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Case Report of Multidisciplinary Management of Peripartum Cardiomyopathy

Abstract

Peripartum cardiomyopathy (PPCM) is a form of heart failure that occurs during pregnancy or in the postpartum period, characterized by reduced left ventricular systolic function [1]. Peripartum cardiomyopathy is one of the leading causes of maternal mortality and morbidity worldwide.

Aim. To demonstrate, through a clinical case, the importance of early diagnosis, timely risk factor identification, and optimized strategies for treatment, delivery, and postpartum care.

Case Report. Patient B, a 36-year-old woman, was urgently admitted to the hospital on the sixth day postpartum due to progressive symptoms of acute heart failure. Examination confirmed peripartum cardiomyopathy, acute heart failure (Killip III, pulmonary edema), severe mitral regurgitation, left atrial dilation, moderate tricuspid regurgitation, moderate pulmonary hypertension, and massive bilateral pleural effusion. The left ventricular ejection fraction (LVEF) was 36 %, with NYHA Functional Class IV. Bilateral thoracentesis was performed, and acute heart failure therapy was administered in the intensive care unit for five days, leading to clinical improvement and improved laboratory and instrumental findings. The patient received heart failure therapy, including bromocriptine, and was managed by a multidisciplinary team of cardiologists, obstetricians, cardiac surgeons, and intensivists. At discharge, the patient's condition was stable. Echocardiography revealed reduced mitral regurgitation (from severe to moderate), decreased left atrial size, and an LVEF increase to 40 %. NT-proBNP was 533.2 pg/mL, with other laboratory parameters within normal limits. Long-term outcomes were assessed at 2, 6, and 14 months post-discharge. At 14-month follow-up, complete recovery of myocardial and mitral valve function was observed, with a stable clinical condition.

Conclusions. Peripartum cardiomyopathy remains a serious cause of maternal and perinatal morbidity and mortality. Timely diagnosis and management of this condition are possible only through close collaboration within a multidisciplinary team comprising obstetrician-gynecologists, cardiologists, cardiac surgeons, and anesthesiologist-intensivists. Coordinated actions of these specialists contribute to optimizing the management of pregnancy, delivery, and the postpartum period, as well as improving long-term outcomes for both mother and child.

***Keywords**: pregnancy, postpartum period, peripartum cardiomyopathy, heart failure, ejection fraction, bromocriptine, perinatal outcomes, multidisciplinary care.*

Introduction. Peripartum cardiomyopathy (PPCM) is a form of heart failure that occurs during pregnancy or in the postpartum period, characterized by reduced left ventricular systolic function [1]. Symptoms of normal pregnancy may mimic heart failure, potentially delaying diagnosis and treatment and leading to adverse maternal and perinatal outcomes. PPCM is a leading cause of maternal mortality worldwide, accounting for 60 % of cardiogenic shock cases during pregnancy and the postpartum period [1,2]. The incidence of PPCM varies by region, with a global estimate of 1 in 2000 live births, ranging from 1 in 300 in Haiti to 1 in 20,000 in Japan [3-7]. This variation is likely associated with socioeconomic status and ethnicity [8-10]. In Ukraine, a national multidisciplinary team reported an incidence of 1 in 252,000 [11], though this figure may be underestimated compared with global data, highlighting the need for improved detection [3-7]. Risk factors for PPCM include African descent, maternal age >30 years, multiparity (>3 deliveries), multiple gestation, essential or gestational hypertension, preeclampsia, prolonged tocolysis, metabolic disorders (e.g., obesity, prediabetes, diabetes), and low socioeconomic status [11,12]. At the preconception stage, evaluation should include a history of prior PPCM episodes due to the high recurrence risk. According to the modified World Health Organization (WHO) cardiovascular risk (CVR) scale, a history of PPCM stratifies patients into the highest risk classes (III or IV). Differential diagnosis of PPCM includes other forms of dilated cardiomyopathy (DCM; e.g., familial, Takotsubo), tachycardia-induced cardiomyopathy, DCM associated with systemic diseases, acute coronary syndrome, pulmonary embolism, and myocarditis. Timely diagnosis and differential diagnosis of PPCM enable a significant reduction in maternal and perinatal mortality and patient disability.

Aim. To demonstrate, through a clinical case, the importance of early diagnosis, timely risk factor identification, and optimized strategies for treatment, delivery, and postpartum care.

Case Report. Patient B, a 36-year-old woman, was urgently admitted to the hospital on the sixth day postpartum due to severe dyspnea, lower limb edema, hypertension (150/100 mmHg), and progressive acute heart failure with pulmonary edema. Her medical history included a first pregnancy achieved through in vitro fertilization, uterine fibroid, and gestational hypertension (180/100 mmHg) at 13 weeks, requiring inpatient management. Pre-pregnancy cardiology evaluation ruled out congenital heart disease. Cesarean delivery was performed at 40 weeks due to cephalopelvic disproportion. In the early postpartum period, the patient developed postpartum hemorrhage due to uterine atony and anemia, treated with uterine balloon tamponade and blood transfusion, followed by progressive dyspnea, peripheral edema, tachycardia, diffuse pulmonary rales, and oxygen desaturation (SpO_2 88 %).

Chest radiography at the maternity hospital suggested pneumonia, prompting empiric antibacterial therapy, oxygen supplementation, antihypertensive treatment, and thromboembolism prophylaxis. Despite therapy, the patient's condition worsened, with progressive dyspnea and hypoxemia, leading to consultation with specialists from the Amosov National Institute of Cardiovascular Surgery. Clinical assessment and diagnostic testing revealed heart failure with pulmonary congestion. The patient was transferred to the intensive care unit (ICU) for further evaluation and treatment.

The diagnostic workup included comprehensive laboratory and instrumental evaluations: electrocardiography (ECG), echocardiography with speckle-tracking, contrast-enhanced cardiac magnetic resonance imaging (MRI), chest X-ray and computed tomography (CT), complete blood count, biochemistry, coagulation profile, D-dimer, C-reactive protein (CRP), procalcitonin, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and cardiac and liver enzymes.

Electrocardiography showed sinus rhythm, heart rate 110 bpm, PQ interval 0.12 s, low R-wave progression, and incomplete right bundle branch block. Echocardiography revealed:

- Mitral valve: leaflet thickening, severe regurgitation (regurgitant orifice area 11 cm^2);
- Aortic valve: trace regurgitation, gradient 4 mmHg, ascending aorta 28 mm;
- Tricuspid valve: moderate regurgitation;
- Moderate pulmonary hypertension;
- Left atrium: 48 mm; right atrium: 41 mm;
- Left ventricle: end-diastolic volume 119 mL, end-systolic volume 75 mL, stroke volume 44 mL, ejection fraction 35–37 % due to diffuse hypokinesia;
- Bilateral pleural effusion (Figure 1).

Chest CT confirmed pulmonary edema and bilateral hydrothorax (Figure 2). Cardiac MRI demonstrated features consistent with peripartum cardiomyopathy: diffuse myocardial edema, left ventricular (LV) dilatation, reduced LV systolic function (ejection fraction 33 %), and small pericardial effusion (Figure 3).

Laboratory findings included:

- Complete blood count: hemoglobin 160 g/L, RBC 5.3×10^{12}/L, ESR 20 mm/h, WBC 7.1×10^{9}/L (basophils 1 %, eosinophils 0 %, band neutrophils 4 %, segmented neutrophils 85 %, lymphocytes 7 %, monocytes 3 %), platelets 177×10^{9}/L;
- Biochemistry: NT-proBNP 5089 pg/mL, CK-MB 10 U/L, total CK 66 U/L, lactate 1.46 mmol/L, albumin 38.2 g/L, creatinine 89 μmol/L, ALT 27 U/L, AST 16 U/L, glucose 4.8 mmol/L, serum amylase 34 U/L, alkaline phosphatase 268 U/L, CRP 7.5 mg/L, procalcitonin 0.14 ng/mL;
- Coagulation: prothrombin index 84 %, fibrinogen 6.75 g/L.

Elevated NT-proBNP (5089 pg/mL) confirmed severe heart failure, correlating with clinical symptoms (dyspnea, edema) and myocardial dysfunction.

Following diagnostic evaluation, the final diagnosis was: peripartum cardiomyopathy; acute heart failure

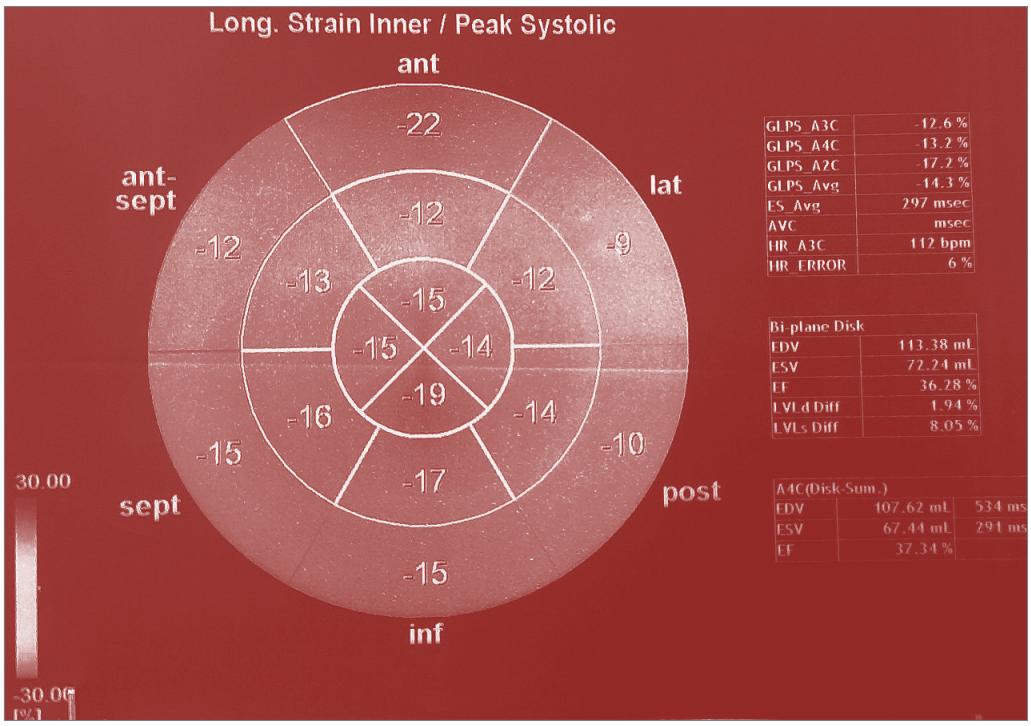

Figure 1. *Echocardiography with speckle tracking (ST): GLS = –14.3 % (global longitudinal strain is reduced)*

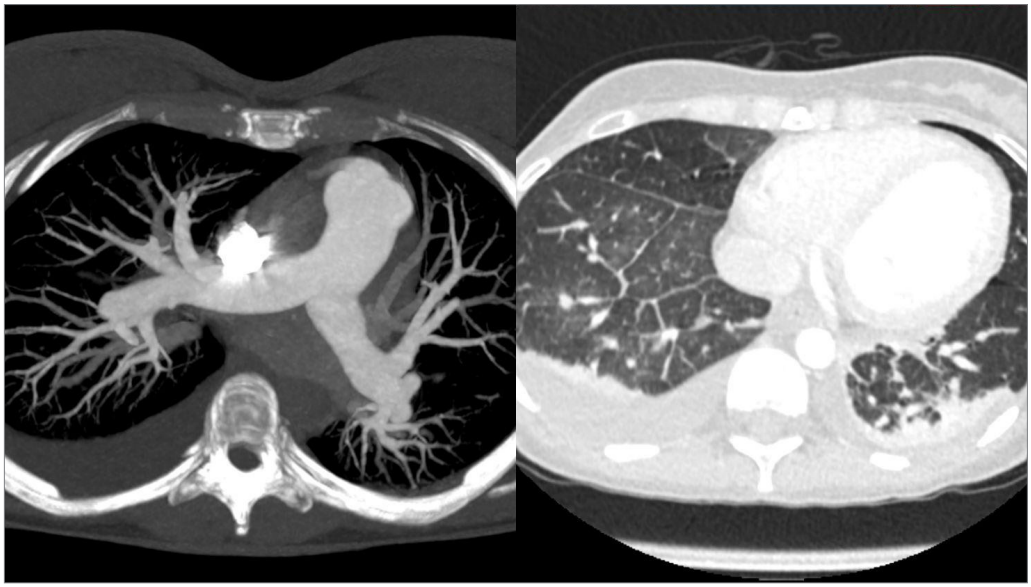

Figure 2. *Chest CT of a 36-year-old woman with peripartum cardiomyopathy at admission, showing cardiomegaly with pulmonary edema and bilateral pleural effusion, without signs of PE*

(Killip Class III, pulmonary edema); severe mitral regurgitation; left atrial dilation; moderate tricuspid regurgitation; moderate pulmonary hypertension; significant bilateral pleural effusion; NYHA Functional Class IV.

The patient was managed by a multidisciplinary team of cardiologists, obstetricians, cardiac surgeons, and intensivists. Bilateral thoracentesis was performed, and acute heart failure therapy was initiated in the intensive care unit (ICU) for five days, resulting in improvement of the clinical condition as well as laboratory and instrumental findings. Following stabilization, guideline-directed medical therapy (GDMT) for heart failure was initiated. GDMT included:

Sacubitril/valsartan 50 mg twice daily, titrated to 200 mg twice daily or the maximum tolerated dose;

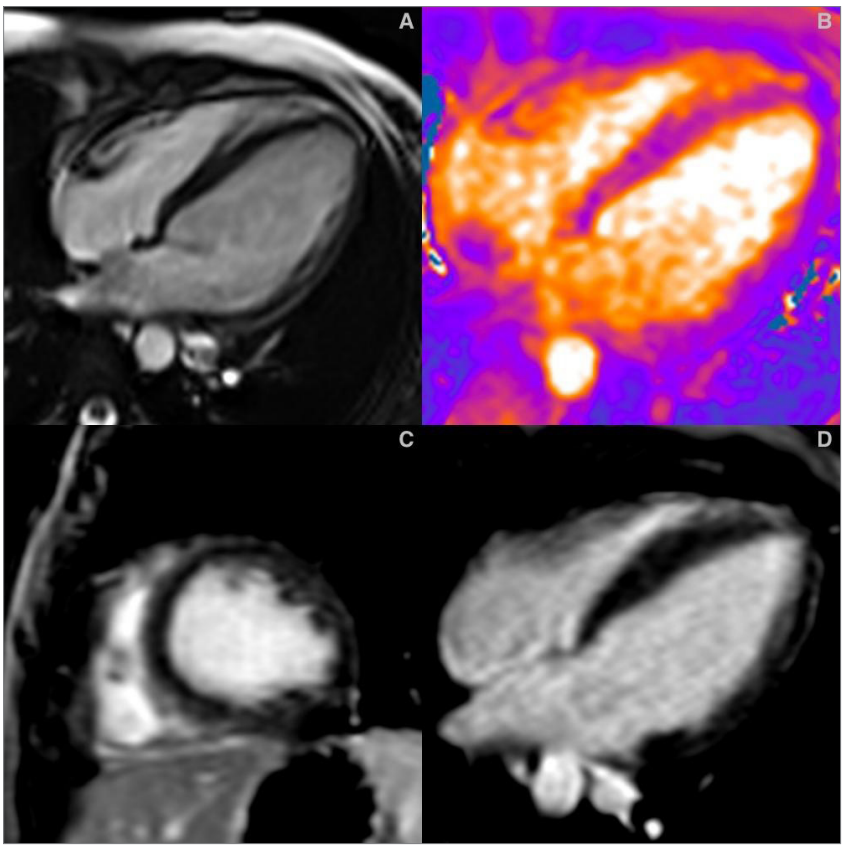

Figure 3. *Cardiac MRI of a 36-year-old woman with peripartum cardiomyopathy.*
A – four-chamber cine SSFP: dilatation of the LV and reduced LV ejection fraction;
B – four-chamber T2-mapping: normal T2 time (49 ms);
C, D – short-axis and four-chamber LGE: no signs of inflammation or myocardial fibrosis.

- Carvedilol 3.125 mg twice daily, titrated to 25 mg twice daily or the maximum tolerated dose;
- Eplerenone 25 mg daily, titrated to 50 mg daily;
- Empagliflozin 10 mg daily;
- Enoxaparin 60 mg/0.6 mL daily for 6 weeks, with coagulation monitoring;
- Bromocriptine 2.5 mg daily for 6 weeks.

At discharge, the patient's clinical condition was stable, with blood pressure 110/75 mmHg, heart rate 80 bpm, oxygen saturation 98 %, and no peripheral edema. Echocardiography showed reduced mitral regurgitation (from severe to moderate), decreased left atrial size, and an increase in left ventricular ejection fraction (LVEF) to 40 %. No pleural effusion was observed. NT-proBNP was 533.2 pg/mL, with other laboratory parameters within normal limits.

Follow-Up Outcomes

At the 2-month follow-up, further improvement was documented. Mitral and tricuspid regurgitation reduced to mild, left ventricular end-diastolic volume (LVEDV) decreased from 119 to 89 mL, LVEF increased to 41 %, and NT-proBNP normalized to 106 pg/mL (reference range: <125 pg/mL).

At 6-month follow-up, near-complete recovery of myocardial and mitral valve function was observed, with no evidence of pathological left ventricular remodeling. Echocardiography revealed:

- Mild mitral and tricuspid regurgitation (hemodynamically insignificant);
- No significant pulmonary hypertension;
- LVEDV 92 mL, LVEF 50 %, with mild interventricular septal mechanical dyssynchrony;
- Global longitudinal strain (GLS) -20.7 %, indicating preserved myocardial function (Figure 4).

14-Month Follow-Up

At 14-month follow-up, the patient achieved complete recovery of myocardial and mitral valve function. Her clinical condition was stable, with no symptoms reported. Echocardiography revealed:

Normal cardiac chamber dimensions (left atrial size 29 mm);

Preserved left ventricular systolic function (LVEF 60 %, LVEDV 92 mL);

Minimal mitral and tricuspid regurgitation;

Global longitudinal strain (GLS) -22.2 %, within normal range (-18 % to -22 %) (Figure 5).

The patient had discontinued heart failure therapy three months prior to the 14-month follow-up.

Discussion. Early diagnosis of peripartum cardiomyopathy (PPCM) is critical for maternal and perinatal out-

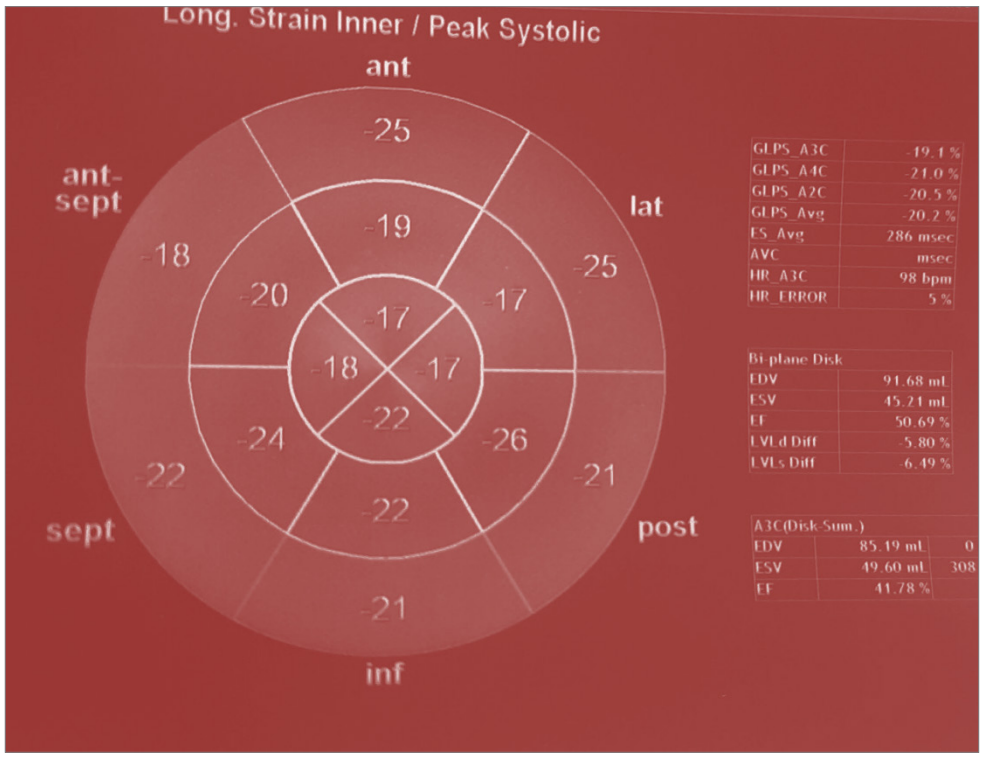

Figure 4. *Echocardiography with speckle tracking (ST): GLS = -20.7 % (globally preserved) at 6-month follow-up*

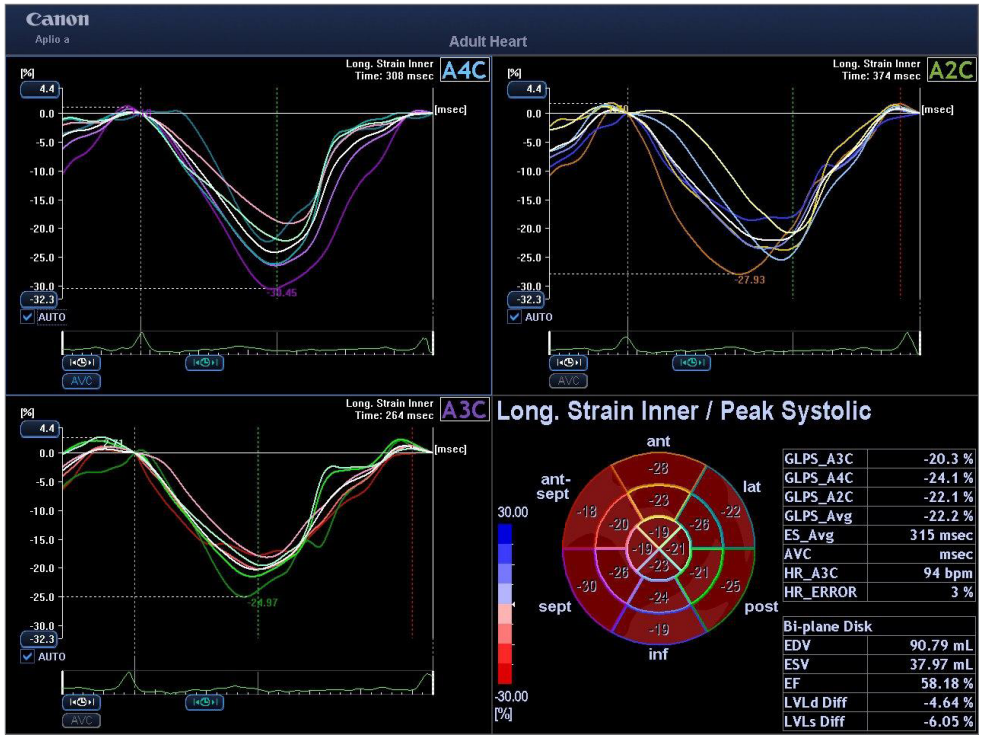

Figure 5. *Echocardiography with speckle tracking (ST) at 14-month follow-up. GLS = -22.2 % (preserved)*

comes, enabling timely treatment and reducing the risk of complications such as heart failure, arrhythmias, thromboembolism, and sudden cardiac death. No epidemiological data on PPCM prevalence are currently available in Ukraine. However, clinical cases and the experience of our multidisciplinary cardio-obstetric team confirm the presence of PPCM in the country. Between 2015 and 2024, 15 pregnant and postpartum women with PPCM

were treated at the Amosov National Institute of Cardiovascular Surgery and the Lukyanova Ukrainian Center of Maternity and Childhood. In the absence of national epidemiological data, international statistics suggest that PPCM prevalence in Ukraine may be underestimated due to limited diagnostic and reporting capabilities, underscoring the need for large-scale studies and a structured surveillance system to ensure timely diagnosis and effective management.

Despite its rarity, ongoing research and clinical efforts in Ukraine aim to improve PPCM diagnosis and management. Recent literature indicates that PPCM has a genetic basis, with shared mutations linking it to dilated cardiomyopathy (DCM) [13]. This highlights the need for lifelong follow-up and monitoring of pharmacological therapy to prevent recurrence and optimize long-term outcomes.

Conclusions. Peripartum cardiomyopathy remains a significant cause of maternal and perinatal morbidity and mortality. Timely diagnosis and effective management of this condition are achievable only through close collaboration within a multidisciplinary team comprising obstetrician-gynecologists, cardiologists, cardiac surgeons, and anesthesiologist-intensivists. Coordinated efforts of these specialists are essential for optimizing the management of pregnancy, delivery, and the postpartum period, as well as for improving long-term outcomes for both mother and child.

Conflict of Interest
The authors declare no conflicts of interest.

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Клінічний випадок мультидисциплінарного супроводу перипартальної кардіоміопатії

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Резюме

Перипартальна кардіоміопатія – це форма серцевої недостатності, яка виникає під час вагітності або у післяпологовому періоді та характеризується зниженням систолічної функції лівого шлуночка [1]. Перипартальна кардіоміопатія посідає вагоме місце серед причин материнської смертності у всьому світі.

Мета. Продемонструвати на прикладі клінічного випадку значення ранньої діагностики, своєчасного виявлення факторів ризику та визначення оптимальної стратегії лікування, розродження та супроводу післяпологового періоду.

Клінічний випадок. У статті описано випадок супроводу мультидисциплінарною командою та віддалені результати породіллі із перипартальною кардіоміопатією. Пацієнтка Б., 36 років екстрено поступила до кардіохірургічного закладу на 6-ту добу післяпологового періоду у зв'язку із наростанням симптомів гострої серцевої недостатності. Після дообстеження встановлено діагноз –перипартальна кардіоміопатія, гостра серцева недостатність, Killip III (набряк легень), важка мітральна недостатність, дилатація лівого передсердя, помірна тристулкова недостатність, помірна гіпертензія легеневої артерії, значний двосторонній гідроторакс, фракція викиду (ФВ лівого шлуночка ЛШ) 35-37 %, ФК NYHA IV. Проведена плевральна пункція з обох сторін, терапія гострої серцевої недостатності в умовах відділення реанімації та інтенсивної терапії протягом 5 днів, після чого загальний стан покращився та відзначено позитивну динаміку клініко-лабораторних та інструментальних показників. Пацієнтка отримувала інтенсивну терапію серцевої недостатності, та перебувала під наглядом мультидисциплінарної команди у складі кардіологів, акушер-гінекологів, кардіохірургів, лікарів реаніматологів. При виписці загальний стан пацієнтки був задовільним, за даними ехокардіографії зменшилася мітральна недостатність з важкої до помірної, зменшилися розміри лівого передсердя, фракція викиду підвищилася до 40%, знизився NT-proBNP до 533,2 пг/мл, інші лабораторні показники в межах нормативних значень. Оцінено віддалені результати через 2, 6 та 14 місяців після лікування. У віддаленому періоді через 14 місяців спостереження відзначалося повне відновлення функції міокарду та роботи мітрального клапану серця, загальний стан пацієнтки був добрим.

Висновки. Перипартальна кардіоміопатія залишається серйозною причиною материнської та перинатальної захворюваності й смертності. Вчасна діагностика та лікування цієї патології можливі лише за умови тісної співпраці мультидисциплінарної команди, до складу якої входять акушери-гінекологи, кардіологи, кардіохірурги та лікарі-анестезіологи-реаніматологи. Скоординовані дії фахівців сприяють оптимізації тактики ведення вагітності, пологів і післяпологового періоду та покращенню довготривалих результатів для матері й дитини.

Ключові слова: *вагітність, післяпологовий період, міокардіальна недостатність, патологія міокарда, фракція викиду, бромкриптин, перинатальні наслідки.*

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