Pregnancy and Peripartum Cardiomyopathy: Case Report

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Abstract

Peripartum cardiomyopathy is a rare but potentially life threating form of dilated cardiomyopathy of unknown etiology. Early diagnosis is important to decrease morbidity and mortality but delays in diagnosis are commonly observed because symptoms can be confused with physiological changes associated with advanced pregnancy. We herein report a case emerging in the postpartum period accompanied by preeclampsia and discuss the important issues in diagnosis and treatment.

Keywords: Cardiomyopathy, pregnancy, postpartum period

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Introduction

Peripartum Cardiomyopathy (PPCM) is defined as the development of cardiac failure in the last month of pregnancy or within the first five months following delivery in the absence of predetermined cardiac disease and absence of demonstrable aetiology. Demonstration of left ventricular dysfunction (ejection fraction<45%, or fractional shortening<30%, or both) in echocardiography is also essential for the diagnosis of the disease [1]. The disease is seen in 1/1,300 to 1/15,000 pregnancies and characterized by congestive heart failure with unknown etiology. Disease-related mortality rate is 0.5-1.36% [2,3].

Risk factors associated with PPCM are advanced maternal age, multifetal pregnancy, multiparity, family history, ethnic origin (African race), smoking, diabetes, high blood pressure, preeclampsia, nutritional disorders, and prolonged beta-blocker usage [4]. Thus, symptoms should be carefully evaluated in patients that are in the risk group. Treatment is not different from acute and chronic heart failure treatment.

We herein report a case of PPCM who had more than two risk factors and whose echocardiographic evaluation was normal before delivery.

Case Presentation

A 36 years old G5P4 woman, who is 37 weeks and 4 days pregnant was admitted to the emergency room with the complaints of shortness of breath and fatigue lasting for 15 days. At admittance, her body temperature was 36.6 ^{0}C, blood pressure was 100/60 mm/Hg and heart rate was 91 beats per minute. Her past medical history was unremarkable. Vaginal examination and obstetric ultrasound examination were within normal limits. Hb was 7.8 g/dl, Htc was %23.4, white blood count was 8200, platelet count was 206.000 in complete blood count; biochemical parameters and urine analysis were within normal limits. The case was hospitalized for further evaluation and treatment of anemia. Cardiologic consultation was requested for dyspnea and tachycardia. Echocardiographic examination revealed no abnormality and complaints were attributed to anemia. 2 unites of erythrocyte suspension were transfused for this purpose and Hb reached to 9.1 g/dl and Htc reached 27.1% after the transfusion. On the fifth day of hospitalization, spontaneous contractions started and she had a healthy 2900 gr weighting male baby by uncomplicated vaginal delivery . Apgar score of

the newborn was within normal limits. At the 24th hour of delivery, arterial blood pressure was measured as 180/120 mmHg and proteinuria was measured as (++). The diagnosis of postpartum preeclampsia was done and MgSO4 infusion was started. General condition of the patient was good, vital signs were stable and she had no complaint in the follow-up. She was discharged at 4th day postpartum. She was re-admitted two days after the discharge with complaints of sudden-onset tachycardia, shortness of breath aggravated by lying down and coughing. The case was conscious and excessively agitated. Blood pressure was 90/60 mmHg, body temperature was 37.5C, heart rate was 110 bpm, respiratory rate was 26/min, S3 and diffuse crepitant rales were detected with auscultation. Diffuse infiltrative pattern and increased cardiothoracic index were detected on pulmonary radiography. Echocardiographic examination revealed an increase in left ventricular space diameter and ejection fraction was measured as 28%. She was admitted to coronary intensive care unit with the diagnosis of PPCM and first degree mitral insufficiency. Routine supportive treatment for heart failure was initiated and breast feeding was stopped because of the potential adverse effects of the drugs to the neonate. General status of the patient improved at the 9th day of admittance and she was discharged from the hospital with recommendation of monthly control. No symptom was present at the first visit and echocardiographic and PA pulmonary radiographic examinations revealed no abnormality.

Discussion

Hemodynamic changes observed during pregnancy are physiological changes that target to supply sufficient blood flow to growing fetus and increased maternal metabolism. Cardiovascular changes start early in the first trimester, continues progressively until the end of the second trimester and keep a steady state until the end of the pregnancy. Right after delivery preload increases secondary to volume increase caused by return of the maternal blood from uteroplacental bed back to circulation [5]. Findings of cardiac failure emerge in last month of pregnancy or first few months after the delivery in case of peripartum cardiomyopathy and as seen in our case, pre-detected cardiac failure or cardiac disease that may cause this situation is not present.

The etiology of PPCM is not certain however, inflammation, infection and autoimmunity are blamed [6]. Prolactin hormone, increased oxidative stress, proteolytic and angiostatic factor

and division of apoptotic particles to primers (cathepsin D) are thought to cause PPCM [7]. The diagnosis is established by exclusion of other causes of cardiac failure. It should be differentiated from dilated and hypertrophic CMP. Signs of dilated CMP are similar to those of PPCM and left ventricular dilatation is also observed in dilated CMP. Time of emergence of disease is important in differential diagnosis. Dilated CMP presents usually at first or second trimester in which the hemodynamic load starts to increase. Hypertrophic CMP is the most common genetic cardiac disease [8]. Initial diagnosis is usually made during pregnancy due to increased hemodynamic load but these cases mostly tolerate pregnancy well.

Therapeutic options need to consider both the health of the mother and the fetus in pregnancy but after delivery the treatment is similar to that of other types of heart failure including ACE inhibitors, beta-blockers, spironolactone, diuretics, digoxin, vasodilators and inotropes [9]. Targeted therapies including bromocriptine, intravenous immunoglobulin, pentoxifylline need further evaluation before widely used in clinics. Severe heart failure, cardiogenic shock, thromboembolic complications, arrhythmias and death may be observed in these patients. Prognosis is best in early diagnosis and treatment. Delay in diagnosis, severe LV dysfunction (LVEF<25%), multiparity, advanced maternal age, African American race increases mortality [7]. Our patient was multiparous and had advanced maternal age but her LVEF was %28 at the time of diagnosis and she recovered completely. Rarely, cardiac transplantation may be the only single choice of treatment in advanced cases refractory to medical treatment [10]. Recovery usually occurs in 3 to 6 months but might occur as late as 48 months postpartum [11]. In our patient complete recovery of the left ventricle happened in one month which is earlier than indicated in the literature.

Early delivery is not required if fetus and the mother are stable [12]. Decisions on the type of delivery and on the timing should be made with a multidisciplinary team. Vaginal delivery is preferred in hemodynamically stable cases if no indications for caesarean section are present. Hemodynamic status must be controlled carefully during labour and operative methods such as vacuum should be performed to shorten the 2^{nd} phase of delivery. Regional anesthesia reduces preload and afterload and minimizes the fluctuations in cardiac output associated with labour. Emergent delivery should be considered in case of advanced cardiac failure since delivery of the fetus reduces the hemodynamic stress on the heart. Mother should be observed under intensive care unit circumstances and pulmonary edema which may occur due to uterine

auto-transfusion should be considered [13]. Our patient delivered vaginally without any complication. Second phase of the labour was around 10 minutes in our patient.

PPCM have a recurrence rate of 30-50% [14]. Besides the adequate medical treatment left ventricular dysfunction becomes permanent in 50% of the cases [15]. Since the recurrence in subsequent pregnancy is high even after complete recovery, current cardiomyopathy guidelines advise against future pregnancies [4]. Diagnosis of PPCM must always be remembered in mind in patients experiencing shortness of breath and fatigue in the peripartum period. Echocardiography is an important diagnostic tool. Multidisciplinary approach is important to decrease morbidity and mortality.

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