



## Case Report

# A case report of management of cardiomyopathy in a patient with prior COVID infection

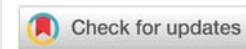
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## Abstract

The novel coronavirus (SARS-CoV-2) is responsible for the current pandemic and while most patients have mild symptoms, severe COVID-19 infections can have long-lasting symptoms. There is data to suggest that sequelae from COVID-19 persist for months. Viral myocarditis and cardiomyopathy related to COVID-19 have been described in the non-pregnant population. We present a case of a parturient presenting with left ventricular global hypokinesis and ejection fraction of 38% two months after initial COVID-19 infection. Pregnant patients with COVID-19 related cardiomyopathy should be managed by a multidisciplinary team. We suggest considering SARS-CoV-2 infection in parturients presenting with symptoms of decompensated heart failure.

## Glossary of terms

COVID-19: Coronavirus Disease 2019; CXR: Chest X-Ray; BMI: Body Mass Index; CT: Computed Tomography; HD: Hospital Day; LMWH: Low Molecular Weight Heparin; ECG: Electrocardiogram; TTE: Transthoracic Echocardiogram; LVEF: Left Ventricular Ejection Fraction; BNP: Brain Natriuretic Peptide; PPD: Post-Partum Day; RVSP: Right Ventricular Systolic Pressure; CVP: Central Venous Pressure; SARS: Severe Acute Respiratory Syndrome; MERS: Middle Eastern Respiratory Syndrome; CSE: Combined Spinal Epidural; TWI: T-Wave Inversion

## Introduction

As of April 2021, the coronavirus SARS-CoV-2 has infected more than 134 million humans per the Johns Hopkins Coronavirus Resource Center. It has become apparent that while many patients have a mild course, severe COVID-19 infections can have both crippling and longlasting symptoms. Cardiomyopathy and viral myocarditis, associated with COVID-19, have been described in the non-pregnant population[1]. However, there is limited literature related to cardiovascular sequelae of COVID-19 parturients [2]. We present a case of suspected COVID-19 associated cardiomyopathy in

a parturient and the multidisciplinary approach to achieve a safe Caesarean delivery. The patient gave written HIPAA authorization to write this case report.

## Case description

A 36-year-old G2 P0010 woman presented at 25 weeks 5 days gestation with dyspnea. Her medical history included gestational hypertension, hypothyroidism, mild asthma, type 2 diabetes, and morbid obesity (BMI 52.5kg/m<sup>2</sup> pre-pregnancy). She had known exposure to COVID-19 and a reverse transcription polymerase chain reaction test of a nasopharyngeal swab was positive. Her presenting symptoms were exertional dyspnea, fever (101.7°F), and hypoxia (requiring 2L oxygen via nasal cannula to maintain oxygen saturation >92%). Vital signs on admission included HR 121, BP 199/58, RR 25, temperature 98.7°F (37.1°C). Laboratory values included procalcitonin 0.06 ng/mL (normal <0.5 ng/mL), troponin <0.01 ng/mL (normal <0.11 ng/mL), C-Reactive Protein 161.55 mg/dL (normal <100 mg/dL), hemoglobin 9.7 g/dL (normal 11.4 - 15.2 g/dL), and D-dimer 1.21 mcg/mL (normal <0.50 mcg/mL), Brain Natriuretic Peptide (BNP).

8 pg/mL (median BNP levels remain less than 20 pg/mL in normal pregnancy). Chest radiograph (CXR) revealed

bilateral fluffy infiltrates; pulmonary embolism was excluded with Computed Tomographic (CT) angiography. Chest CT demonstrated diffuse multifocal consolidative and ground glass opacities bilaterally and she was diagnosed with COVID-19 pneumonia. Her symptoms improved over four days and she no longer required supplemental oxygen by Hospital Day (HD) five. She received betamethasone on HD 1 and 2 given the risk of preterm labor. She was discharged to home on HD5.

The patient was readmitted 10 weeks later with a one-month history of worsening exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. She reported 28-pound weight gain over the prior one month (BMI 64.64 kg/m<sup>2</sup> on admission). Vitals on admission included BP 146/90 mmHg, HR 111 bpm, SaO<sub>2</sub> 95–97% on room air, Temp 98 °F (36.7°C), with anasarca on exam, and diminished breath sounds bilaterally. CXR was without overt pulmonary edema or pleural effusions. ECG demonstrated normal sinus rhythm, left axis deviation, no acute ST changes, no Qwaves, TWI in V1 and TW flattening in III and V2. A Transthoracic Echocardiogram (TTE) demonstrated global hypokinesis and LVEF 38%. Laboratory findings of note included a BNP 51pg/mL, urine protein:creatinine ratio 0.2, and 24 hour urine protein <4mg/24 hours [3]. She was diagnosed with a presumed cardiomyopathy of unclear etiology. The differential diagnosis included myocarditis from SARS-CoV2, underlying other cardiomyopathy, preeclampsia, and peripartum cardiomyopathy. A multidisciplinary approach to her care was taken. The team articulated management goals including delay of delivery to facilitate diuresis. A furosemide infusion was initiated to decrease preload, as well as hydralazine for afterload reduction.

She began to have severe range blood pressure on HD 3, attributed to gestational hypertension. Induction of labor was initiated, however an intracervical foley catheter was unable to be placed and the decision was made to proceed with Cesarean delivery. Pre-delivery, a pulmonary artery catheter was placed to monitor cardiac output and guide inotropic therapy. An arterial line was also placed prior to delivery. The patient underwent a primary Cesarean delivery under Combined-Spinal Epidural (CSE) anesthesia. The patient received an initial intrathecal dose of bupivacaine 0.75 % 1.6 mL, morphine 0.1 mg, fentanyl 10 mcg. She was placed supine with left uterine displacement and an infusion of phenylephrine was initiated to maintain systolic blood pressure within 20% of baseline. As the intrathecal dose did not provide adequate anesthesia, she received an 15ml of 2% lidocaine with 1:200,000 epinephrine in divided doses to achieve anesthesia to pinprick at the level of T4. She recovered with continuous telemetry, MAP, CVP and SVR monitoring until Post-Partum Day (PPD) 3. Furosemide and nitroglycerin infusions were initiated PPD 1 per cardiology as her MAP, CVP and SVR were all increasing. These were transitioned to oral furosemide and enalapril on PPD 4. A repeat TTE on PPD 3 demonstrated normal left ventricular chamber size, moderately reduced systolic function with an EF estimated at 35 – 40%, grade 1 diastolic dysfunction. The right ventricular systolic function was lownormal with RVSP mildly elevated at ~39 mmHg. The patient was unable to complete a cardiac MRI due to claustrophobia. The patient

was discharged to home on PPD 8. Six weeks postpartum, the patient reported improvement of both dyspnea and fatigue. Repeat echocardiography demonstrated LVEF 50% with mild global hypokinesis.

## Discussion

There is limited data on critically ill parturients with COVID-19, and COVID-19-related cardiomyopathy in pregnancy is even less well understood. This case highlights the additional challenge of diagnosing cardiomyopathy presenting in pregnancy. Because of the antenatal presentation, the temporal relationship to the prior COVID-19 infection and the normal BNP values, the authors felt this case of cardiomyopathy was related to COVID-19 versus peripartum cardiomyopathy or related to preeclampsia.

The clinical course of COVID-19 infection in pregnancy follows a trend similar to the general population with 86% of infections being mild, 9.3% severe, and 4.7% critical [4]. A multicenter study examined the clinical course of severe and critically ill pregnant patients with COVID-19 in the United States and found that 69% of patients admitted to the hospital had a severe course and 31% a critical course [5]. They reported one case of maternal cardiac arrest but no cases of cardiomyopathy. There is one prior report of two cases of COVID-19 related cardiomyopathy in pregnancy, where those two patients presented acutely with respiratory illness [2]. In a single center study in China, 19% of COVID-19 patients had evidence of myocardial injury, while a US case series revealed cardiomyopathy in 33% of patients with COVID-19 [6,7]. The presence of myocardial injury has also recently been documented in patients who recovered from COVID-19 illness, as in our patient. Indeed, severe myocarditis related to COVID-19 presents 2–3 weeks following initial infection [8]. Cardiac MRI studies suggest high prevalence of persistent cardiac involvement and inflammation even two months after initial diagnosis [9]. It is hypothesized that myocardial injury in SARS-CoV2 infection is related to an inflammatory response with hypercoagulability, downregulation of ACE-2 receptors, and increased circulating catecholamines [10]. Hypoxemia in the setting of diminished pulmonary function can exacerbate myocardial injury which may present as decompensated heart failure, myocardial infarction, and arrhythmia.

Physiologic changes of the cardiovascular, respiratory, and immune system during pregnancy increase the risk of severe infection. However, the outcomes of SARS-CoV2 infections in pregnancy are more favorable compared to SARS or MERS infections (case fatality rate of 0%, 18%, and 25% respectively) [11]. Nonetheless, it is unclear if pregnant patients with COVID-19 and multiple risk factors for cardiovascular disease are at higher risk of myocardial injury, hypertension-associated heart failure, or peripartum cardiomyopathy. It is also unclear if hypertensive disorders predispose to or exacerbate myocardial injury related to COVID-19 similarly to peripartum cardiomyopathy [12]. With an EF of 38%, our patient is classified as modified World Health Organization class III with a maternal cardiac event rate of 19–27% [13].



The management of COVID-19-related cardiomyopathy is unclear. Patients at risk for cardiovascular disease in pregnancy should be managed by a multidisciplinary team (Obstetrician, Cardiologist and Anesthesiologist at a minimum) at an expert center. Patients with peripartum cardiomyopathy must be counseled on the possibility of recurrence of cardiomyopathy in subsequent pregnancies, even after full recovery. Induction of labor should be considered at 40 weeks gestation for all patients with cardiac disease [13]. Pulse oximetry and continuous electrocardiogram monitoring are recommended. An arterial line allows for continuous monitoring and quick responses to interventions and events. Epidural anesthesia can be used for labor as well as Cesarean delivery but should be carefully titrated in the setting of decreased ventricular function. In this case, a CSE was placed in the sitting position with continuous hemodynamic monitors that allowed for careful titration of medication in response to anticipated hemodynamic changes. Intravenous fluids must also be carefully managed.

As vaccination efforts improve, hopefully fewer pregnant patients will present with COVID-19 infections or be diagnosed as asymptomatic carriers when presenting to labor and delivery. It continues to be important to consider extra-respiratory manifestations of SARS-CoV2 infections in this population where altered physiology may change disease presentation. It may be prudent to perform echocardiography early and often for parturients who present with dyspnea and are diagnosed with COVID-19.

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