

Acute heart failure in a 35 weeks pregnancy. A Case Report

Abstract

We present the case of a 18-year-old patient, primigravida primiparous at 35 weeks pregnancy who presented at the hospital with acute heart failure without having any history of cardiovascular disease. Therefore, it becomes quite difficult to establish the etiology of heart failure in this case. The echocardiography proved particularly useful in excluding a congenital heart disease and valvular disease and showing severe systolic left heart dysfunction and cardiac dilatation. Taking into account the absence of a recognized cardiac disease before pregnancy and the symptoms' debut late during pregnancy, the peripartum cardiomyopathy diagnosis was established. The patient was addressed to the intensive coronary care unit receiving adequate treatment and on the third day after hospital admission she gave birth by cesarean section to a live female fetus, weighing 1710 g and receiving an Apgar score of 8/9. Postpartum, clinical and biological evolution was favorable, but the left ventricle systolic dysfunction persisted. In this particular case, the left ventricular ejection fraction did not return to its normal value 6 months after birth which implies a high mortality risk when a future pregnancy occurs.

Keywords: heart failure, systolic dysfunction, peripartum cardiomyopathy, mortality

Introduction

Hemodynamic changes that occur during pregnancy (e.g. blood volume expansion, enhancement of cardiac output, decreased in systemic vascular resistance) trigger symptoms which resemble the ones that also appear in cases of heart failure. The latter should be taken into consideration especially when symptoms that are unusual during normal pregnancy appear such as: chest pain, nocturnal paroxysmal dyspnea, coughing, pulmonary congestion signs, fourth heart sound, sinus tachycardia with an enhancement of heart rate over 20%, pleural effusion(1). Echocardiography is indicated during pregnancy whenever dyspnea or a new pathological heart murmur is diagnosed.

Heart failure during pregnancy can have multiple causes: valvular disease, congenital heart disease, acute myocarditis and cardiomyopathies, arterial coronary disease (i.e. rarely). Cardiomyopathies occurring during pregnancy can also have multiple etiologies, congenital or acquired like peripartum cardiomyopathy, toxic cardiomyopathy, hypertrophic cardiomyopathy, storage diseases etc⁽¹⁾.

Peripartum cardiomyopathy is an idiopathic cardiomyopathy associating heart failure secondary to systolic dysfunction which appears in late pregnancy or first months after giving birth. It is considered an exclusion diagnosis when no other cause of heart failure is identified. The left ventricle may not be dilated, but its ejection fraction is always reduced under 45%⁽²⁾. Briefly, the diagnosis criteria were:

1. developing heart failure secondary to systolic dysfunction late in pregnancy or in the first 5 months after

giving birth⁽³⁾; 2. absence of a known cardiac condition before last month of pregnancy and 3. echocardiography criteria of left ventricular ejection fraction (LVEF) <45%, with or without heart dilatation.

Peripartum cardiomyopathy should be suspected whenever heart parameters do not come back to normal before pregnancy values. Echocardiography is the preferred method to evaluate LVEF. Often, patients may develop acute heart failure. Complex ventricular arrhythmia and sudden cardiac death can also occur, being an important cause of maternal mortality or progressive heart failure. Most of the patients with peripartum cardiomyopathy are diagnosed in the peri- and postpartum periods. The ones being diagnosed during pregnancy need a combined obstetrical and cardiac care plan.

The adverse effects on the fetus should also be taken into consideration when prescribing medicines. Urgent delivery, without respect to gestational age, should be performed in all cases of advanced heart failure with hemodynamic instability⁽⁴⁾. Soon after the fetus is born and the patient becomes hemodynamically stable, the standard heart failure therapy should be initiated.

Vaginal delivery is always preferred if the patient is hemodynamically stable and there are no indications for cesarean section. Close hemodynamic observation is required.

Prognosis on future pregnancies

In up to 50% of the cases, in spite of adequate treatment, a left ventricular function deterioration has been described⁽⁵⁾. In a future pregnancy, the risk of recurrence

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Figure 1. Apical four chamber view with LVEF measurement Simpson biplan method. LVEF 16% showing severe left ventricular systolic dysfunction



Figure 2. Apical four chamber view showing tricuspid regurgitation and measurement of right ventricle-right atrium pressure gradient to obtain systolic pulmonary artery pressure value. The patient presents severe pulmonary artery hypertension



Figure 3. LVEF measurement on discharge day showing a persistent left ventricular systolic dysfunction (LVEF 41%)

for peripartum cardiomyopathy is 30%-50%^(3,5). When LVEF has not return to its normal values, a future pregnancy should be postponed and although LVEF becomes normal, patient counseling remains important given the high recurrence risk.

Case Report

We report the case of a 18-year-old, primigravida primiparous 35 weeks pregnant patient who was hospitalized in the Obstetrics and Gynecology Department of Elias University Hospital (being transferred from Ploiesti Obstetrics and Gynecology Hospital) for progressive dyspnea, dry cough and bilateral lower extremities edema that occurred during the past 2 weeks. From the personal patient history we mention an episode of acute bacterial pneumonia early in pregnancy for which the patient was hospitalized and received antibiotic treatment.

Discussion

Clinical examination showed minimal effort dyspnea with signs of systemic congestion (lower extremities edema and jugular venous stasis) and on auscultation examination rhythmic cardiac sounds were noted with tachycardia and protodiastolic gallop sound, pulmonary murmur bilaterally without pathological sounds. Vaginal examination revealed no significant cervical alterations and no amniotic fluid discharge. The non stress test was reactive.

The clinical data pointed out towards heart failure. Nonetheless, pulmonary thrombembolism (taking into account dyspnea, lower extremities edema, normal pulmonary auscultation) or a lung condition (taking into account dyspnea and coughing, yet without fever and abnormal pulmonary sounds) should be excluded. Consequently, we decided this was the case of an acute heart failure, class III-IV, apparently unexplained occurring in a young woman without any cardiac disease history.

From biological point of view, we report: high white blood cells count (i.e. 12360/ μ L) with neutrophilia, ferripriva anaemia (i.e. Hb 7.6 g/dL, iron load 20 μ g/dL), increased transaminases (AST 275U/L, ALT 193 U/L), increased bilirubin (i.e. total Brb 2.1 mg/dL, direct Brb 1.4 mg/dL), mild hyponatremia, azotemia (creatinine 1.26 mg/dL, urea 101 mg/dL), hypoproteinemia (i.e. total proteins 3.8g/dL), urine sediment test (hematuria and proteinuria- 30 mg/dL), negative urine culture, negative endocervical culture, negative HbSag and negative lues tests.

The electrocardiogram showed sinus tachycardia, unspecified ventricular repolarization modifications and left ventricular hypertrophy signs.

Obstetrical ultrasound showed a singleton pregnancy with fetal intrauterine growth restriction, anterior placenta, AFI 13 cm, HR 148 bpm, RI- MCA 0.72 and RI- OA 0.59.

Echocardiography was revealing for the diagnosis showing cardiac dilatation, severe left ventricular dysfunction (LVEF 16%, Figure 1 (normal value over 55%)),

severe hypokinesia, severe tricuspid regurgitation, severe pulmonary hypertension (systolic pressure in the pulmonary artery 73 mmHg, (Figure 2) and moderate aortic regurgitation.

The differential diagnosis of dilatative cardiomyopathy (DCM) is required in this case:

1. familial DCM- not the case for our patient
2. postmiocarditis DCM- the patient reported no viral infection
3. peripartum cardiomyopathy- taking into account the debut late in pregnancy, the absence of a cardiac condition before 32 weeks of gestation and the absence of a cardiac condition before pregnancy.

Therefore, the peripartum cardiomyopathy diagnosis was established and the patient was addressed to the intensive coronary unit receiving adequate treatment: nasal oxygen, diuretics, digitalis, low-dose beta-blockers, low molecular weight heparin prophylactically, antispastic therapy and corticosteroids for fetal lung maturation and parenteral iron.

Blood pressure, heart rate and oxygen saturation were constantly being monitored and cardiotocography was performed twice a day.

The patients clinical evolution was slowly favorable with decreased dyspnea and the disappearance of lower extremities edema. Biologically, we report: Hb enhancement to 8.1 g/dL, white blood cells count 9730/ μ L, mild hepatic cytolysis and cholestasis syndrome improvement, rising proteinuria and rising creatinine.

On the third day, cesarean birth is decided under general anaesthesia. The fetus was a female, weighing 1710 g with an Apgar score of 8/9.

Immediately postpartum, a both clinical and biologically improvement was noticed: hemodynamically and respiratory stability, absence of signs of pulmonary and

systemic congestion, hepatic cytolysis and cholestasis syndrome improvement, azotemia reduction and a persistent moderate anaemia.

Echocardiography evaluation 4 days after hospital admission showed a slightly improvement of the LVEF (26%, with a persistent severe systolic dysfunction), a reduction of the pulmonary artery pressure (PAPs 35 mmHg) and an improvement in both aortic and tricuspid regurgitation.

On discharge, the patient had a LVEF of 32% with persistent cardiac dilatation; therefore, diuretics, digitalis, betablockers, conversion enzyme inhibitors and iron were prescribed.

At 6 months postpartum, left ventricular function had not normalized (LVEF 41%- Figure 3). The patient reported no symptoms of heart failure.

The absence of LVEF returning to normal values 6 months postpartum is a sign of poor prognosis and of maternal mortality risk in case of a future pregnancy, reason for which the patient was counseled to defer a second pregnancy.

Conclusions

This is a case of peripartum cardiomyopathy in a young patient (with a possible risk factor for this condition being a lung infection 7 months prior to the diagnosis), with severe LVEF and who had a favorable clinical evolution without requiring cardiac tonic therapy. Still, it is one of the cases in which LVEF did not return to its normal values in 6 months postpartum, which represents a risk for maternal mortality in a future pregnancy. Multidisciplinary care (including cardiologists, obstetricians, neonatologists and anaesthesists) was an important aspect in the patients management. ■

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