A 37-year-old male with past medical history of hypertension and type 2 diabetes mellitus presented to our hospital due to an episode of syncope. On admission, patient was febrile with T:103.1°F and tachycardic at 132bpm, SpO2:100% at room air and blood pressure within normal limits. An EKG showed sinus tachycardia with no ST-T wave changes. Cardiac biomarkers revealed troponin of 0.04ng/mL. SARS-CoV-2 PCR from nasopharyngeal specimen was positive. Initial chest X-ray revealed no acute pulmonary disease [Fig.1].

On hospital day #1 patient became hypotensive and he was started on volume resuscitation and vasopressor therapy. Chest radiograph showed the development of pulmonary edema [Fig.2]

Hospital day #2 an echocardiogram revealed diffuse left ventricular hypokinesis and LVEF of 15-20%. Patient was started on intravenous milrinone with improvement in hemodynamic status. He was then started on guideline-directed heart failure therapy. A follow-up MUGA scan on hospital day #9 revealed an LVEF of 50-55%. Patient was then discharged on guideline-directed heart failure medication.

The pathophysiology of SARS-CoV-2 induced myocardial injury is hypothesized to involve direct damage to cardiac myocytes, interstitial fibrosis and its associated immune response. [1,3]

Large cohort studies have shown the prevalence of elevated troponins, and associated poor prognosis, in SARS-CoV-2 infected patients [2,4]. However, reports of SARS-CoV-2-induced-myocarditis and treatment outcomes are limited.

Our patient had minimal comorbidities, echocardiogram findings significant for cardiogenic shock and hemodynamic instability which responded to inotropes in the presence of confirmed SARS-CoV-2 infection. Additionally, ischemic etiology was excluded making the diagnosis of fulminant myocarditis well-supported.

Our patient responded to standard heart failure. This case adds support that standard of care therapy for fulminant viral myocarditis is effective in SARS-CoV-2 fulminant myocarditis. Further study and monitoring of patients with SARS-CoV-2 induced fulminant myocarditis is required to understand long term sequelae.