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Case Report

Diagnostic dilemma: COVID-19 related cardiomyopathy or peripartum cardiomyopathy?



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ABSTRACT

Peripartum cardiomyopathy is a relatively rare condition, that usually presents with features of heart failure in the peripartum period. The ongoing pandemic caused by coronavirus disease 2019 (COVID-19) has been reported to be associated with myocarditis, with progression to dilated cardiomyopathy and heart failure. Dilated cardiomyopathy in a peripartum patient with COVID-19 infection may present a diagnostic dilemma. We report a case of dilated cardiomyopathy in a peripartum patient with COVID-19 infection. She presented with shortness of breath in the peripartum period. Chest X-ray showed a grossly enlarged heart with bilateral pulmonary infiltrates consistent with congestive heart failure or viral pneumonia. Echocardiography revealed dilated chambers with 22% left ventricular ejection fraction (LVEF) and global hypokinesis. Despite completing 5 days of remdesivir and dexamethasone, she had worsening dyspnea on postpartum day 10, a repeat echocardiogram showed further reduction in LVEF to 10–15% and was discharged with a life-vest after acute management. She had multiple hospital admissions for decompensated heart failure. Myocardial core biopsy showed marked acute inflammation and necrosis. She had an intra-aortic balloon pump, left ventricular and right ventricular assist devices placed on account of persistent hemodynamic instability, and is now scheduled to have a cardiac transplant.

<Learning objective: Coronavirus disease 2019 (COVID-19) is an inflammatory disease involving multiple organs including the heart. Myocarditis and cardiomyopathy are possible short-term and/or long-term sequelae of COVID-19 infection. In peripartum women with COVID-19 infection, it may be difficult to distinguish between peripartum cardiomyopathy and COVID-19 related cardiomyopathy. This case report highlights such a dilemma.>

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Introduction

The ongoing global pandemic caused by a novel severe acute respiratory syndrome; coronavirus-2 infection also known as coronavirus disease 2019 (COVID-19) is associated with multisystem dysfunction. Although most of the attention has been on the respiratory manifestation of COVID-19, cardiac complications have also been reported [1, 2]. Studies evaluating non-pregnant patients suggest that COVID-19 related myocarditis may progress to dilated cardiomyopathy and subsequent heart failure. COVID-19 related cardiomyopathy has been proposed as a prognostic factor in critically ill patients [2, 3].

Peripartum cardiomyopathy is a rare condition presenting with features of heart failure in the late third trimester or the early postpartum period. The incidence ranges from 1 in 102 deliveries in Nigeria to 1 in 15,533 births in Japan [4]. The etiology remains poorly understood. Theories such as peripartum hormonal changes, effects of prolactin, and myocarditis have been proposed.

Studies evaluating COVID-19-related cardiomyopathy among pregnant women are scarce. Given the similarities in presentation, differentiating between peripartum cardiomyopathy and COVID-19-related cardiomyopathy may be challenging in a COVID-19 positive peripartum patient. We present a case of cardiomyopathy presenting with new-onset heart failure in a peripartum woman with COVID-19 infection.

Case report

A 28-year-old Gravida 2 para 1 African American female with no significant past medical history and class III obesity presented

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Fig. 1. Chest X-ray showing cardiomegaly and bilateral pulmonary opacities consistent with congestive heart failure/ atypical or viral pneumonia.

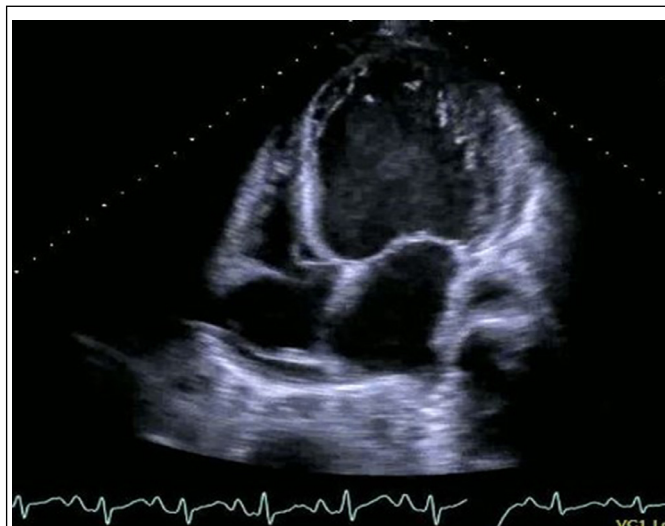


Fig. 2. Transthoracic echocardiogram showed global hypokinesis with a reduced left ventricular ejection fraction of 15%.

with severe shortness of breath 24 hours after spontaneous vaginal delivery of a single female neonate at term. She had no history of hypertension in pregnancy, gestational diabetes mellitus, kidney, or cardiac disease. She had no complications in the prenatal period, however, she complained of shortness of breath on exertion with bilateral leg swelling, which was attributed to term pregnancy about one week before delivery. She had no fever, chills, cough, or chest pain. The patient had not traveled outside the USA. Her vaccinations were up to date, and she had no sick contacts. She never smoked, drank alcohol, or used recreational substances. Physical examination on admission showed an obese young woman in respiratory distress. Blood pressure was 114/71 mmHg, pulse rate 129 beats per minute, respiratory rate 20 cycles per minute, temperature 36.4°C, and oxygen saturation 92% on 3 L oxygen. There was jugular venous distension and S1, S2, and S3 were heard with gallop rhythm. Pulmonary auscultation was remarkable for bibasilar decreased breath sounds. The abdomen was mildly distended and there was hepatomegaly on palpation. She had 3+ edema of the lower extremities, to the level of the thighs. Arterial blood gases showed PaO₂ 67 and PaCO₂ 26. Chest X-ray (Fig. 1) showed cardiomegaly with bilateral infiltrates suggestive of pulmonary vascular congestion. Electrocardiogram showed sinus tachycardia, heart rate 111 beats per minute, normal axis, and no ST-T wave abnormalities. B-type natriuretic peptide was 727 pg/ml (normal <125 pg/ml) and increased to 1261.7 pg/ml two days after admission, troponin was 0.07 ng/ml on admission, the trend remained flat over 6 hours and on-site screening COVID-19 test was positive. Inflammatory biomarkers were checked every 48 hours: Ferritin level was 9.30 ng/ml increased to a maximum of 158.0 ng/ml two days after admission (normal 8–252 ng/ml), lactate dehydrogenase was 309 U/L, increased to a maximum of 1200 U/L two days after admission (normal 81–234 U/L), and C-reactive protein was 3.92 mg/dl, increased to a maximum of >8 mg/dl two days after admission (normal 0–0.9 mg/dl), Procalcitonin 0.74 ng/ml (normal <0.5 ng/ml), increased to a maximum of 1.42 ng/ml two days after admission. Echocardiogram on admission revealed moderately dilated left ventricle with severe global hypokinesis and left ventricular ejection fraction (LVEF) of 22%. Chest computed tomography (CT) angiography showed numerous patchy ground-glass nodules in the right lung and no evidence of a pulmonary embolism. An initial diagnosis of peripartum cardiomyopathy with COVID-19 infection

was made, and the cardiology team was consulted. At this point, it was unclear if the patient had developed myocarditis secondary to COVID-19 which may have resulted in heart failure or had peripartum cardiomyopathy with an incidental finding of COVID-19 infection. The patient was given 40 mg of intravenous furosemide twice daily and completed 5 days of remdesivir and intravenous dexamethasone 6 mg daily. She was also given subcutaneous enoxaparin sodium 40 mg twice daily for venous thromboembolism prophylaxis. The patient's clinical status improved, and she was discharged thereafter. On postpartum day 10, she had worsening dyspnea and she was readmitted. Chest X-ray was unchanged when compared to the X-ray at initial admission. Repeat echocardiogram (Fig. 2) revealed further reduction in LVEF to 10–15%. A cardiologist recommended further diuresis with furosemide and started her on metoprolol. With the improvement in clinical status, she was discharged home with a Life-Vest [Zoll live vest, Pittsburgh, USA], furosemide, and metoprolol. Five days following discharge, the patient re-presented with further deterioration, chest pain, hemoptysis, and bilateral leg swelling. Heart rate was 112 bpm, respiratory rate was 46 cycles/minute, and blood pressure was 104/82 mmHg, temperature 36.7°C, and oxygen saturation 92% on room air. Pulmonary examination revealed reduced bibasilar breath sounds and she had bilateral pedal edema. Laboratory work-up at this time showed transaminitis; alanine aminotransferase >500 units/L, aspartate aminotransferase 964 units/L. B-type natriuretic peptide was elevated at 989 pg/ml, maximum troponins 0.073 ng/ml, and repeat COVID-19 test was positive. D-dimer was elevated at >7650 ng/ml (normal <500 ng/ml), procalcitonin 0.16ng/ml, lactate dehydrogenase 990 U/L, ferritin 53.50 ng/ml, interleukin-6 172.93 pg/ml (normal <5 pg/ml), and fibrinogen 251 mg/dl (normal is 200–393 mg/dl). Electrocardiograph showed atrial tachyarrhythmias with features of left atrial enlargement and right axis deviation, there were no significant ST-T wave segment changes. Chest CT angiography at this time showed diffuse pulmonary infiltrates worse on the right and sub-segmental pulmonary embolism. Heparin infusion was started per venous thrombo-embolism protocol. She was given digoxin, empiric antibiotics, and a 10-day course of 6 mg of dexamethasone. The clinical course continued to improve albeit complicated by acute kidney injury likely related to recent contrast exposure and possible cardiorenal syndrome. At discharge, she had achieved rate control, resolution of respiratory

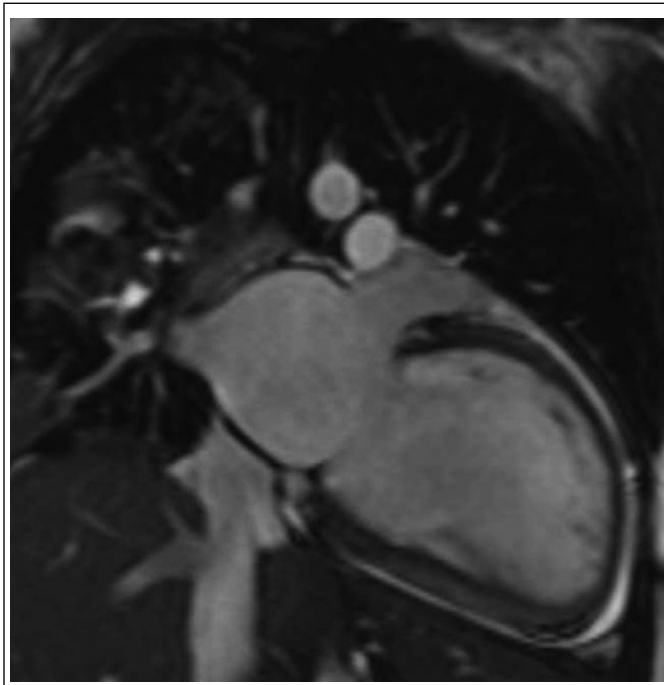


Fig. 3.

Cardiac magnetic resonance imaging showed severely dilated left ventricle with normal wall thickness and severely decreased left ventricular function, ejection fraction 10%, and moderately dilated right ventricle with severely decreased right ventricular, ejection fraction 17%. Findings were consistent with non-ischemic cardiomyopathy without evidence of macroscopic myocardial scarring or fibrosis.

distress, and improved renal function. Cardiac magnetic resonance imaging (Fig. 3) showed severely dilated left ventricle with normal wall thickness and moderately dilated right ventricle. LVEF was 10% and the right ventricular ejection fraction was 17%. There was also severely dilated left atrium and moderately dilated right atrium with mild to moderate pericardial effusion. Diffuse infiltrates were seen throughout the right lung and at the base of the left lung likely representing pulmonary edema. These findings were consistent with non-ischemic cardiomyopathy without evidence of macroscopic myocardial scarring or fibrosis. Myocardial core biopsy showed marked acute inflammation and necrosis. The patient continued to have relapsing episodes of acute decompensated heart failure and she had an intra-aortic balloon pump, left ventricular, and right ventricular assist device placed on account of persistent hemodynamical instability. The patient is scheduled to have a cardiac transplant.

Discussion

This case highlights a case of dilated cardiomyopathy that may be related to either peripartum changes or COVID-19 infection.

Cardiomyopathy is a less-discussed association of COVID-19. The exact mechanism of myocarditis in the setting of COVID-19 like many other associations of COVID-19 is not fully understood. The proposed mechanisms of myocardial injury from COVID-19 infection include ischemia due to respiratory and cardiovascular failure, myocyte necrosis from thrombotic obstruction of epicardial or intramyocardial small coronary arteries, and myocarditis caused by systemic inflammation or direct binding of the virus to angiotensin-converting enzyme 2 (ACE-2) receptors in the heart [5, 6]. ACE-2 receptors are highly expressed in the myocardium and vascular endothelium and are reported to be functional receptors

for COVID-19 [7]. Cardiomyopathy has been reported in about 33% of critically ill non-pregnant patients with COVID-19 [8], yet data on pregnancy-specific effects of COVID-19 are limited.

Peripartum cardiomyopathy is potentially life-threatening. It presents as congestive heart failure with left ventricular dysfunction with ejection fraction <45% in the peripartum period without previous known structural heart disease [9]. Volume overload coupled with vascular dysfunction triggered by hormonal changes in pregnancy is believed to be the etiology. Pregnancy is an immunocompromised state with increased but physiologic cardiovascular demands. This immunocompromised state increases the risk of infections such as COVID-19 and related complications.

The presence of dilated cardiomyopathy in this COVID-19 infected patient in the postpartum period raises the diagnostic dilemma of COVID-19-related cardiomyopathy versus peripartum cardiomyopathy. There have been a few case reports of COVID-19 infection in peripartum women. These include a report by Juusela et al. in which two cases of peripartum women with COVID-19 infection presented with findings of cardiomyopathy [10]. This case of peripartum cardiomyopathy in a peripartum patient is similar. Numerous questions remain unanswered, including: can COVID-19 infection increase the risk of peripartum cardiomyopathy? If so, does this give credence to the theory of myocarditis as a possible etiology of peripartum cardiomyopathy? What are the long-term effects of cardiomyopathy in patients infected with COVID-19? How does this affect future childbirth options? Could this be dilated cardiomyopathy from other causes?

Given the direct association between ACE-2 receptors and COVID-19 infection, and the reported prognostic indication of cardiac effects, further studies including long-term assessments are encouraged to evaluate cardiac function in COVID-19 infected patients.

Declaration of Competing Interest

The authors declare there are no conflicts of interest.

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