Peripartum-related conditions – treat one for the sake of two

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KEYWORDS: cardiomyopathies, maternal mortalities, pregnancy complications.

CITATION: Cardiol Croat. 2025;20(5-6):137. | https://doi.org/10.15836/ccar2025.137

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Introduction: Peripartum cardiomyopathy (PPCM) is characterized by symptomatic left ventricular (LV) systolic dysfunction with ejection fraction (EF) usually <45%, with or without LV enlargement, developing during the last month of pregnancy or in the first 5 months after delivery, abortion, or miscarriage in women without previously known heart disease[1]. Diagnosis is made after exclusion of other causes of LV dysfunction that are encompassed in a wider term of peripartum-associated cardiomyopathies (PPAC). They vary in etiology, including multiple acquired and inherited disorders[2-4]. We present a case series of multiple PPACs.

Case series: The first case is a 45-year-old woman with a history of multiple pregnancies, hospitalized in 2016 for heart failure (HF), three months postpartum. Echocardiography confirmed significantly reduced LVEF of 20% with LV dilatation. An invasive and noninvasive cardiology workup yielded normal results. Cardiac MRI confirmed reduced LV function with no fibrosis. With regular monitoring and therapy, LV function recovered (EF 60%) within a year. She is now asymptomatic.

The second case involves a 37-year-old woman hospitalized in 2024 for HF, two weeks postpartum. She had previously been diagnosed with congenital LV apical aneurysm. Echocardiography showed preserved LV function (EF 55%) and asymmetric hypertrophy of the interventricular septum, primarily at the midventricular level. CT coronary angiography identified an anomalous origin of the right coronary artery. Cardiac MRI confirmed hypertrophic obstructive cardiomyopathy (HCM). Further testing showed no significant pressure gradient across the LV. Genetic testing confirmed a hereditary form of HCM. The patient is now stable and under treatment.

Lastly, the most recent case involves a 21-year-old woman hospitalized for cardiogenic shock and preeclampsia, one day postpartum. She required mechanical ventilation and vasopressor support. Echocardiography confirmed significantly reduced LV function (EF 30%). After brief stabilization, she experienced cardiac arrest, was resuscitated and emergency percutaneous implantation of veno-arterial extracorporeal membrane oxygenation followed. Her condition was further complicated by multiple surgical interventions due to a retroperitoneal hematoma, multi-organ failure, and ultimately, cerebral edema with intracerebral hemorrhage. Despite intensive treatment, she died a month later.

Conclusion: This case series demonstrates the heterogeneity of PPACs, ranging from reversible PPCM to previously unrecognized HCM and finally, cardiomyopathy associated with preeclampsia. The outcomes varied from full recovery to maternal death. Etiology-specific understanding is essential for optimizing both maternal and fetal outcomes in these high-risk scenarios.

RECEIVED:
March 2, 2025
ACCEPTED:
April 2, 2025

LITERATURE

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