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Peripartum Cardiomyopathy: Case Reports

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Abstract **Case Report**

Peripartum cardiomyopathy (PPCM) is a potentially fatal peripartum condition marked by left ventricular systolic dysfunction and heart failure in the absence of any established cardiac illness. Though its prevalence is increasing, there is still ambiguity about its prevalence, pathophysiology, and best management strategy. Our case study is about a patient who was hospitalized for shortness of breath that quickly worsened into asthma cardiale shortly after spontaneous birth. Because the origin of the symptoms was determined to be heart failure caused by peripartum cardiomyopathy, diuretics, inotropes, beta-blockers, and ACEIs were used to treat the symptoms. Mechanical cardiac assistance was unnecessary. Within three weeks, the symptoms of heart failure eased, and the left ventricular ejection fraction returned to its original level.

Keywords: Peripartum cardiomyopathy, cardiac illness, pathophysiology, heart failure.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an uncommon kind of heart failure that affects women in their late pregnancy or puerperium. The total prevalence of PPCM is between one in 1300 and one in 15,000 pregnancies [1]. However, the prevalence varies across the globe and is greater in underdeveloped countries [2]. When a patient has significant myocardial dysfunction, PPCM is generally diagnosed; however, less severe types of PPCM frequently go unnoticed [3]. The European Society of Cardiology (ESC) Working Group in 2010 described PPCM as an idiopathic cardiomyopathy having the following features:

1. The onset of heart failure (HF) at the end of pregnancy or within five months of birth.
2. The absence of an identified cause of heart failure.
3. Systolic dysfunction of the left ventricle (LV) with an LV ejection fraction (LVEF) less than 45%.

Despite several attempts to determine the actual cause of PPCM, the reason remains unexplained. Viral, autoimmune, nutritional deficiencies and idiopathic factors might all have a role1. The prevalence of PPCM in families implies a role for genetic predisposition [1-4]. PPCM pathophysiology has also been linked to altered prolactin processing and increased soluble Fms-like tyrosine kinase 1 (Flt 1). Increased oxidative stress during pregnancy causes cathepsin D to cleave prolactin into an aberrant 16 kDa protein. This protein is harmful to the heart and blood vessels. The placenta secretes soluble Flt 1, which suppresses vascular endothelial growth factor signaling, resulting in angiogenic imbalance and endothelial dysfunction [5].

Approximately 6% of PPCM patients have thromboembolic problems such as deep vein thrombosis, pulmonary thromboembolism, stroke, acute limb ischemia, and so on. PPCM has a significant morbidity and fatality rate, especially when accompanied with cardioembolic phenomena. Fortunately, despite the significant morbidity, mortality, and recurrence risk in subsequent pregnancies, many patients with peripartum cardiomyopathy recover within three to six months [6]. We present an unusual instance of peripartum cardiomyopathy in a young female.

CASE REPORT

A woman named Mrs Ruma age 29 years, at 8th POD withour having any history of heart disease presented to our urgent-care clinic five days after giving

birth, reporting breathlessness with high BP. She went to the clinic for evaluation. She was found to be BP was 160/110mmHG, oxygen saturation 78 and was sent to the Emergency Department (ED) for further treatment.

Her medical history included obesity, but the patient was in relatively good health until approximately her last month of pregnancy, when she developed gestational hypertension (without other significant pre-eclampsia signs and symptoms), dependent peripheral edema, as well as some symptoms of an upper respiratory infection. She was given labetalol, 200 mg orally, twice daily, for blood-pressure management.

During examination in the ED, the patient was noted to be afebrile and had a blood pressure of 160/11mm Hg, an oxygen saturation of 75% while receiving oxygen through a 2-L nasal cannula. Her lungs were clear to auscultation and her heart rate was regular, with an S3 gallop. Her extremities were nonedematous, and she had no calf tenderness. Urinalysis results were negative for any proteins. Plasma levels of D-dimer and circulating levels of B-type natriuretic peptide (BNP) were 1981 pg/mL and 864 pg/mL, respectively. An electrocardiogram showed a normal sinus rhythm. Chest radiographs showed cardiomegaly with increased vascular congestion bilaterally. A computed tomography (CT) chest scan to evaluate for possible pulmonary emboli showed evidence of pleural effusion and cardiomegaly but no emboli. In addition, her EF was 35%.

The patient was subsequently admitted to the hospital for new-onset PPCM and was given furosemide intravenously for diuresis. A transthoracic echocardiogram done at admission showed an LV ejection fraction of 35% to 40%, with trace aortic and mitral regurgitation.

Follow-up examination at six months showed a stable cardiomyopathy and well-controlled hypertension, and a repeat echocardiogram at the same point showed an improved ejection fraction.

DISCUSSION

During a normal pregnancy, around 60% to 70% of women will feel dyspnea [6]. Although PPCM risk factors have generally been associated with older women and black women, current trends reveal that there is a growing incidence (24%-37%) in young primigravid and white patients [7-9]. The two examples described here support this tendency; both women are young primigravidas and Caucasian. Because dyspnea is prevalent in normal pregnancy and even in the early postpartum period, PPCM is frequently ignored, especially if the patient group does not meet the conventional epidemiology [10].

Etiology

A putative link between pregnancy and dilated cardiomyopathy was identified as early as the 1870s10 and was designated as a separate clinical entity in the 1930s [11]. However, the etiology of PPCM remains uncertain. Most believe it is due to the cardiovascular stress of pregnancy (increased fluid load); some believe it is due to myocarditis. On endomyocardial biopsy, Felker *et al.*, [12] discovered that 26 of 51 women with PPCM exhibited histologic signs of myocarditis. Other researchers believe that PPCM is an inflammatory response during pregnancy, indicating an increase in tumor necrosis factor-alpha and interleukin-6 levels [13, 14].

CONCLUSION

The cases provided here highlight the wide range of clinical manifestations of PPCM. On chest radiographs, Case 1 shows a classic PPCM presentation, including gestational hypertension, S3 gallop, a high BNP level, cardiomegaly, and pulmonary congestion. As a result, it is critical that clinicians are aware with PPCM and consider it when evaluating dyspneic patients in order to speed medical treatment for a potentially fatal ailment and heart tissue. There is also contradictory information about the role of dietary deficiencies, notably selenium insufficiency, in the development of PPCM.

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