CEOG Clinical and Experimental Obstetrics & Gynecology

Peripartum cardiomyopathy: a case of patient with triplet pregnancy

B. Kastratović Kotlica[1], A. Ćetković[2], S. Plešinac[1], Đ. Macut[3], M. Ašanin[4]

[1] Clinic of Gynecology and Obstetrics, Clinical Center of Serbia, School of Medicine, University of Belgrade
[2] Clinic of Gynecology and Obstetrics, Clinical Center of Serbia, Belgrade; [3] Clinic for Endocrinology, Diabetes and Metabolic Disorders, Clinical Center of Serbia, School of Medicine, University of Belgrade, Belgrade
[4] Cardiology Clinic, Clinical Center of Serbia, School of Medicine, University of Belgrade, Belgrade (Serbia)

Summary

Peripartum cardiomyopathy (PPCM) is a rare but potentially devastating complication of pregnancy associated with heart failure due to left ventricular systolic dysfunction occurring within the last month of pregnancy and five month postpartum with no obvious other cause of heart failure and no pre-existing heart disease. In the present case report the authors present a woman who developed PPCM on the day after she delivered by cesarean section in 35th weeks of gestation of triplet pregnancy conceived after ovarian stimulation and insemination. A treatment with diuretics, ACE inhibitors, antiarrhythmics, low weight heparin, antibiotics and bromocriptine was applied and resulted in complete recovery. In conclusion, timely detection and initiation of treatment are important factors for complete recovery of patients with PPCM.

Key words: Heart failure; Peripartum cardiomyopathy; Infertility treatment.

Introduction

Peripartum cardiomyopathy (PPCM) is a rare but potentially devastating complication of pregnancy associated with heart failure due to left ventricular systolic dysfunction occurring within the last month of pregnancy and five month postpartum with no obvious other cause of heart failure and no pre-existing heart disease [1]. Classical symptoms of this condition, such as worsening dyspnea or ankle swelling, are often attributed to the hemodynamic stress of pregnancy, which frequently lead to delay in the diagnosis of PPCM [1]. There are several predictors of unfavorable outcome that include increasing maternal age, multiple pregnancies, history of hypertension, late onset of symptoms following delivery, non-Caucasian origin, and delayed diagnosis [2].

Despite significant improvements in the management of heart failure, the morbidity and mortality related to this condition remain significant, with mortality rates of between 9% and 32%, and cardiac transplantation required in up to 10% of survivors [3, 4].

Case Report

A 33-year-old woman with polycystic ovarian syndrome and infertility was successfully treated with clomiphene citrate. She had undergone intrauterine insemination "swim up" and triple pregnancy was conceived. Because of uterine contractions, she received intravenous tocolytic therapy for two days during second and progesterone vaginally 200 mg twice daily, during third trimester of pregnancy. Her blood pressure was normal during each hospitalization and she had no signs or history of previous cardiovascular disease. The patient delivered by cesarean section in 35th weeks of gestation with three healthy newborns with following measures: male 1,800 g/Apgar score 5/7, male 2,100 g/Apgar score 5/6, and female 1,730 g/Apgar score 4/5. One day after delivery patient developed symptoms of cardiovascular and respiratory dysfunction with neurological symptoms presenting in the form of five convulsive attacks. Initial examination revealed a blood pressure of 130/80 mmHg, a regular pulse of 130 bpm, an elevated jugular venous pressure (JVP), and inspiratory crepitations in the left lung.

Initial blood results revealed slightly elevation of creatine kinase MB fraction 52 U/l (normal range 0.1-40.0) and markedly elevated AST 142 U/l (normal range <37), lactate dehydrogenase 1,288 U/l (normal range 226-460), brain natriuretic peptide (BNP) 1,835 pg/ml (normal range <18.4 pg/ml), while blood count, urea, and electrolytes were all within normal range. Blood gas analysis was: pH 7.49, pCO2 3.7, pO2 19.3, oxygen saturation 78%. Chest X-ray showed cardiomegaly with left basal shadowing and left pleural effusion. ECG showed sinus tachycardia of 130 bpm without significant change in S-T segment. Echocardiography revealed a dilated left ventricular internal diameter of 5.5 cm with global hypokinesis, impaired left ventricular (LV) systolic function, and ejection fraction (EF) of 25-30%. CT scan of endocranium showed normal findings without ischemic or vascular lesions. The patient was transferred to the intensive care unit, and because she developed respiratory insufficiency, she was immediately intubated and placed on mechanic ventilation, with dobutamine and furosemid started to treat heart failure, anti-edematous therapy (manitol), anti-aggregation therapy (low weight heparin), anti-

Revised manuscript accepted for publication August 5, 2014

convulsive (MgSO4), ACE inhibitors, xylocaine because of the sporadic ventricular extra systolic arrhythmias, digitalis, antibiotics (teicoplanin, meropenem, vancomycin), and bromocriptine.

Concurrently LV function improved, heart failure and respiratory insufficiency symptoms and signs decreased. On the third day after delivery she was extubated, on the repeated chest X-ray done on the 10th day after delivery there was no plural effusion. Echocardiography was done on the 18th day after delivery revealing normal dimension of the left ventricle, EF was 64%, no pericardial effusion was detected, BNP level was reduced to 411 pg/ml after three weeks of delivery, and to 156 pg/ml after two months. Patient was discharged from the hospital on the 22nd day after delivery.

Discussion

PPCM is a pregnancy associated myocardial disease that is heterogeneous and seems to have important phenotypic variations in different geographical regions, so it is difficult to formulate uniform recommendations for the treatment of this condition [5]. The etiology of PPCM is still unknown, and many potential causes have been proposed but not proven, such as viral myocarditis, abnormal immune response to pregnancy, inadequate response to increased hemodynamic burden of pregnancy, hormonal abnormalities, inflammation, and apoptosis [6]. Prolonged tocolytic therapy also could be a possible cause of PPCM, but it is not determined whether it has direct influence or if it just unmasks a preexisting subclinical disease [7]. Recent investigations in large cohorts of familial dilated cardiomyopathy reveal that genetic etiologies may be identified in a substantial fraction of women with PPCM [8].

The treatment for PPCM is similar to that for other forms of heart failure and consists of angiotensin-converting enzyme inhibitors that are used to reduce afterload by vasodilatation if PPCM occurs after pregnancy, β-blockers used to treat tachycardia and arrhythmias, diuretics that reduce preload, and because of a high incidence of thromboembolic complications in these patients, the use of heparin is also necessary.

Several recent reports have noted that bromocriptine and prolactin antagonist carbergoline could preserve LV function in PPCM [9]. The treatment of PPCM with bromocriptine was based on the study results that increased oxidative stress could lead to an increased expression and activity of the cathepsin-D, which is responsible for cleaving the 32-kDa form of prolactin to a smaller fragment which possesses antiangiogenic, proinflammatory, and vaso- constrictory properties and could cause dilated cardiomyopathy [1].

The present patient with acute PPCM, who in addition to standard therapy for heart failure, received also bromocriptine and had a successful recovery despite initially dramatic clinical presentation. Regarding the risk factors for development of PPCM, the present patient had triplet pregnancy and once received intravenous tocolytic therapy during second trimester of pregnancy. Positive response to applied therapy in this case could be attributed to LV diastolic dimension and systolic function (left ventricle ejection fraction -LVEF). A multivariate analysis by Goland *et al.* in 187 patient with PPCM [10] found that LVEF >30% and LV end-diastolic dimension < 55mm were significantly related to LV recovery, suggesting a relationship between the degree of initial myocardial insult and recovery.

In conclusion, timely detection and initiation of treatment are important factors for complete recovery of patients with PPCM. The identification of predictors of LV recovery may help stratify patients who would benefit from more invasive and expensive forms of therapies. Even with full recovery, some additional risk of relapse remains present in patients with PPCM.

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Address reprint requests to:
A. ĆETKOVIĆ, M.D.
Clinic of Gynecology and Obstetrics
Clinical Center of Serbia
Milentija Papovića, 33
Belgrade (Serbia)
e-mail: aleksa_cetkovic@vektor.net