

CASE REPORT

Severe peripartum cardiomyopathy complicated by COVID-19 infection and small intestinal obstruction

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Abstract

COVID-19 infection can be a possible trigger for peripartum cardiomyopathy. Multidisciplinary teamwork was crucial for the favorable outcome in our patient. Small bowel strangulation is a rare complication post-cesarean section.

KEY WORDS

COVID-19, peripartum cardiomyopathy, small bowel obstruction

1 | CASE PRESENTATION

A previously healthy 31-year-old woman, who had an uncomplicated elective cesarean section for delivery of a breech newborn, presented to the emergency department on day six postpartum with a complaint of progressive shortness of breath and dry cough for two nights.

Soon after arrival, the patient required emergent intubation, sedation, and mechanical ventilation for acute hypoxemic respiratory failure attributed to cardiogenic shock. Additionally, the patient also tested positive for SARS-CoV-2. Initial work-up included CT of the chest and abdomen that showed bilateral ground-glass opacities of the lungs and an incarcerated small bowel volvulus. Transthoracic echocardiogram showed global hypokinesia and an ejection fraction estimated at <25%.

During hospitalization, the patient required cardiovascular support from a temporary percutaneous left ventricular assist device. She also underwent a two-stage surgical intervention for small bowel resection and anastomosis.

She subsequently improved and was discharged in a good clinical condition. At the time of discharge, her cardiac function had an estimated ejection fraction of 35%.

A 31-year-old Caucasian woman presented to the emergency department on her sixth day after an uncomplicated elective cesarean section with a complaint of progressive shortness of breath and dry cough for two days followed by an acute onset epigastric pain, it was associated with nausea and vomiting. En route to the hospital, the patient was in hypoxemia with the reported blood pressure of 60/40 mmHg, her oxygen saturation 80% on supplemental oxygen.

In the emergency department, resuscitation with intravenous fluids was immediately initiated. The assessment revealed normal temperature, somnolence, tachypnea, tachycardia, diffuse coarse crackles on pulmonary auscultation, and epigastric tenderness without peritoneal signs or evidence of cesarean incision site infection. Initial laboratory findings included a mixed respiratory and metabolic acidosis with a pH of 7.28, lactate of 3.7 mmol/L, potassium of 2.4 mmol/L, troponin of 8.37, normal creatinine, normal liver enzymes, and total bilirubin of 1.6 mmol/L. Oxygen saturation was ranging between 87 and 93% on 15 L supplemental oxygen. An electrocardiogram showed atrial fibrillation with a heart rate of 123 bpm (Figure 1). Intravenous potassium replacement was initiated. Due to the concern of hemodynamic instability and the questionable ability of the patient to protect her airway, a decision was made to emergently intubate the patient and start mechanical ventilation.

Computed tomographic angiography (CTA) of the chest was performed, and it showed diffuse ground-glass nodular infiltrates bilaterally, suggestive of COVID-19 pneumonia. There was no evidence of pulmonary embolism (Figure 2).

CT of the abdomen and pelvis was performed with intravenous administration of contrast which revealed focally dilated fluid-filled loops of small bowel in the lower midabdomen herniating into the rectus sheath. It was associated with swirling of the mesentery and mesenteric vessels, suggestive of closed-loop small bowel obstruction (Figure 3). Echocardiography was performed and revealed severe impairment in left ventricular systolic function with ejection fraction <25%.

A multidisciplinary team including intensive care, cardiology, obstetrics/gynecology, and surgery were involved in her care. The OBGYN team recommended MgSO₄ infusion for possible eclampsia in the settings of a brief seizure-like activity and muscle stiffness reported by the family.

The surgery team recommended orogastric tube decompression of the stomach, serial assessments, broad-spectrum antibiotics, and an urgent exploratory laparotomy as soon as the patient's hemodynamics stabilizes. The intensivist initiated treatment with multiple vasopressors,

inotropes, and antibiotics for the diagnoses of cardiogenic and septic shock.

The interventional cardiologist recommended an emergent right heart catheterization which was performed, it confirmed the diagnosis of cardiogenic shock as evident by increased pulmonary capillary wedge pressure and reduced cardiac index. The condition was attributed to peripartum cardiomyopathy or myocarditis. During the catheterization, a percutaneous assist device was placed for mechanical circulatory support. The patient then admitted to ICU. SARS-COV-2 PCR subsequently came back positive confirming COVID-19 infection.

COVID-19 treatment protocol was initiated; therefore, dexamethasone and remdesivir were added. Magnesium Sulfate infusion was discontinued given the low suspicion for eclampsia.

Exploratory laparotomy was performed by the acute care surgeon in the presence of the OBGYN team. A necrotic segment of the small bowel was identified in the form of volvulus herniating through the parietal peritoneum, which was repaired in the cesarean section. A 40 cm of small intestine was resected (Figure 4), and anastomosis was deferred. A temporary abdominal closure was performed using open abdomen negative pressure device. Inspection of the uterus, fallopian tubes, and the rest of the abdominal cavity did not show any abnormalities.

The patient's clinical condition was gradually improving as oxygen and vasopressors requirement decreased. A follow-up echocardiogram showed a slight improvement of the ejection fraction (estimated at 35%) with global hypokinesis.

A second-look laparotomy was performed two days later, which revealed healthy bowel loops and anastomosis was completed. The patient was successfully extubated, the mechanical circulatory support was weaned off, and eventually removed. The patient was hemodynamically stable, and she was able to tolerate diet. Guideline-directed therapy for congestive heart failure was initiated. The patient was discharged after 10 days in a good clinical condition. Echocardiography displayed ejection fraction of 55% three months after the initial presentation.

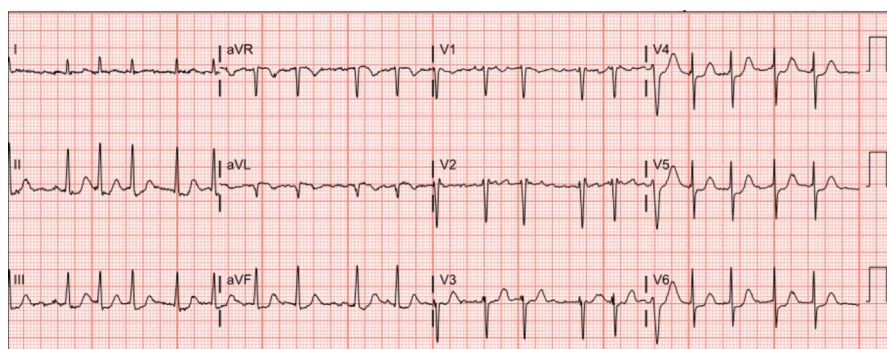


FIGURE 1 EKG with Atrial fibrillation

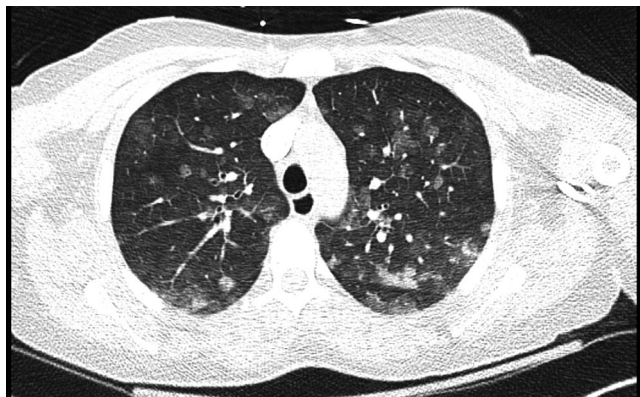


FIGURE 2 CTA Chest showed diffuse ground-glass nodular infiltrates bilaterally typical for COVID-19 pneumonia



FIGURE 3 CT Abdomen showed focally dilated fluid-filled loops of small bowel in the lower midabdomen herniating into the rectus sheath associated with a swirling of the mesentery and mesenteric vessels

2 | DISCUSSION

Peripartum cardiomyopathy (PPCM) is a rare clinical entity of heart failure affecting women in pregnancy and the postpartum period. It is defined by the presence of left ventricular systolic dysfunction, which occurs at the last month of pregnancy and within 5 months following delivery. The diagnosis requires exclusion of other identifiable causes of heart failure and an ejection fraction of 45% according to the guidelines developed by the European Society of Cardiology (ESC).¹

The incidence of PPCM is 1 per 1000 per live birth, this incidence varies geographically from 1 per 1:20,000 live births in Japan to 1 per 100 per live birth in Nigeria. The incidence has increased over the last 20 years due to improvement in echocardiography diagnostic accuracy and the drastic change in maternal characteristics such as advanced maternal age,



FIGURE 4 resection of the ischemic small bowel

increase in assisted reproductive techniques, and multifetal gestation.^{2,3}

The literature review identified risk factors associated with PPCM such as maternal age of more than 30 years, African descent, gestational hypertension, preeclampsia, tocolysis therapy, multifetal gestation, and cocaine use. Multiparity is associated with PPCM but recent studies showed more than 50% of cases occur in the first two pregnancies.^{4,5} Additionally, low-platelets syndrome, diabetes mellitus, anemia, and cesarean delivery have been linked to PPCM.⁶

The etiology of peripartum cardiomyopathy and is believed to be triggered by multifactorial components, which eventually result in hemodynamic compromise. Different etiologies are implicated in the development of PPCM; myocarditis has been reported variably as a potential cause after analyzing endomyocardial biopsies in 78% of patients diagnosed with PPCM. Due to the lack of specific histological criteria to diagnose myocarditis in the endomyocardial samples and varying in the timing of biopsies, it has been a challenge to predict the accurate prevalence of myocarditis in such patients.⁷ Furthermore, certain viral genomes are disclosed in endomyocardial biopsy specimens; Parvovirus B19, human herpesvirus 6, Epstein-Barr virus, and human cytomegalovirus.⁸

Genetic susceptibility is addressed as a potential etiology predisposing to PPCM. Mutation in *TTNC1*, *TTN* genes that encode cardiac protein Troponin C has been identified in such patients.² *TNNC1* mutation is associated with the development of idiopathic dilated cardiomyopathy (DCM) and some studies hypothesize that PPCM can be an initial presentation of familial DCM. PPCM patients with gene mutations have a low recovery rate.⁹

Furthermore, alternation in the prolactin process in pregnancy is a possible etiology of PPCM development. It is hypothesized that in patients with PPCM, pituitary prolactin is cleaved by cathepsin D to 16 kDa prolactin fragment which induces microRNA-146a leading to cardiomyocyte ischemia and ventricular dysfunction. Therefore, bromocriptine, D2 receptor agonist which suppresses prolactin secretion, has

been investigated in the management of PPCM in small clinical trials. A promising outcome was observed with improvement in LV function at 6 months.¹⁰ However, bromocriptine was not integrated into the management of our patient due to concerns of potential adverse vascular events.

Fetal microchimerism is proposed to be a possible etiology of PPCM. Fetal cells in maternal circulation can trigger an autoimmune response, antibodies against cardiac muscle proteins: adenine nucleotide translocator, branched chain a-keto acid dehydrogenase, and myosin were recognized in patients with PPCM. This causes the release of cytokines and local inflammatory reactions leading to myocarditis.^{11,12} However, a study conducted on 39 Nigerian women with PPCM did not find show a significant difference in serum immunoglobulins assay, serum immune complexes, and heart muscle autoantibodies between PPCM cases and healthy postpartum patients.¹³

In light of concurrent COVID-19 infection and small bowel obstruction in our case, it is challenging to delineate the cascade of events that trigger the PPCM in our patient. A similar presentation of PPCM and COVID-19 in the postpartum period occurred in Italy as published in a case report, it affected a patient in a similar age group and parity (first pregnancy) but presented about 4 weeks after delivery.¹⁴

COVID-19-related myocarditis has been reported in the literature, but the incidence is not clear and there are no established criteria to confirm the diagnosis. The clinical presentation is variable and SARS-CoV-2 myocarditis can be manifested by arrhythmia, heart failure, or cardiogenic shock. The nature and history of preexisting arrhythmia are not fully explored in the literature.¹⁵ In our patient, EKG showed atrial fibrillation (AF) in the emergency department which can be identified in 1.3% of cases of PPCM in one review.¹⁶ Additionally, heart failure has been recognized as an initial presentation in COVID-19 cases as well as the outcome of 23% of COVID-19 hospitalized patients.^{17,18}

Proposed mechanism of SARS-COV-2 induced myocarditis: angiotensin-converting enzyme 2 acts as a receptor for the virus to obtain entry cardiac cells. Viral entry causes inhibition of stress granules in cardiomyocytes, which leads to viral replication. This triggers CD8 T-lymphocyte-mediated inflammation, the release of abundant cytokines, and myocardial damage.¹⁵ Hypoxemia caused by COVID-19-related pneumonia can cause significant intracellular acidosis, which has a detrimental effect on the cell membrane of cardiomyocytes and activate apoptosis through hypoxia-induced calcium influx.¹⁹ However, the definite diagnosis of SARS-COV-2 myocarditis requires an endomyocardial biopsy demonstrating viral genome and inflammation, but a biopsy was not performed in our patient.²⁰

The occurrence of COVID-19 in two case reports of PPCM in literature raises the question if SARS-COV-2 can be listed in the spectra of viral pathogens causing peripartum

cardiomyopathy. Furthermore, the diagnosis of intestinal obstruction could be a contributing factor in the development of cardiomyopathy. In the literature review using PubMed, only one case report of intestinal pathology (paralytic ileus) was associated with stress (Takotsubo) cardiomyopathy.²¹ It is stress-related cardiomyopathy with unclear pathogenesis, it is believed to be induced by catecholamines surge in cases of physical or emotional stress. Elevated catecholamines result in structural alterations such as increased extracellular matrix, contraction band necrosis, and neutrophil infiltration.²² Cytokines storm and systemic inflammatory response associated with intestinal ischemia could be implicated in the pathogenesis of cardiomyopathy in our patient.²³

Data showed most patients (72%) of PPCM in North America recover with an ejection fraction of more than 50% in an interval of 12 months. The initial LVEF is predictive of the prognosis in black women whereas this correlation was not established in white women.²⁴ The recovery is influenced by racial and geographic factors considering the poor prognosis of patients in Haiti, Turkey, and South Africa as the recovery rate was 21–43%.^{6,25}

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AUTHOR CONTRIBUTIONS

EA wrote the first draft of the manuscript and reviewed literature. MS critically reviewed and edited draft. DK ensured the accuracy of the case report and approved the final version.

ETHICAL APPROVAL

Ethics approval was not required for this study.

CONSENT STATEMENT

The consent is obtained from the patient prior to submission.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon request.

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