Cardiogenic Shock Secondary To SARS-2-CoV-Induced Viral Myocarditis

Rivera, Carlos M.D. PGY-2; Nicholson, Michael D.O. PGY-3; Dedousis, John M.D. Program Director; Choudhry, Hammad MD Associate Program Director; Tsompanidis, Antonio DO Director of Medical Education; Litinski, Mickhail M.D, ICU Director
Hoboken University Medical Center – Bayonne Medical Center

Case Synopsis

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:103.1F 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.

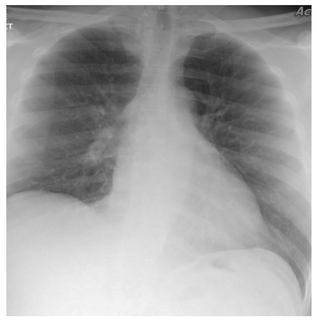

Figure 1. Chest X-ray on admission

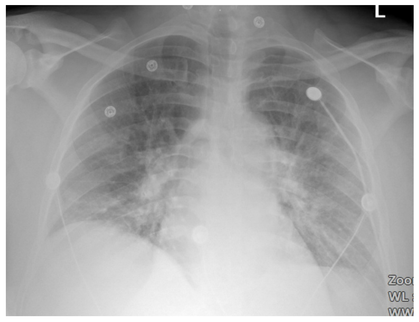

Figure 2. Chest X-ray on hospital day #1

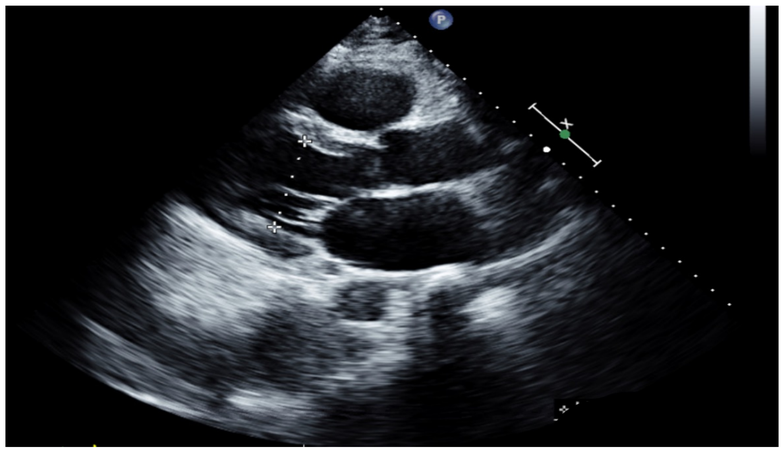

Figure 2. Echocardiogram on hospital day #2

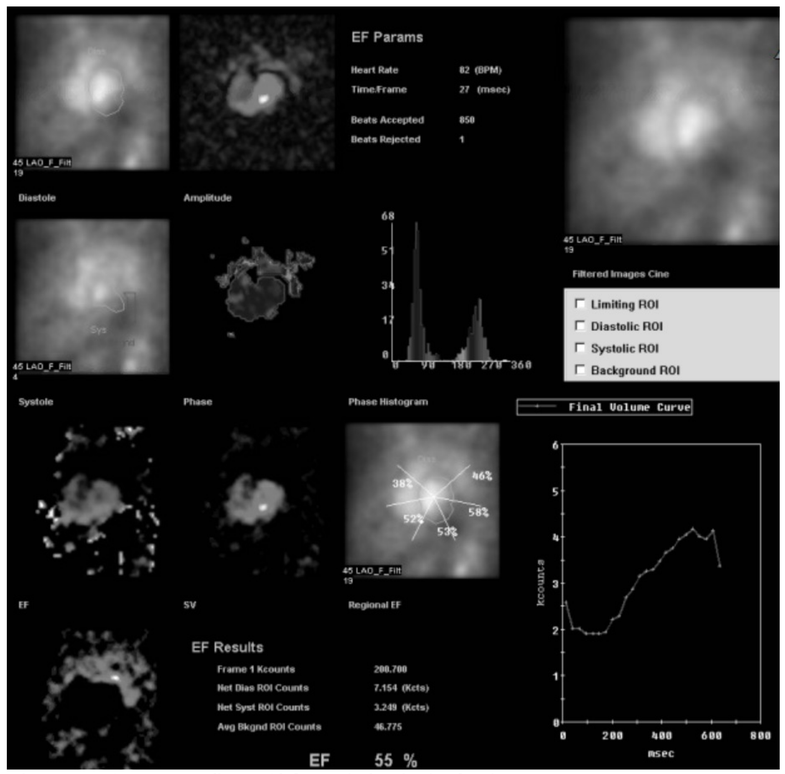

Figure 4. MUGA scan revealing 55% left ventricular ejection fraction.

Medical Literature

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.

Unique aspects

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.

Conclusions

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.

Contact

Mark Cutola, Program Coordinator
Choudhry, Hammad MD Associate Program Director
Dedousis, John MD Program Director
Mickhail Litinski M.D, ICU Director
Tsompanidis, Antonio DO Director of Medical Education
Hoboken University Medical Center- Bayonne Medical Center
29E 29th street, Bayonne NJ 07002
Phone: 201-858-6594

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