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Dyspnea After Delivery: A Case of Postpartum Cardiomyopathy

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Abstract

Peripartum cardiomyopathy is an uncommon and potentially devastating disease, with unacceptably high rates of morbidity and mortality. Early recognition and prompt intervention is critical for optimal outcomes in this special demographic. Here, a case is described of a 33-year-old female who presented with progressively worsening dyspnea several months after an induced delivery secondary to pre-eclampsia. Further investigation revealed the cause to be cardiomyopathy with a significantly depressed cardiac function.

Keywords: Cardiomyopathy; Disease; Dyspnea; Hemoptysis; Edema

Case Report

A 33-year-old African American female presented to the emergency department for evaluation of worsening dyspnea with a nonproductive cough over the past several days. She had been in her normal state of health prior, and denied any pain, fevers, chills, hemoptysis, nausea, vomiting, lower extremity edema, orthopnea, paroxysmal nocturnal dyspnea, or weight gain. The dyspnea was exertional in nature, without a pleuritic component. She denied recent travel or hospitalizations, except for a short hospital stay for pre-eclampsia four months prior for which she had a cesarean section at 36 weeks gestation. The patient reported a benign postpartum course, and admitted compliance with her only medication, labetalol. Other pertinent medical history includes a stillbirth at 26-months gestation several years ago. She denied a personal or family history of venous thromboembolic disease, coronary artery disease, heart failure, diabetes, or malignancy. She denied tobacco, alcohol, or illicit drug use.

The patient appeared comfortable on initial evaluation; initial vital signs were significant for a resting hypertension (185/106) and sinus tachycardia (119). The respiratory examination was notable for mild conversational dyspnea; equal chest expansion without rales, rhonchi, wheezing, stridor, or accessory muscle use. There was no jugular venous distension, extra heart sounds, lower extremity edema, calf tenderness, or varicosities. Examination was otherwise unimpressive.

The electrocardiogram was significant for sinus tachycardia and T-wave inversions laterally with borderline left ventricular hypertrophy (no priors were available for comparison). A chest X-ray was performed (Figure 1). The complete blood count and basic metabolic panel were unremarkable; because of an elevated D-dimer (2.96 μg/mL; reference: < 0.49 μg/mL), a CTA of the chest with PE protocol followed (Figure 2).

A diagnosis of acute decompensated heart failure secondary to postpartum cardiomyopathy was made. Nitroglycerin paste was applied, and furosemide was given to the patient. A set of cardiac enzymes were then ordered and found to be negative. BNP was 305 pg/mL (reference < 100). She was admitted to the cardiology service. An echocardiogram showed a left ventricular ejection fraction of 20%, mild pulmonary hypertension (40 mmHg), left atrial and ventricular enlargement, and a small pericardial effusion.

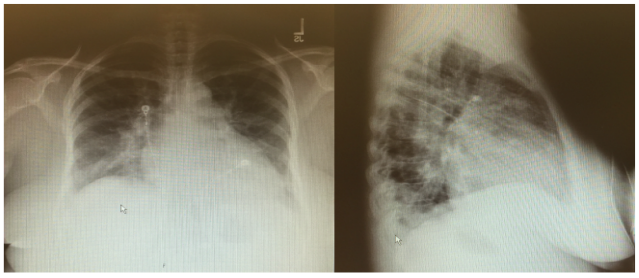

Figure 1: PA and lateral X-ray views of the chest, notable for interstitial prominence.

The cardiology service initiated beta-blockade therapy (carvedilol), losartan, and furosemide; and the patient was counselled to stop breastfeeding. She improved clinically, and was discharged on hospital day 3 with instructions for follow-up for repeat echocardiography in 3-6 months. There was also discussion for placing a Life-Vest if her symptoms did not improve.

Discussion

Peripartum cardiomyopathy may be a devastating disease process to the mother and fetus. Although recognized as a potentially life-threating clinical entity, there is a dearth of literature available on the etiologic, diagnostic, therapeutic, and prognostic planes. According to the Heart Failure Association of the European Society of Cardiology Working Group, peripartum cardiomyopathy is "an idiopathic

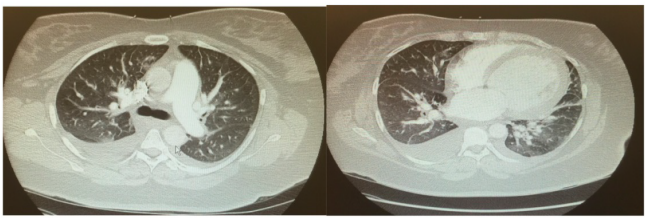

Figure 2: CTA of the chest PE protocol (lung windows) at the levels of the carina and mitral valve: bilateral pleural effusions enlarged bilateral interlobular septal thickening, patchy bilateral upper lobe ground-glass opacities, and a small pericardial fluid. No evidence of pulmonary embolism.

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cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy, or in the months following delivery, where no other cause of heart failure is found" [1].

Although no definitive etiology has been found, some have postulated a genetic component [2], autoantibodies against myocardial proteins [3], and low selenium levels may lead to this debilitating disease. Viruses have also been implicated, including parvovirus B19, Epstein-Barr, cytomegalovirus, and human herpes virus 6.

Risk factors for developing peripartum cardiomyopathy include multiparity, advanced maternal age, twin pregnancy, a history of miscarriages, African descent, and pregnancy-induced hypertension. It is interesting to note that, although preeclampsia is a risk factor [4], some reports have found it to be the cause of cardiomyopathy itself, with an ejection fraction as low as 10-15%. Follow-up echocardiograms in 6 months were normal, however.

Patients usually presents with dyspnea, orthopnea, paroxysmal nocturnal dyspnea, fatigue, chest pain, and cough (i.e., the classic symptoms of systolic dysfunction). On auscultation, an exaggerated S1, presence of an S3 (although this may be a normal finding in pregnancy), and a loud pulmonic component might be heard. Rales may be auscultated as well. (It is interesting to note that tidal volumes are often increased in normal pregnancy due to endogenous progestins, but tachypnea is not normally found). More telling are tachycardia, hypoxia, and tachypnea. Jugular venous distension may be evident on examination. Peripheral edema is found in upto a third of normal, healthy pregnant women, and therefore is not a sensitive or specific finding.

As clinicians, it is important to maintain a broad differential approach. Thromboembolism valvulopathy (included aortic/mitral stenosis), coronary artery disease, other forms of cardiomyopathy, malignant hypertensive disease (including preeclampsia), as well as cardiogenic and noncardiogenic forms of pulmonary edema must be considered. Electrocardiography classically shows sinus tachycardia with left ventricular hypertrophy and nonspecific ST-T wave abnormalities. An echocardiogram usually demonstrates chamber enlargement and a diminished left ventricular ejection fraction (usually < 45%). Both of these findings were present in our case.

If uncertain and the clinician has a high clinical suspicion for coronary disease, a stress echocardiogram is preferred. Thallium stress test is also available, although there exists a minimal risk for fetal radiation exposure. In severe cases, right and left heart catheterization may be instituted. Several cases reports found some utility of magnetic resonance imaging. Although myocarditis is often the cause of peripartum cardiomyopathy, routine enodmyocardial biopsies are not suggested as it rarely affects the patient's treatment. As clinically warranted, the physician may decide to explore other causes of cardiomyopathy with a drug screen, thyroid panel, various antibodies (e.g. for collagen vascular disease), and infectious serologies (e.g. HIV, Chagas, and Syphilis).

Once diagnosed, patients with peripartum cardiomyopathy are usually classified along the same guidelines as those with other forms of heart failure: the New York Heart Association classification system. Briefly, class I is defined by lack of symptoms; class II describes mild symptoms, seen only with extreme exertion; class III is symptoms with some exertion; and, class IV being with worst, defined as symptoms at rest. Our patient was in class III upon her presentation to our ED.

Prevalence rates of peripartum cardiomyopathy vary considerably internationally. In America, the incidence rate is quoted as anywhere between 1 per 1,300 to 15,000 live births, with the greatest majority proclaiming itself within the first month of delivery. This wide range is also seen internationally (1 per 6,000 live-births in Japan, as compared to 1 per 400 cases in Haiti) [5]. African American women tend to be more predisposed (up to 16 times more often in this demographic) [6].

Medical management is the same as treating other causes of systolic dysfunction. Preload and afterload reduction (hydralazine, nitrates), diuretics (furosemide, spironolactone), beta-blockade (metoprolol, carvedilol), and digoxin have all been shown to decrease hospitalization, morbidity and mortality. In the context of pregnancy or preeclampsia, diuretics should be used judiciously for concerns of volume status and uteroplacental hypoperfusion. ACE inhibition should be reserved until postpartum. Digoxin and dobutamine are available when ejection fraction is affected. Some studies have shown evidence for anticoagulation (heparin) if there's evidence of chamber enlargement, an affected ejection fraction (< 30%), or dysrhythmias because of the significant rate of venous and arterial thromboembolic disease. A few studies have found some benefit with pentoxifylline and bromocriptine, although discussion should be made with the patient and inpatient services before instituting these therapies. Importantly, consideration should be made with many of these medications as they have been shown to cross into the placenta and breast milk. Surgical interventions are reserved for the sickest population: those requiring intra-aortic balloon pump support, left ventricular assist devices, and heart transplantation [7].

The cardiology should be at the forefront of managing these patients; if still pregnant, high-risk obstetrics and neonatology should be considered as well. Unless the patient is acutely decompensating, vaginal deliveries are the preferred route because of decreased rates of pulmonary embolism and endometritis. Complications are common and oftentimes devastating. Progressive heart failure is the usual course if the patient is not aggressively managed. One study found thromboembolic disease to be present in as much as 50%. Cardiac dysrhythmias are also common. Misdiagnosis with preeclampsia can be problematic as treatment plans are usually conflicting (volume expansion versus restriction). Fetal complications include distress (from maternal hypoxia) and placental hypoperfusion (poor cardiac output, intravascular volume depletion, and hypotension, often as a consequence of treatment with afterload reduction and diuresis). Mothers are often advised against future pregnancies because of the higher rates of mortality and complication rates (e.g. heart failure, stillbirths, and need for therapeutic abortions), especially if their ejection fraction has not stabilized. Certain precautionary measures are warranted, including repeat echocardiography and/or dobutamine stress testing. Prognostically, a two-year mortality rate has been found to be 3-16% based on certain demographic markers. The most important marker for this determination is the extent of left ventricular function, with up to half significantly improving in the first six months after delivery.

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