ACTA MEDICA LITUANICA. 2012. Vol. 19. No. 3. P. 224–227

Peripartum cardiomyopathy: case report

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Peripartum cardiomyopathy is an unusual and uncommon form of dilated cardiomyopathy that is often fatal to young women, the cause of which is unknown. Diagnostics is difficult and requires vigilance. The treatment does not differ from other forms of heart failure. Fetal outcome, however, is quite good. Maternal outcomes depend on 2–6 months recovery of the left ventricular function. We describe a previously asymptomatic patient who presented with pulmonary edema one day after caesarean section. In this case the solution was favorable to the patient. Complete recovery of the left ventricular function happened earlier than indicated in literature.

Key words: peripartum cardiomyopathy, breastfeeding, prognosis

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy, characterized by the heart failure, left ventricular systolic dysfunction towards the end of pregnancy or WITHIN 5 months of delivery, when no other cause of the heart failure is found (1). There are no data about the incidence of PPCM in Lithuania. In the USA, there is one case in 2 300 women giving birth and about 1 300 new cases a year (2). The incidence in South Africa is higher because of the Africo-American women.

The exact pathogenetic mechanism is not fully understood. Several pathogenetic factors are as follows: inflammatory, infectious, genetic, autoimmune, oxidative stress. The diagnosis is confirmed on the basis of diagnostic criteria (3): a) development of the heart failure during the last month of pregnancy or within 5 months of delivery; b) absence of an identifiable cause for the heart failure; c) absence of recognizable heart disease prior to the last month of pregnancy; d) left ventricular dysfunction determined during echocardiography with ejection fraction <45% (4).

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PPCM treatment is not different from acute and chronic heart failure treatment. Discontinuation of therapy is recommended only in case of recovery of the left ventricular function, gradually monitoring cardiac function and repeating the 2D cardiac ultrasound imaging (6). Recovery of the left ventricular function usually occurs within 2–6 months after diagnosis (7). The mortality rate ranges from 1% to 19% (8, 9). Repeated risk of PPCM during subsequent pregnancy is 30–50%, if the left ventricular dysfunction remains.

CASE REPORT

A 38-year-old woman was admitted to the Department of Obstetrics and Gynecology for premature delivery and rupture of fetus membranes. It was her third pregnancy of 34 weeks. The patient had never had any heart disease. Her pregnancy was going well. She did not use any medication. The first two pregnancies and births were smooth. No bad habits. After arrival she complained about amniotic fluid flow and occasional, mild pain in the lower abdomen. Analysis performed in the Obstetrics and Gynecology Department showed increase in inflammatory markers: 19.37 × 10^9/l leukocytosis, increased C-reactive protein (CRP) – 80.14 mg/l. However, there were no signs of clinical infection. Empirical antibacterial therapy was started (with cefuroxime). Gynecological examination revealed ruptured amniotic bubble, flow of clear amniotic fluid. Infection of the uterus was suspected therefore it was decided to perform the caesarean section. Lung maturation of premature fetus was started using glucocorticoids, tocolytic therapy with calcium (Ca) channel blockers was applied during lung maturation of the fetus. Clinical diagnosis: Graviditas III (III) 34 hebdomas. Partus praematurus. Defluxus liqori amnii spontaneus praeterminalis. Cholestasis gravidarum. One day later, the planned cesarean section was performed under endotracheal anesthesia after maturation of fetal lungs. After extubation, a short episode of hypoxaemia in the patient was observed (SpO_2 – 97%–88%–94%), which was corrected with O_2 therapy mask over 6 l/min. Further treatment was applied in the Intensive Care Unit of the Obstetrics and Gynecology Department. The patient received infusion of 2 500 ml crystalloid, and duresis was only 500 ml over four hours.

About 11 hours after surgery she started to complain of progressive shortness of breath at rest, chest discomfort, cough. During examination the overall condition was difficult: forced semi-sitting position. The patient was breathing spontaneously with supplied O_2 8 l/min through a face mask. Tachypnea was 28 rates / minute, SpO_2 87%. Vesicular breathing in lungs, on both sides, significant bilateral basal crackles. Sinus tachycardia was124 beats / minute, arterial blood pressure 114/82 mmHg. The cardiac auscultation showed normal first and second heart sounds. No significant cardiac murmurs were detected. She was afebrile. Metabolic acidosis determined during arterial blood gas analysis: pH – 7.30; PCO_2 – 33.1 mmHg; PO_2 – 53.8 mm Hg; BE – (–9) mmol/l; HCO_3 – 17.1 mmol/l. D-dimer was 7.8 mg/l, hypokalemia K 3.2 mmol/l, renal functions, liver and clotting parameters were normal. Differentiation between pulmonary embolism and edema. Chest X-ray showed a massive pulmonary interstitial edema, heart size and shape unchanged. Initial echocardiography showed poor left ventricular ejection fraction (LVEF – 25%), middle and top parts were hypokinetic. Diastolic function was of a disordered relaxation type. Heart cavities were not dilated. Valves were in good condition. There was no pulmonary hypertension. Vena cave superior was completely collapsed. The patient was hospitalized for further treatment to the Cardiac Intensive Care Unit because of the suddenly developed acute heart failure with pulmonary edema. The electrocardiogram showed nonspecific T-wave inversion in thoracic derivations. Elevated troponin I was 5.07–0.74 μg/l and NTproBNP was 6 364 pg/l. Additional clinical diagnosis: Cardiomyophatia peripartum. Insufficientia cordis acuta. Oedema pulmonum.

Pulmonary edema and heart failure treatment was applied with O_2 therapy 6 l/min through nasal cannula, intravenic nitrates, diuretics, preoral B-blockers (BAB) and angiotensin converting enzyme inhibitors (ACE), low molecular weight anticoagulants (LMWH). The patient was allowed to breastfeed. She was treated in the Cardiologic Intensive Care Unit for 5 days. During the treatment, the left ventricular function significantly improved LVEF – 25% → 38%. Pulmonary edema regressed. After two weeks the patient was discharged from hospital for out-patient treatment of the heart fail-

ure. A month later, repeated 2D echocardiography showed normal-sized cardiac cavities, fully recovered function of LVEF – 60%.

DISCUSSIONS

In this case, peripartum cardiomyopathy was diagnosed upon exclusion of other reasons for the heart failure, according to the diagnostic criteria of peripartum cardiomyopathy (3). There were difficulties regarding diagnosis because PPCM belongs to the group of rare diseases. In this case, PPCM was clinically observed on the first day after giving birth. According to literature, 93% of PPCM occur after delivery, of which 75% occur in the first month after delivery, and only 7% during the last trimester of pregnancy (10).

The risk of developing this disease is caused by the mother's age, number of pregnancies, multiple pregnancy, preeclamsia, gestational hypertension, oral tocolytic therapy with beta adrenergic agonists. From patient's history it is known that she has never had heart and vascular diseases, but there was a number of risk factors, i. e. older age and a third pregnancy.

Symptoms and signs that can mimic heart failure are often attributed to normal pregnancy. Therefore, the disease is usually suspected only after pulmonary edema, arrhythmic, thromboembolism complications and, in rare cases, by accident performing heart ultrasound. In this case, pulmonary edema, which could be provoked by a general anesthesia and liquids overload, allowed suspecting PPCM. It was diagnosed on the basis of the above mentioned diagnostic criteria (3), of which echocardiography is the most important to identify PPCM. Other tests such as the electrocardiogram and troponin I changes are considered to be nonspecific in this disease (6).

However, according to literature, such tests as high troponin I, plasma BNP levels, LVEF <30%, LV diastolic dimension <5.5 cm at the time of diagnosis, are factors of poor prognosis (6). Although in our case the patient had a number of such factors, the outcome was better than expected.

The patient was treated according to the treatment guidelines of heart failure established by the European Society of Cardiologists (5). Attention was paid to the safety of medication to breastfeeding mothers (11). Because of high incidence of thromboembolic complications typical of this disease, anticoagulant treatment with LMWH was prescribed for as long as the left ventricular function improved (LVEF >35%) (12). Thromboembolic complications were not observed.

Breastfeeding in our clinic has caused a lot of discussions and opinions. The patient expressed a strong desire to breastfeed, so it was decided to allow moderate lactation. In this case it was a correct solution. Safirstein et al. showed that the recovery of the left ventricular function was statistically significantly better for breastfeeding mothers (13). According to literature, in about 50% of all cases, the left ventricular function is restored within 2–6 months after diagnosis (7). In our case, the correct choice of treatment is proved by quick patient's recovery, total recovery of LV function within a month.

CONCLUSIONS

PPCM is a rare disease, the cause of which is unknown, typical of women of childbearing age. Diagnostics is difficult and requires vigilance. The main objective of treatment is to reduce the symptoms of congestive heart failure.

Received 20 July 2012
Accepted 1 August 2012

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PERIGIMDYVINĖ KARDIOMIOPATIJA. VIENOS PACIENTĖS ATVEJIS

Santrauka

Perigimdyvinė kardiomiopatija yra reta nežinomos etiologijos dilatacinės kardiomiopatijos forma, pasireiškianti vaisingo amžiaus moterims. Jos diagnostika sudėtinga, tad būtinas budrumas. Gydymas panašus į kitų širdies nepakankamumo formų gydymą. Vaisiaus išeitis dažniausiai gera. Gimdyvės išeitis priklauso nuo daugelio veiksnių, visiškas pasveikimas galimas per 2–6 mėnesius. Mes aprašėme pacientės atvejį, kai perigimdyvinė kardiomiopatija pasireiškė plaučių edema kitą parą po cezario pjūvio operacijos. Šiuo atveju išeitis buvo gera, kairiojo skilvelio funkcija atsistatė anksčiau, negu nurodoma literatūroje.

Raktažodžiai: perigimdyvinė kardiomiopatija, maitinimas kūtimi, prognozė