Preeclampsia and Peripartum Cardiomyopathy in a 24-Year-Old Woman

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ABSTRACT

Herein we report the case of a 24-year-old woman who presented with gestational hypertension and a history of preeclampsia with a previous pregnancy as well as a family history of preeclampsia and eclampsia. She subsequently developed a definitive preeclampsia with proteinuria in the 30th week of gestation, which necessitated a caesarean delivery. Two weeks postpartum the patient developed dyspnea with bilateral pulmonary infiltration and was hospitalized for an atypical pneumonia. After completion of an antibiotic course she continued to experience dyspnea and orthopnea and ultimately developed systolic heart failure with an ejection fraction of 30%. Following treatment with diuresis and rate control, she greatly improved. This case demonstrates the importance of close monitoring of patients with atypical preeclampsia by a primary care and coordinated specialties team. It demonstrates a sequential progression of worsening hypertension, preeclampsia, and peripartum cardiomyopathy driven by the underlying mechanism of elevated systemic vascular resistance. It also proposes an association of peripartum cardiomyopathy with increased gestational body mass index (BMI).

INTRODUCTION

Preeclampsia affects 4.6 percent (95% confidence interval [CI] 2.7-8.2) of pregnancies worldwide.¹ The criteria for the diagnosis of preeclampsia include an elevated blood pressure occurring after 20 weeks of gestation and proteinuria (≥0.3 g of protein in a 24-hour urine sample, a protein (mg/dL):creatinine (mg/dL) ratio of ≥ 0.3 or a 1+ dipstick).² In patients with new onset hypertension, signs of end organ damage may be substituted for proteinuria to make the diagnosis of preeclampsia. In most cases symptoms develop before 34 weeks of gestation. Early-onset preeclampsia occurs in 10% of patients and is associated with an increased morbidity to the mother and fetus, increasing the risk of fetal death greater than five-fold and increasing the risk of perinatal death/severe neonatal morbidity sixteen-fold.³⁴ Our patient presented with atypical signs of preeclampsia at 25 weeks of gestation but did not meet definitive criteria.

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until 30 weeks of gestation, at which point delivery was induced due to the development of placental insufficiency and intraterine growth restriction. Preeclampsia presentation is highly variable and a high index of suspicion for development during the third trimester must be maintained, especially in the setting of multiple risk factors as was the case for our patient. Preeclampsia may lead to eclampsia, the development of tonic-clonic seizures and coma during delivery that can result in fetal and/or maternal demise. Peripartum cardiomyopathy is a form of systolic heart failure which affects 1/1300-4000 live births. Cardiomyopathy can develop in a small subset of preeclampsia cases; it is important to consider this etiology in the setting of pulmonary infiltration consistent with transudative versus exudative fluid. If untreated, peripartum cardiomyopathy can lead to atrial or ventricular arrhythmia, thromboembolism, or sudden cardiac death. An involved and mindful primary care and coordinated specialties team is essential to the successful case management of the preeclamptic patient.

CASE PRESENTATION

A 24-year-old African American woman first presented at 19 weeks gestation to clinic after an emergency department (ED) visit for abdominal pain and a blood pressure of 190/90 mmHg (Table 1). Upon initial presentation she was normotensive and not experiencing pain. The patient had a positive family history of preeclampsia and eclampsia; her mother had an eclamptic episode and miscarriage of twins within a span of five normal pregnancies. The patient’s first pregnancy was complicated by preeclampsia, requiring induction at 36 weeks of gestation.

The patient continued to return for prenatal visits and at 28 weeks presented with elevated home blood pressures, 6/10 migraine with bilateral visual scotomas, unremitting abdominal pain, 2+ pitting pedal edema, 1+ facial and hand edema, and trace urine protein. The patient’s weight had continued to increase, now measuring 219 pounds as compared to a pre-pregnancy weight of 180 pounds. She was noted to have gained 12 pounds within the last 2 weeks. Fundal height was 26 cm and fetal heart tones were in the range of 140 beats per minute. At that time the patient was started on labetalol 100 mg and referred to a perinatologist.

An ultrasound conducted at 29 weeks revealed a single fetus in a vertex position with an estimated weight of 2 pounds, 9 ounces (less than 5% of normal fetal growth). As previous scans had revealed the fetus to be at the 25th percentile for growth, intraterine growth restriction became a primary concern. The ultrasound also revealed a posterior, premature grade III placenta with extensive basal calcifications and a chorionic plate interrupted by indentations, a finding which could lead to placental insufficiency. The patient was admitted at that time for preeclamptic evaluation and received a course of beclomethasone to expedite fetal lung maturity in anticipation of premature delivery. Labetolol was increased to 200 mg.

At 30 6/7 weeks of gestation, the patient presented to the ED with headache, scotomata, and blurred vision. She was found to have a blood pressure of 160/110 mmHg and 2+ proteinuria. Perinatology noted that her amniotic fluid volume was decreased markedly, with an amniotic fluid index (AFI) of 6.5 cm as compared to 7.9cm-27.3 cm from 15-40 weeks gestation. At this time it was noted the patient was experiencing irregular contractions. Pelvic examination confirmed cervical dilation of 1-2 cm. After increasing labetalol to 300 mg, the amniotic fluid volume improved to an AFI of 9 cm. However the fetal biophysical profile score was 6, with no evidence of fetal breathing, including in re-
Table 1. Vital Signs, Fetal Monitoring, Laboratory Results, Imaging, Clinical Signs, and Treatment in Pre- to Postpartum Period

Based on clinical signs and radiology, peripartum cardiomyopathy was a concern and the patient was sent to the cardiology outpatient clinic for echocardiography. The patient, however, did not present to the cardiology clinic for follow-up.

Three days later, the patient presented to the ED with a complaint of dyspnea, subjective fever, and chills. She was found to have an uncompen-

Figure 1. Chest radiograph demonstrating right middle lower lobe pulmonary infiltration and concurrent cardiomyopathy.
sated respiratory alkalosis with a pH of 7.5, pCO2 of 24, bicarbonate of 18, pO2 of 67 and a hemoglobin saturation of 92.7. She also had an extremely elevated d-dimer of 2.21 μg/mL (normal values= <0.5 μg/mL), an increased platelet count of 480/mm³, an elevated troponin of 0.05 ng/mL, and a slightly elevated chloride of 110 mEq/L. Uric acid was also elevated at 7.7 mEq/L, which could indicate renal dysfunction due to systemic shock. The patient continued to have a decreased O₂ saturation on exertion.

Sputum gram stain showed elevated polymorphonuclear cells and moderate respiratory flora while chest radiography continued to display the right middle lobe consolidation. The patient was treated with amoxicillin/clavulanic acid and naproxen; she continued to improve while hospitalized. The patient’s respiratory symptoms resolved with treatment of the atypical pneumonia, and she was discharged three days later. Echocardiogram at this time found trace tricuspid regurgitation and trace pulmonary insufficiency with a pulmonary arterial pressure of 30 mmHg. The mitral valve E-F slope was 217 mm/sec and the D-E separation was 22 mm indicating a mild to moderate mitral valve regurgitation with an E/A ratio of 1.5. The left atrium was found to be enlarged at 4.2 cm (normal range 1.9-3.8 cm). Left ventricular ejection fraction at this time, however, was preserved at 60%.

Thirteen days later the patient presented to her primary care practitioner for follow-up of the pneumonia. After completing the antibiotic course, the patient continued to experience dyspnea and cough exacerbated by the supine position. She reported expectorating a clear fluid from the lungs and was tachypneic. She was then referred to a pulmonologist to follow-up for pneumonia and investigate alternative causes of the dyspnea.

Eight days later the patient presented to the pulmonology clinic. At that time she reported significant dyspnea on exertion after walking less than 50 feet as well as significant paroxysmal nocturnal dyspnea with 4-pillow orthopnea. She had a cough productive of white sputum but no complaint of fever or chills. Upon physical exam she had apparent jugular venous distension and bilateral basilar crackles but did not have wheezing or ronchi. Cardiovascular exam revealed an S3 gallop and 2+ lower extremity edema. A chest radiograph showed a mildly enlarged cardiovascular silhouette with diffuse bilateral airspace opacities. The patient was admitted to the hospital, referred to a cardiologist, and was started on furosemide 40mg.

The echocardiogram completed the next day demonstrated mild tricuspid regurgitation, trace pulmonary insufficiency with a pulmonary arterial pressure of 40 mmHg, and moderate mitral regurgitation with an ejection fraction of 30%. At that time she was diagnosed with systolic heart failure exacerbation of peripartum cardiomyopathy and started on a beta blocker.

**DISCUSSION**

Preeclampsia results in uteroplacental hypoxia, an imbalance in angiogenic and anti-angiogenic proteins, oxidative stress, maternal endothelial dysfunction, and elevated systemic inflammation (Figure 2). It is accompanied by increased sensitivity of the maternal vasculature to pressor agents leading to vasospasm and hypoperfusion of multiple organs. Microthrombi develop from the activation of the coagulation cascade. Vasodilation results in plasma leakage, causing edema. The pathogenesis is thought to arise from placental insufficiency secondary to failure of the trophoblast to invade the myometrium. There is decreased placental secretion of the vasodilatory and growth factors adrenomedulin, prostacyclins, thromboxane
A2, and vascular endothelial growth factor (VEGF). The expression of the angiotensin I receptor is increased, resulting in a decrease in nitric oxide and increase in endothelin-1, causing an increase in maternal systemic vascular resistance in an attempt to improve placental perfusion. Inflammatory mediators such as tumor necrosis factor-alpha and interleukin-6 are also secreted which drive maternal hypertension, endothelial dysfunction, and oxidative stress. Cerebral edema and hypertension can result in the seizures and coma of eclampsia.

Our patient did not meet the definitive criteria for the diagnosis of preeclampsia before 30 weeks of gestation. She had elevated blood pressures which elicited clinical signs of hypertension such as headache, scotomata, and pulmonary edema, which are considered signs of severe preeclampsia beginning at week 25. She presented with worsening hypertension throughout gestation superimposed upon a background of chronic hypertension but did not present with signs of end organ damage such as platelet count <100,000/uL, serum creatinine >1.1 mg/dL, or liver transaminases twice the normal concentration. She did have certain risk factors for preeclampsia, such as preeclampsia with her first pregnancy (increases the risk 7-fold; relative risk 7.19, 95% CI 5.85-8.83), family history of preeclampsia (mother with history of eclampsia), blood pressure >130/80 mmHg at the first prenatal visit, BMI >26, and black race. Our patient presented with an atypical preeclampsia, with early clinical signs in the absence of diagnostic criteria and then rapid development of a definitive early-onset preeclampsia at 30 weeks gestation, which has a particularly bad prognosis. This development highlights the importance of close monitoring in patients with preeclampsia risk factors which may not present with the classic criteria for preeclampsia diagnosis.

Systolic heart failure can result from decompensation of a dilated left ventricle which had previously maintained normal ejection fraction by increasing left ventricular end diastolic pressure and end diastolic volume. The elevated systemic vascular resistance becomes an afterload that the left ventricle can no longer approximate, and eventually the ejection fraction can no longer be maintained. Mitral valve regurgitation results due to chamber dilation, and an early diastolic filling S3 may be heard. The elevation of systemic vascular resistance that occurs in preeclampsia can contribute to the development of peripartum cardiomyopathy. As the pressure in the left ventricle increases, it is transmitted to the pulmonary veins and lungs, resulting in fluid transudation into the alveolar spaces and pulmonary edema. Alveolar macrophages that engulf transudated erythrocytes and proteins are termed “heart failure cells” (Figure 3). The phenomenon of excess pulmonary arterial pressure elicits pulmonary edema, particularly in the postpartum period in the preeclamptic patient. Our patient did develop an elevated pulmonary arterial pressure of 40 mmHg at thirty days postpartum, which contributed to the systolic heart failure. In addition to this mechanism, there is evidence that other factors play a role. Systolic strain was found to be depressed in preeclamptic...
patients compared to pregnant women with non-proteinuric hypertension with similar resting blood pressure.\textsuperscript{16}

It is important to consider the etiology of peripartum cardiomyopathy in patients presenting with pneumonia-like vs. transudative symptoms. Our patient was treated ten days post-partum for pneumonia with only normal respiratory flora cultured. It was atypical, possibly caused by an imbalance of normal flora within the pulmonary interstitium and a nidus for infection created by the presence of excess basilar fluid. The patient’s dyspnea did not resolve upon completion of an antibiotic course. At that time, mild cardiomegaly was noted on chest x-ray and a cardiogenic pulmonary edema was investigated. Thirty days post-partum the patient was noted to have clinical signs of heart failure. A diuretic was started and the patient’s clinical features improved markedly. She then developed moderate mitral regurgitation, mild tricuspid regurgitation, pulmonic insufficiency, and an ejection fraction of 30%. The ejection fraction during and after pregnancy had previously been preserved at or above 53%. The patient’s brain natriuretic peptide (BNP) was elevated to 397 pg/mL at 31 days post-partum, supporting the diagnosis of systolic heart failure.

The patient’s development of systolic heart failure in conjunction with preeclampsia throughout the pregnancy was unexpected but promptly treated with rapid amelioration of symptoms. This fortunate outcome highlights the importance of close monitoring and integrated continuity of care among the primary care and specialty consulting physicians of the treatment team. Awareness of potential risks and complications can allow for rapid diagnosis and prompt treatment. Prophylaxis against seizures during delivery is of the utmost importance and would have prevented morbidity to the mother and fetus in less closely monitored situations.

Obesity and an elevated BMI are associated with an increased risk for preeclampsia.\textsuperscript{17} Prenatal counseling for lower calorie diets and moderate exercise before and during pregnancy may help decrease the severe risks of gestational hypertension and preeclampsia to mother and fetus. The importance of awareness among physicians of the risks of these clinical features in healthy-appearing gravid young women, as well as continuity among primary and specialty care providers is the key to a safe pregnancy for women with preeclampsia.

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**LEARNING POINTS**

- Acute on chronic hypertension in pregnant women should be closely monitored due to the potential late-term development of preeclampsia.

- Development of peripartum cardiomyopathy, which can present with respiratory distress, is a risk associated with preeclampsia.

- Preeclampsia is associated with intrauterine growth retardation that may necessitate preterm delivery and intrapartum seizure prophylaxis.
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14. Courtesy of Dr. Bradley Cheek, Louisiana State University Health Sciences Center New Orleans Department of Pathology, 19 Dec 2014.

