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When Hypertension Meets Heart Failure: A Case of Preeclampsia and Peripartum Cardiomyopathy (Poster Presentation)

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Abstract

Introduction:
Peripartum cardiomyopathy (PPCM) is a serious form of heart failure occurring in late pregnancy or the early postpartum period and is defined by new-onset left ventricular systolic dysfunction without prior structural heart disease. Its pathophysiology is multifactorial and overlaps with preeclampsia through shared mechanisms including endothelial dysfunction and anti-angiogenic signaling, creating heightened risk for pulmonary edema, arrhythmias, and hemodynamic instability. Structural inequities further delay recognition and limit access to specialized obstetric and cardiovascular care.

Case Description:
A 26-year-old G5P2032 female presented to the emergency department one week postpartum following an uncomplicated spontaneous vaginal delivery at 38w5d with two days of chest pain, dyspnea at rest, headache, bilateral pedal edema, and urinary frequency. Her history included chronic hypertension on labetalol, superimposed preeclampsia, gestational thrombocytopenia, depression, HSV2, and marijuana use. She was hypertensive to 183/129 mmHg with bilateral lung crackles and visible edema. Workup showed elevated troponin (149→169 pg/mL), elevated BNP (1302 pg/mL), and a protein/creatinine ratio of 0.39. Chest radiograph demonstrated cardiomegaly with pleural effusions and diffuse opacities consistent with pulmonary edema. Bedside echocardiography suggested a moderate pericardial effusion, and transthoracic echocardiogram confirmed reduced systolic function with left ventricular ejection fraction of 40–45% and mild hypokinesis, supporting PPCM.

Initial treatment included IV antihypertensives, magnesium, electrolyte repletion, and diuresis. She was admitted with continuous telemetry and managed collaboratively by Obstetrics/Gynecology with Cardiology and Internal Medicine consultation. Cardiology initiated guideline-directed medical therapy (GDMT) for PPCM (carvedilol, sacubitril-valsartan, empagliflozin, spironolactone, and furosemide), and pregnancy-directed antihypertensive agents were discontinued. Breastfeeding implications were reviewed with Maternal-Fetal Medicine and Cardiology; the patient chose to continue heart failure therapy and avoid breastfeeding. She stabilized quickly and was discharged on hospital day two with anticoagulation prophylaxis and close outpatient follow-up. At four months postpartum on GDMT, she had no recurrent admissions or symptoms and a repeat echocardiogram showed improvement in her ejection fraction to 55-60%.

Discussion:
This case demonstrates the clinical complexity of distinguishing PPCM from postpartum preeclampsia with severe features, as both can present with dyspnea, edema, pulmonary congestion, and hypertension. Echocardiography was pivotal in identifying reduced ejection fraction and confirming PPCM. The coexistence of superimposed preeclampsia may represent a synergistic “double hit,” where pregnancy-related vascular and hormonal stressors combine with endothelial dysfunction and anti-angiogenic signaling to worsen maternal cardiovascular risk. Rapid symptomatic improvement following early initiation of guideline-directed heart failure therapy reinforces the importance of prompt diagnosis, telemetry monitoring, and coordinated multidisciplinary care. Additionally, the patient’s emotional distress related to newborn separation and the need to discontinue breastfeeding highlights psychosocial considerations that directly shape postpartum decision-making and adherence; these challenges can be amplified by structural barriers to timely access and continuity of specialized care.

Conclusion:
PPCM with superimposed preeclampsia poses significant diagnostic and management challenges due to overlapping presentations and compounded hemodynamic risk. Early echocardiographic assessment,

immediate initiation of guideline-directed heart failure therapy, and multidisciplinary coordination were key to favorable outcomes in this patient. Improving maternal outcomes requires not only clinical vigilance but also attention to psychosocial needs and systemic inequities that contribute to delayed recognition and disparities in postpartum cardiovascular care.