Abstract: Fulminant lymphocytic myocarditis with cardiogenic shock and multiorgan failure with complete recovery after percutaneous mechanical support and immunosuppression.

Authors: I Jurcova, V Melenovsky, M Pindak, H Riha, I Netuka, J Maly, O Szarszoi, J Maluskova, E Honsova, W Bracamonte-Baran, D Cihakova, M Kubanek, J Kautzner, 1Institute for Clinical and Experimental Medicine (IKEM), Cardiology - Prague - Czechia, 2Institute for Clinical and Experimental Medicine (IKEM), Anaesthesiology and Resuscitation Department - Prague - Czechia, 3Institute for Clinical and Experimental Medicine (IKEM), Cardiovascular Surgery - Prague - Czechia, 4Institute for Clinical and Experimental Medicine (IKEM), Department of Pathology - Prague - Czechia, 5Johns Hopkins University of Baltimore, Department of Pathology - Baltimore - United States of America,

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Fulminant myocardiitis is rare but serious myocardial inflammatory disease that presents with acute heart failure, cardiogenic shock and/or life-threatening arrhythmias. The management often requires intensive care, inotropes and mechanical circulatory support. Immunosuppressive therapy may result in a complete recovery.

We report a case of previously healthy 19- year-old female that presented to regional hospital after two days of gastrointestinal symptoms with 3rd degree AV block, left ventricular (LV) systolic dysfunction (LVEF 40 %), significantly elevated cardiac biomarkers (hsTnT 13850 ng/l, NTproBNP 19686 ng/l) and cardiogenic shock requiring inotropes, temporary pacing and endotracheal intubation. Coronary angiography showed patent coronary arteries. Within next 24 hours, her status deteriorated with development of severe biventricular dysfunction (without wall thinning), widening of QRS complex and multiple organ failure. She was transferred to our center for further management and peripheral VA-ECMO was introduced. However, LVEF dropped to less than 10% with further QRS widening. An endomyocardial biopsy (EMB) revealed diffuse lymphocytic myocarditis (LM), resembling severe cellular allograft rejection with diffuse interstitial edema, but relatively sparse myocyte necrosis. FACS analysis of EMB showed CD4/CD8 ratio shifts to 1/3, normal ratio is 3/1. Both CD4 and CD8 Tcells were activated, rather than in a naïve status. Activated cells were predominantly Th1 cells. There was a significant expansion of CD14+CD16low cells (inflammatory, classical monocytes). PCR for panel of possible viral pathogens in the EMB specimen was negative. Therefore, immunosuppression with 1g/day (in 3 days) of methylprednisolone and intravenous IgG (0.5 g/kg, once) was instituted. Because of development of long runs of incessant VT (rate 220/min.) and LV dilatation, Impella 2.5 was introduced to achieve LV venting and to improve myocardial edema drainage. Immediately after addition of Impella to VA-ECMO, VTs ceased and QRS complex narrowed. Due to continuing LV dysfunction, Impella 2.5 was exchanged for Impella 5.0. On the day 10, echocardiogram showed significant recovery of LVEF. Therefore, VA-ECMO was discontinued on the day 11 and Impella was explanted on the day 13. Further recovery was uneventful and the patient was discharged after 44 days. Clinical status was stabilized with normal cardiac function by echocardiography and normal BNP and troponin levels. She is currently being tapered off of low dose of prednisone 2.5mg/day.

Conclusions: Our case illustrates that if acute viral infection is ruled out, immunosuppressive protocol can be successfully used in management of fulminant LM together with temporary mechanical circulatory support consisting of VA-ECMO + Impella. Rapid resolution of QRS prolongation and LV dysfunction with LV unloading by Impella suggest possible role of myocardial edema in development of severe mechanical ventricular dysfunction.