-19 related interstitial pneumonia. 12-leads ECG demonstrated sinus rhythm with diffuse concave ST-segment elevation. T-T echocardiogram showed: 1) severely thickened LV walls; 2) decreased LV systolic function; 3) segmental wall motion abnormalities consisting of akinesis of the LV distal segments with relatively preserved function at the base; 4) LVEF) of 25%; 5) pericardial effusion. Thickened LV walls meant myocardial inflammatory infiltration/edema and the final diagnosis was FULMINANT COVID-19 related MYOCARDITIS. Patient underwent oro-tracheal intubation+invasive ventilation. An intra-aortic balloon pump (IABP) was placed on top of adrenaline (0.07µg/kg/min), and noradrenaline (0.1µg/kg/min) was added for worsening hypotension (systolic blood pressure: 80/67/60 mmHg). Additional therapy consisted of intravenous high dose diuretics, methylprednisolone, tocilizumab, immunoglobulin, ceftriaxone, remdesivir. After 3 weeks, patient recovered a good respiratory and haemodynamic status, LV wall thicknesses decreased and EF increased to normal values. The final outcome was favorable.

**Discussion:** The prevalence of fulminant myocarditis among COVID-19 patients is unclear. This simultaneous presentation of fulminant myocarditis and COVID-19 pneumonia favors an alternative pathogenetic pathway with possible acute replication and direct dissemination of the virus through the blood or the lymphatic system from the respiratory tract to the myocardium.

## COVID & MYOCARDITIS

### P301 FULMINANT COVID-19 RELATED MYOCARDITIS: A CASE REPORT

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**The case:** A 58-year-old man presented to ED because of fever, non-productive cough and worsening dyspnea. The patient's medical history was unremarkable. On examination there was a hypoxic respiratory failure and the clinical picture rapidly evolved towards a cardiogenic shock characterized by altered mental status and cool extremities. Vital parameters: peripheral blood pressure was of 60/30 mmHg, pulse rate of 126 bpm, respiratory rate of 30 breaths/min, temperature of 38 °C. Two episodes of ventricular fibrillation were resolved by electrical shocks. The nasopharyngeal swab tested positive for COVID-19 on real-time reverse transcriptase-polymerase chain reaction assay. Arterial blood gas analysis showed a severe metabolic acidosis with hyperlactacidaemia (pH 7.26, excess basis-22 mEq/L,

