Peripartum Cardiomyopathy Leading to Multiorgan Dysfunction in a Previously Healthy 30-Year-Old Puerto Rican Woman

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Peripartum cardiomyopathy (PPCM) is a rare and potentially life-threatening cause of systolic heart failure that usually arises in the late stages of pregnancy or the early postpartum period. Its clinical presentation can range from moderate symptoms to severe forms of heart failure, such as cardiogenic shock. However, there is scarce data on severe forms of PPCM leading to multiple organ dysfunction and its management. We report a case of a 30-year-old G2P2 previously healthy Puerto Rican woman postpartum day #56 who presented to our Institution with respiratory symptoms. Further evaluation confirmed cardiogenic shock with ischemic hepatitis and acute kidney injury. Echocardiography revealed a dilated left ventricle with an ejection fraction of less than 20%, which suggested PPCM. This case highlights severe manifestations of PPCM, its challenges in management, and the importance of considering it in the differential diagnosis of respiratory symptoms during the peripartum period.

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Peripartum cardiomyopathy (PPCM) is a rare pregnancy-associated cardiac disease usually observed in the late third trimester of pregnancy and up to 5 months after delivery (1). The etiology of PPCM is unclear but likely multifactorial, involving genetics, viral infections, autoimmune reactions, and more. It affects about 1 in 2,000 births, with key risk factors including hypertension, pregestational diabetes, preeclampsia, and advanced maternal age (2). While data on PPCM in Puerto Rico is limited, a study of pregnancy-related deaths (2009-2018) showed higher cardiomyopathy-related deaths among Puerto Rican women, suggesting a potential higher incidence (3). A study at University District Hospital also identified risk factors like age over 30, multiparity, and preeclampsia in this population (4).

PPCM is a form of systolic heart failure with reduced ejection fraction that is diagnosed using echocardiography and in the absence of other potential causes (1). It usually presents acutely with nonspecific symptoms such as dyspnea, palpitations, fatigue, and cough. Such symptoms can be dismissed leading to the disease going underrecognized and resulting in devastating consequences of PPCM.

Case reports have documented many of these severe manifestations such as arrhythmias (5), thromboembolic events (6), and cardiac arrest (7). However, we are not aware of any reports describing multiorgan dysfunction due to PPCM and its management. Usually, PPCM is managed with the same guideline-directed medical treatment as heart failure, including intravenous diuretics, vasodilators, ACE inhibitors or ARBs, mineralocorticoid receptor blockers, and SGLT inhibitors. In cases of volume overload and hypoperfusion, inotropes are recommended. For patients resistant to medical therapy, mechanical support like ECMO, ventricular assist devices, or even heart transplantation may be required. Additionally, although not standard, bromocriptine has shown potential in improving LVEF in patients with LVEF <25% (8,9). Although treatment of PPCM is similar to heart failure in a non-peripartum patient, a therapeutic dilemma may arise when multiple organ systems are affected (10). This case highlights the presentation of multiorgan dysfunction in a postpartum patient due to PPCM and reveals the challenges in its treatment.

Case report

A 30 y/o G2P2002 Puerto Rican female without any past medical or surgical history and BMI 29.2 kg/m^2 presented to our Institution on postpartum day #56 with cardiopulmonary symptoms. The patient had her delivery in New York and moved to Puerto Rico on postpartum day #51. The patient's obstetric history included two vaginal deliveries at term, with the most recent complicated by a lack of postpartum follow-up. Her family history was notable for hypertension in her parents but otherwise unremarkable. The patient denied any additional complications, such as hypertension, preeclampsia, diabetes, or

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postpartum hemorrhage. She reported not having any postpartum care and denied any pregnancy complications. On postpartum day #45, she sought medical attention at another medical facility due to a dry cough, malaise, and chills. At that time, she was discharged home with normal labs. However, her symptoms progressed which prompted her to visit our Institution's Emergency Department. She referred experiencing fatigue, shortness of breath, dyspnea, and palpitations. She denied fever, headache, epigastric pain, or visual disturbances. Vital signs upon arrival were: blood pressure 100/72 mmHg, heart rate 78 bpm, respiratory rate 15 rpm, temperature 36.6 °C, and oxygen saturation 98%. Upon physical exam, the patient appeared acutely ill, pale with slow mentation and in respiratory distress. The cardiovascular exam was remarkable for jugular-venous distention, distant heart sounds with regular rate and rhythm. Lung auscultation revealed poor inspiratory effort and bilateral crackles up to mid-lung. Extremities presented bilateral pedal edema. Chest X-Ray upon arrival showed an enlarged heart (Figure 1) and 2D echocardiogram revealed severe dilation of the left ventricle, ejection fraction of < 20% and global hypokinesia (Figure 2). Further laboratories were remarkable for elevated creatinine (1.04), liver enzymes (AST: 2,092 and ALT: 1,579), troponins (3,358.91), B-type natriuretic peptide (1,312), and lactate (6.28). Electrocardiogram showed normal sinus rhythm without ischemic changes. The patient was admitted to the intensive care unit (ICU) due to acute heart failure in cardiogenic shock and need of vasopressors.

Figure 1. Chest X-Ray upon arrival with bilateral vascular congestion and increased cardiothoracic index suggestive of cardiomegaly. Small bilateral costophrenic effusions noted and no thoracic fractures.

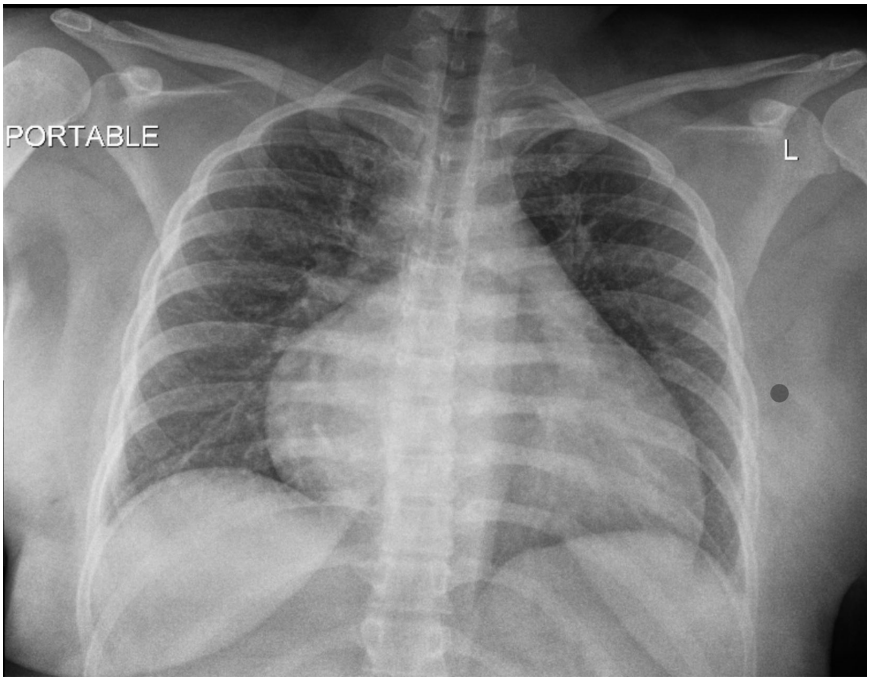


Figure 2. Diagnostic echocardiogram upon arrival. Apical 4-chamber view demonstrating severe ventricular dilatation (A). Parasternal long axis view demonstrating severe left ventricular (LV) and left atrial (LA) dilatation (B).

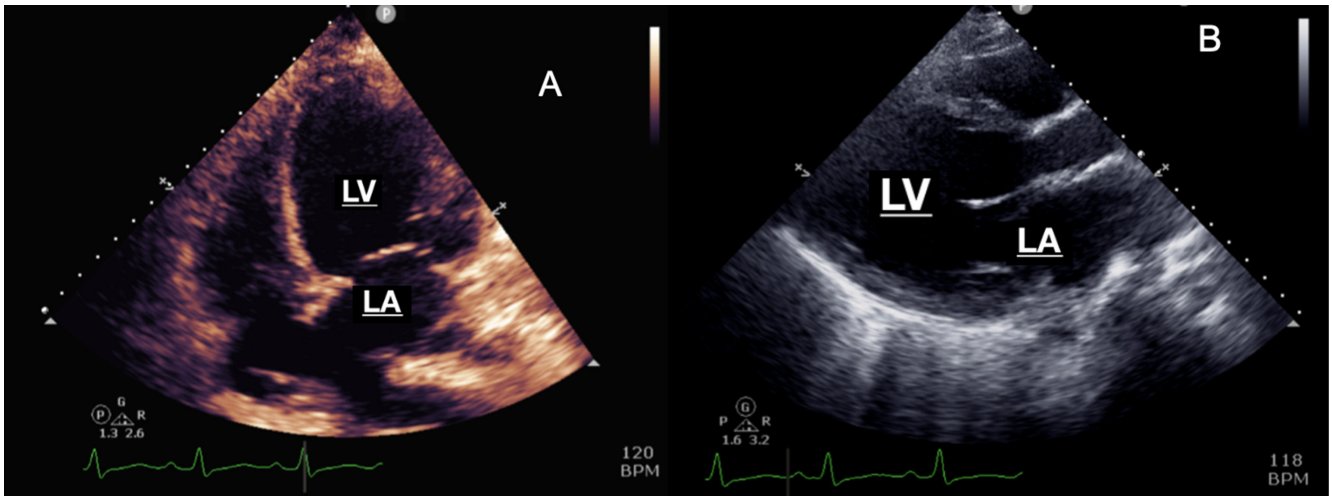
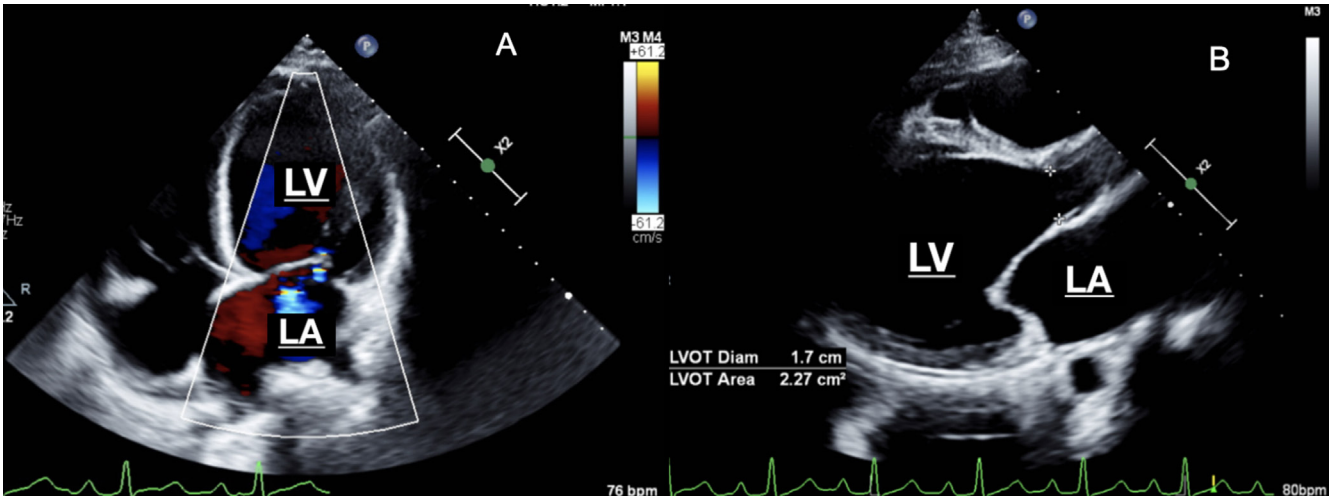


Figure 3. Follow up echocardiogram after 3 months of GDMT, bromocriptine, and life vest use. Parasternal long axis view with no change in comparison to initial view (A). Apical 4-chamber view demonstrating severe LV and LA dilation (B).



Chest CT angiography showed no evidence of pulmonary embolism. The patient was placed on deep vein thrombosis prophylactic anticoagulation and wore pneumatic compression stockings for the remainder of her admission. Additional laboratories such as toxicology screen, autoimmune disorders (Systemic lupus erythematosus, Sjogren's, Autoimmune hepatitis), thyroid disorders, and infectious diseases (Hepatitis, HIV, Influenza, Mycoplasma, Leptospira, COVID) were performed to rule out causes of heart failure, hepatitis, and acute kidney injury, before considering the diagnosis of peripartum cardiomyopathy and were all found negative. The patient declined a minimally invasive procedure to assess cardiac hemodynamics. Efforts to reduce vasopressors were slow, and ECMO was considered but not available due to limited access on the island, so medical management continued. Once the patient was successfully weaned off vasopressors, slowly started on GDMT, and transferred from ICU to telemetry ward. Repeat 2D echocardiogram was found unchanged from initial evaluation and a LifeVest was placed. The patient was advised on the importance of contraception due to the high risks associated with pregnancy in her condition, leading to the decision to insert a levonorgestrel IUD. At this time, the patient was receiving full GDMT and expressed symptomatic relief and was clinically stable for which she was discharged home from the hospital with LifeVest in place, and instructions to continue GDMT and bromocriptine therapy, and close follow-up.

On the 3-month cardiology follow-up, a 2D echocardiogram was repeated and revealed continued global hypokinesia and severely depressed LVEF. Due to those findings, the patient was referred to an Electrophysiologist for pacemaker placement vs. heart transplant consideration. The patient was lost to follow-up and further information could not be obtained.

Discussion

Peripartum cardiomyopathy is a life-threatening illness that, if not detected and treated promptly, could lead to severe complications such as permanent cardiac dysfunction and death. This case describes the diagnostic process of a patient with severe PPCM (EF <20%, ischemic hepatitis and acute kidney injury) and the challenges in management when balancing the treatment of cardiogenic shock with vasopressors and inotropes and GDMT for heart failure with reduced ejection fraction which, not only lower blood pressure, but also decrease cardiac contractility. In this case, GDMT was delayed until the patient's blood pressure stabilized enough for the medications. Due to limited ECMO availability on the island, medical management was optimized instead. Bromocriptine, though not standard, was added to the treatment regimen as studies showed it could improve ejection fraction in patients with LVEF <25% (11,12). Also, although not commonly used in cases of PPCM, in the case of severe illness right heart catheterization could have provided further information on other potential causes of PPCM and better monitoring of cardiac hemodynamics (13). The patient's hepatic and kidney injury, caused by cardiogenic shock, improved with restored organ perfusion. After ruling out other causes of heart failure, PPCM was confirmed. However, after 3 months of treatment, the echocardiogram showed minimal improvement in ejection fraction (less than 20%), leading to a referral for potential intracardiac device placement to prevent fatal arrhythmias. Even though there is significant data to show GDMT is the best way to manage PPCM, there is a lack of evidence of treatment of simultaneous cardiogenic shock and PPCM. In this patient, likely due to the severity of her disease at the time of her presentation, GDMT and bromocriptine did not appear to improve her condition and would have probably benefited from right heart catheterization and ECMO. Therefore, our case is evidence of the severe outcomes of this condition if left untreated for which we

argue the best way to manage these patients is with early detection and management to prevent irreversible deterioration.

Resumen

La cardiomiopatía periparto (CMPP) es una condición poco común y potencialmente mortal que causa insuficiencia cardíaca sistólica, y generalmente se manifiesta en las etapas tardías del embarazo o en las primeras etapas del posparto. Su presentación clínica puede variar desde síntomas moderados hasta formas más severas de insuficiencia cardíaca, como el shock cardiogénico. No obstante, la información sobre formas graves de CMPP que resultan en disfunción de múltiples órganos y su manejo es limitada. Presentamos el caso de una mujer puertorriqueña de 30 años, G2P2002, previamente saludable, que acudió a nuestra Institución en el día postparto #56 con síntomas respiratorios. Evaluación adicional confirmó colapso cargiogénico con hepatitis isquémica y lesión renal aguda. La ecocardiografía reveló un ventrículo izquierdo dilatado con una fracción de expulsión inferior al 20%, indicando la presencia de CMPP. Este caso destaca una de las manifestaciones severas de la CMPP, los desafíos en su manejo y la importancia de considerarla en el diagnóstico diferencial de los síntomas respiratorios durante el periodo periparto.

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