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CASE REPORT WITH LITERATURE REVIEW

Peripartum myocardiopathy after cesarean section: case report

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ABSTRACT

Peripartum myocardiopathy is a form of systolic heart failure involving reduced ventricular ejection fraction (EF<45%), affecting mainly pregnant women in the period close to the newborn birth. The incidence varies globally and is more prevalent in pregnant women of African descent, aged over 30 years, with a history of hypertension and pre-eclampsia and multiple pregnancies. The clinical condition may be mild, but cardiac mechanical support may be necessary in severe cases. The aim of this report is to present a case of a pregnant woman who was admitted to the maternity ward in labor together with cardiac decompensation during cesarean section due to peripartum myocardiopathy. We have reviewed the literature and suggest a better understanding of the disease is urgently needed, especially among anesthesiologists. Thus, early diagnosis and appropriate therapy can improve favorable outcomes.

KEYWORDS

Peripartum cardiomyopathy; cardiac insufficiency; twin pregnancy; cardiogenic shock

INTRODUCTION

Peripartum myocardiopathy (PM) is a form of systolic heart failure characterized by a reduced ventricular ejection fraction (EF <45%), affecting primarily women during the last trimester of pregnancy up to the fifth month postpartum, in the absence of other identifiable causes[1].

The incidence of PM varies globally, being more frequent in Nigeria (1:100 pregnancies) and Haiti (1:300 pregnancies)[2-4]. In the United States, the incidence ranges from 1:1,000 to 1:4,000 pregnancies. An increase in the number of cases involves advanced maternal age, *in vitro* fertilization techniques that predispose to multiple pregnancies, and improved awareness of the condition[5].

Diagnosis may be delayed because the signs and symptoms of PM (cough, orthopnea, paroxysmal nocturnal dyspnea, fatigue, palpitations, hemoptysis, chest, and abdominal pain) can be masked by the typical symptoms of late pregnancy and the peripartum period[6].

The exact pathogenesis of PM remains enigmatic. Various mechanisms, including myocardial inflammation, oxidative stress, vascular abnormalities, and autoimmune factors, have been proposed to explain the onset and progression of the disease. The 16Kda form of prolactin impacts cardiac microvasculature, leading to its dysfunction. Protein genetic alterations (relaxin, dystrophin, and troponins) may similarly predispose women to PM[7,8]. An Israeli study described three cases of PM occurring between December 2020 and January 2021 following COVID-19 infection, suggesting that even mild SARS-CoV-2 infection may predispose individuals to PM[9].

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Management strategies for PM are similar to heart failure from other causes, but it is crucial to ensure that chosen medications are safe for the fetus and neonate[10]. The mortality rate varies from 3% to 40% and is directly related to the recovery of the ejection fraction[11,12].

Here, we described a case of peripartum myocardiopathy after a cesarean section in a patient who provided informed consent. The study was approved by the hospital's ethical committee.

CASE DESCRIPTION

Female patient, 35 years old (77 kg, 165 cm), with a history of gestational hypertensive disorder during her last pregnancy five years ago, G7P2A4 (Gravida 7 Para 2 Abortions 4), with 13 prenatal consultations, twin pregnancy, $36^{3/7}$weeks gestation. No other significant medical history. She was admitted with complaints of lower abdominal pain and a large amount of amniotic fluid loss. On physical examination, blood pressure was 150/100 mmHg, heart rate was 89 bpm, fetal movements were present, breech presentation, fetal heart rate 148 bpm and 128 bpm, cervix was medium, posterior, 1 cm dilated, membranes ruptured. She was admitted for routine management of gestational hypertensive disorder.

Laboratory tests at admission were: Hb=11.1g/dl, Ht=34%, platelets =238,000, Na=134 mEq/L, K= 3.8 mEq/L, BUN=11 mg/dl, LDH=200 U/L, AST=18 U/L, ALT=11 U/L, Total proteins=5.6 g/dl, Albumin=2.7g/dl.

She was scheduled for a cesarean section due to interactivity, premature rupture of membranes, and labor prodromes. She was admitted to the operating room with normal vital signs under usual care and monitoring. Spinal anesthesia was performed with 12.5 mg bupivacaine and 100 µg morphine. Etilefrine 1 mg was used in bolus to maintain blood pressure. Birth of male newborn weighing 2200 g and female newborn weighing 1780 g, both with Apgar scores of 8 and 9 at one and five minutes, respectively. After the birth of the second twin, 10 IU of oxytocin was administered by the anesthesiologist as a continuous infusion. A few minutes after the start of oxytocin, the patient developed bradycardia, progressing to asystole. Resuscitation measures were performed, and after the second dose of adrenaline (1mg), spontaneous circulation returned. The patient was responsive but agitated. Orotracheal intubation was performed using propofol, ketamine, rocuronium, and fentanyl. Vital signs were: HR=120 bpm, BP=95/55 mmHg, SpO2=96% on FiO2 of 0.5, and arterial blood gas analysis: PCO2=7.20, PCO2=36.5 mmHg, PO2=181.8 mmHg, Sat O2=99.3%, HCO3=14.3 mEq/L, BE=-12.2, lactate=5.7 mmol/L. Bilateral crepitant rales were heard up to the middle third of the lung fields, and bilateral lower extremity edema was +3/+4. Post-cardiac arrest care was performed, and a cardiology consultation was requested. The cardiologist suggested the following diagnostic hypotheses: Peripartum cardiomyopathy, Acute coronary syndrome due to thrombosis from Gestational Antiphospholipid Antibody Syndrome (APS), and Takotsubo cardiomyopathy. Electrocardiogram and chest X-ray are presented in Figures 1 and 2. Transthoracic echocardiogram showed increased left ventricular cavity dimensions with normal wall thickness, moderate systolic dysfunction due to mid-apical hypokinesis of all segments, and ejection fraction of 34%. Therapeutic measures for cardiogenic shock were initiated (dobutamine and furosemide were administered).

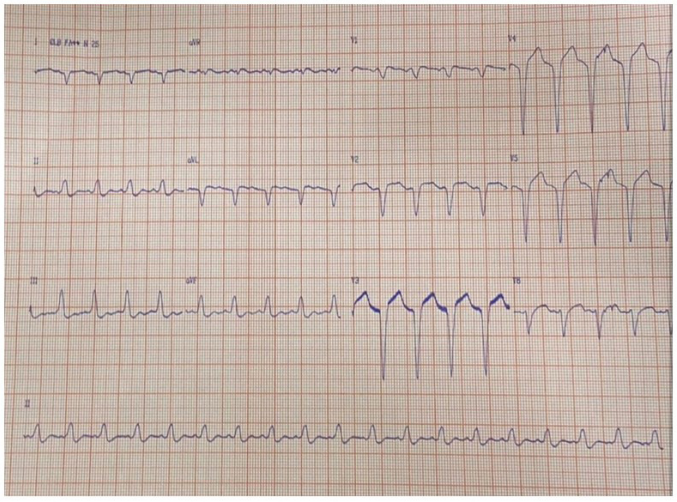

Figure 1. Electrocardiogram after Return of Spontaneous Circulation (Sinus rhythm, left bundle branch block, left ventricular hypertrophy).

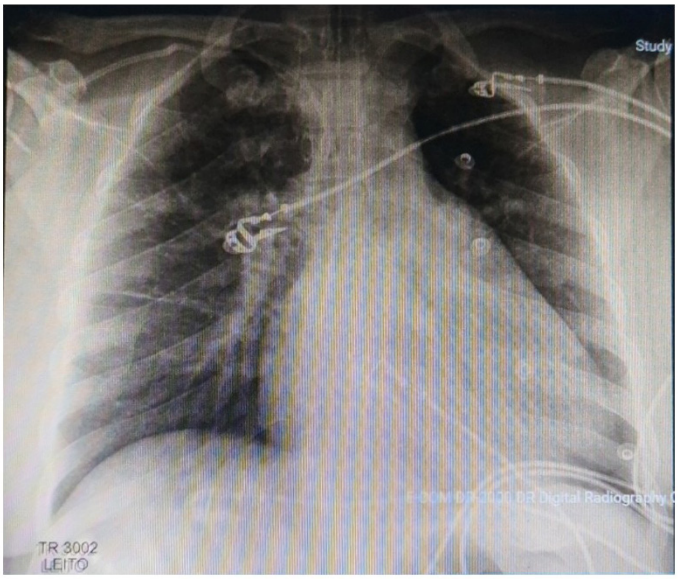

Figure 2. Chest X-ray upon arrival at the Intensive Care Unit.

The patient remained sedated in the intensive care unit with a diagnosis of decompensated heart failure profile C. She was treated with spironolactone, furosemide, carvedilol, orotracheal intubation, advanced invasive monitoring, and vasoactive drugs (milrinone and noradrenaline) for six days. Additional laboratory tests showed high-sensitivity troponin levels in sequence: 0.49 ng/ml, 1.53 ng/ml, 1.36 ng/ml, pro-BNP = 22,721 pg/ml, which later decreased to 9,972 pg/ml. A consultation with a rheumatologist was requested as the patient presented clinical criteria (> three pregnancy losses before 12 weeks of gestational age) for the diagnosis of gestational APS. However, she had only one positive lupus anticoagulant test. Screening for other autoimmune markers (antinuclear antibody, rheumatoid factor, anti-smooth muscle antibody, anticardiolipin IgM/IgG) was negative. Lower limb thrombosis was ruled out by Doppler ultrasound, and coronary thrombosis was excluded by cardiac catheterization. On the sixth day of hospitalization, the patient showed hemodynamic improvement and was successfully extubated. A repeat transthoracic echocardiogram revealed hypertrophic cardiomyopathy with a global reduction in systolic function due to diffuse hypokinesis and two-dimensional ejection fraction = 47%. On the thirteenth day of hospitalization, she was discharged home with her twin newborns and referred for outpatient follow-up. She exhibited normalization of the ejection fraction after six months.

DISCUSSION

A review of the literature indicates that peripartum myocardiopathy is a rare disease of unknown etiology that affects pregnant women from late pregnancy through the postpartum period, leading to left ventricular dysfunction and subsequent congestive heart failure. It is a diagnosis of exclusion after ruling out pulmonary embolism, amniotic fluid embolism, myocarditis, acute coronary syndrome, Takotsubo cardiomyopathy, and preeclampsia[13].

The patient in question had several risk factors, including age over 30, mixed race, multiparity, twin pregnancy, and preeclampsia, with unknown prior heart disease. She experienced decompensation during the cesarean section under spinal anesthesia, which progressed to cardiac arrest and was successfully reversed with resuscitation maneuvers.

A transthoracic echocardiogram performed immediately after the return of spontaneous circulation demonstrated a low ejection fraction (EF=37%) with diffuse hypokinesia, leading to the suspicion of PM.

In treating PM, it is crucial to use medications with established safety profiles for the fetus and newborn. Hydrochlorothiazide and furosemide are usually safe during pregnancy and lactation; however, spironolactone should be used with caution due to insufficient safety data for the fetus and newborn. Angiotensin receptor blockers (ARBs) and angiotensin-converting enzyme inhibitors (ACEIs) are standard treatments for heart failure and are contraindicated during pregnancy (due to risks of renal malformations and fetal death) and lactation because they pass into breast milk[10]. In addition, vasodilators such as hydralazine are safe, and for severe cases, intravenous nitroglycerin may be necessary[10].

The primary goal in managing cardiogenic shock due to PM is to improve inotropy and ensure adequate coronary perfusion. Drugs such as dobutamine, norepinephrine, milrinone, and dopamine are indicated. Levosimendan, which reduces pulmonary capillary pressure and improves cardiac output, could be a therapeutic alternative, although data on its efficacy and safety for the fetus or newborn are lacking[10].

In some studies, Bromocriptine, a dopamine receptor agonist that inhibits prolactin release, has shown promising results. Anticoagulants are similarly recommended due to their potential association with thromboembolic events[10,13].

The patient remained under intensive care, requiring vasoactive drugs (norepinephrine and milrinone), diuretics (furosemide), and a beta-blocker (carvedilol). Her cardiac function improved over five days, leading to hemodynamic stability, orotracheal extubation, and transfer to the general ward.

The clinical course of PM varies considerably, ranging from complete resolution of clinical symptoms and normalization of ventricular function to progression of refractory heart failure, which may require mechanical circulatory support or even heart transplantation[14,15]. Efforts are ongoing to identify markers and risk indicators[16].

A recent publication provides insights into the long-term follow-up of patients over 20 years in European countries. Based on National Health Service (NHS) data, this extensive review observed an 8% mortality rate, involving 76% of patients recovering ventricular function and 13% experiencing worsening cardiac function. Additionally, children born to mothers having PM had three times more adverse events and five times more deaths than children born to control mothers[17].

Patients who experience improved left ventricular dysfunction, like the one described in this case report, will generally have a better prognosis.

Future pregnancies are contraindicated for patients with persistent left ventricular dysfunction due to the risk of

recurrence. Patients who have experienced complete recovery must be closely monitored in subsequent pregnancies until the sixth month postpartum. This monitoring must be done under the guidance of a specialist and include serial examinations to assess ventricular function[18,19]. In this case, the patient underwent a cesarean section and tubal ligation.

Fortunately, the patient was treated at a tertiary hospital where comprehensive diagnostic exams facilitated the diagnostic process. Specific therapy guided by advanced invasive monitoring was initiated in the intensive care unit, a level of care not always available in maternity units across the country.

The consequences of PM are potentially severe, with substantial risks to maternal health, highlighting the importance of a comprehensive understanding of the disease by the multidisciplinary team. We must improve our knowledge about PM to optimize patient care and minimize associated complications.

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