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Case Report

Successful Management of Peripartum Cardiomyopathy

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Abstract

30-year-old parturient with lower limb edema underwent cesarean section under spinal anesthesia developed peripartum cardiomyopathy treated successfully as acute heart failure.

Keywords: *Cardiomyopathy; Peripartum; Cesarean*

Abbreviations

Mg: Milligram; Kg: Kilogram; cm: Centimeter; %: Percentage; $PaCO_2$: Carbon Dioxide Alveolar Partial Pressure; O_2 Sat: Oxygen Saturation; MmHg: Millimeter Mercury; ICU: Intensive Care Unit; Bpm: Breathe Per Minute; ABGs- Arterial Blood Gases; ECG: Electrocardiogram; BP: Blood Pressure; HR: Heart Rate; CXR: Chest X Ray; Temp: Temperature; JVP: Jugular Venous Pressure; CT: Computed Tomography; MRI: Magnetic Resonance Imaging; LMWH: Low Molecular Weight Heparin; ACE-I: Angiotensin-Converting Enzyme Inhibitors; ARB: Angiotensin Receptor Blockers; LVAD: Left Ventricular Assist Device; PCR: Polymerase Chain Reaction; EF: Ejection Fraction; PPCM: Peripartum Cardiomyopathy

Introduction

An idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found. It is a diagnosis of exclusion.

Case Report

A 30-year-old female patient, 80 Kg, 165 cm, Gravida1 Para 0 with 31+1 weeks of gestation, she was known case of primary infertility in the last 10 years. *In vitro* fertilization was successful injected with triplet pregnancy. She was admitted via emergency room complaining of preterm labor.

She also, started to complain of both lower limbs swelling. Laboratory blood tests revealed hypoalbuminemia and trial of exogenous albumin was administered as nephrology team consultation: PCR is negative for nephrotic syndrome, with normal liver function test and they were advised not to give diuretics (as risk for placental insufficiency).

Her lower limbs edema was explained by her multiple gestations. It was planned for cesarean-section at 34+2 gestational week of age under spinal anesthesia.

3 hours after her cesarean section under spinal anesthesia due to preterm labor, the patient developed sudden onset shortness of breath and palpitation.

Obstetric team ordered: ABG's, urgent chest x-ray, and cardiac enzyme. Respiratory team was consulted regarding her respiratory distress condition along with vital signs: BP: 150/80 mmHg HR: 118 beat/min Temp: 37.1 C RR: 28/min, O_2 saturation: 87% and her clinical examination revealed poor air entry bilaterally, JVP was elevated, + 4 lower limbs edema, ABG on room air: 7.40/ 27/ 16.3/ 17.3/ -6.7/ 48/ 82%, her ECG was sinus tachycardia and CXR with pulmonary edema and dilated left ventricle giving a differential diagnosis of Acute pulmonary edema or Pulmonary embolism "amniotic fluid embolism".

The next plan was to do Echo Cardiogram, she was transferred to ICU for closer monitoring, and starting her on therapeutic LMWH. C.T Angiogram was ordered to rule out PE when the patient was stabilized along with oxygen therapy.

Echo cardiogram revealed: LV EF 25%, Grade 2 (moderate) mitral regurgitation, which gave the diagnosis of peripartum cardiomyopathy.

She was managed as acute decompensate heart failure. She was started on intravenous furosemide, oral spironolactone and Beta blocker, with strict input and output while keeping her in the ICU for 5 days. Follow up echo cardiogram was done after one week: LV EF 40%, left atrial enlargement, and grade 1 (mild) mitral regurgitation.

She was discharged home on oral Angiotensin receptor blocker 80mg once daily, furosemide 40mg, and on Beta blocker.

Discussion

Peripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopathy that is defined as deterioration in cardiac function presenting typically between the last month of pregnancy and up to six months postpartum. As with other forms of dilated cardiomyopathy, PPCM involves systolic dysfunction of the heart with a decrease of the left ventricular ejection fraction (EF) with associated congestive heart failure and an increased risk of atrial and ventricular arrhythmias, thromboembolism, and even sudden cardiac death [1-5].

PPCM is a diagnosis of exclusion, where in patients have no prior history of heart disease or other known possible causes of heart failure. Echocardiogram is the diagnostic as well as monitoring the effectiveness of treatment for PPCM [1-5].

The cause of PPCM is unknown. Currently, researchers are investigating cardiotropic viruses, or immune system dysfunction, other toxins that serve as triggers to immune system dysfunction, micronutrient or trace mineral deficiencies, and genetics is a possible components that may contribute to the development of PPCM [1,3,6].

Symptoms usually include one or more of the following: orthopnea, dyspnea, pitting edema, cough, frequent night-time urination, excessive weight gain during the last month of pregnancy, palpitations and chest pain [1,3].

Unfortunately, patients and clinicians sometimes dismiss early symptoms because they appear to be typical of normal pregnancy as initially in our case. Yet, early detection and treatment are critically important to the patient with PPCM. Delays in diagnosis and treatment of PPCM are associated with increased morbidity and mortality [1,3-5,8,9].

It is paramount that clinicians hold a high suspicion of PPCM in any peril- or postpartum patient where unusual or unexplained symptoms or presentations occur [1,3,7,10,11].

Early detection and treatment are associated with higher rates of recovery and decreased morbidity and mortality [1,3-5,8,9].

Treatment for PPCM is like treatment for congestive heart failure. Conventional heart failure treatment includes the use of diuretics, beta blockers (B-B), and angiotensin-converting enzyme inhibitors (ACE-I) after delivery. Diuretics, preferably furosemide, help the body to get rid of excess water weight and lower blood pressure. ACE-I and B-B improve blood circulation and contribute to the reversal of the immune system dysfunction associated with PPCM. If ACE-I is not well tolerated by the patient like ours, it can be replaced by angiotensin receptor blockers (ARB). Hydralazine with nitrates may replace ACE-I in breastfeeding mothers or before delivery; however, evidence suggests that this course of treatment may not be as effective as ACE-I but beneficial when necessary [1,3-5,7,9,12,13].

If EF is less than 35%, anticoagulation is indicated, as there is a greater risk of developing left ventricular thrombi. Sometimes implantation of a left ventricular assist device (LVAD) or even heart transplant also becomes necessary [1,4,5,7,14].

It is important that the patient receives regular follow-up care as we did in our patient, including frequent echocardiograms to monitor improvement or deterioration.

Patients who do not respond to initial treatment, defined as left ventricular EF remaining below 20% at two months or below 40% at three months with conventional treatment may merit further investigation, including cardiac magnetic resonance imaging (MRI), cardiac catheterization, and endomyocardial biopsy for special staining and for viral polymerase chain reaction (PCR) analysis [7].

The most recent studies indicate that with newer conventional heart failure treatment consisting of diuretics, ACE inhibitors and beta blockers, the survival rate is very high at 98% or better, and almost all PPCM patients improve with treatment [13,15,16].

It is a misconception for recovery depends upon improvement or recovery within the first six to 12 months of diagnosis like our case. Still, many women continue to improve or recover even years after diagnosis with continued medicinal treatment [3,17]. Once fully recovered, if there is no subsequent pregnancy, the possibility of relapse or recurrence of heart failure is minimal [13,16].

Subsequent pregnancy should be avoided when left ventricular function has not recovered and the EF is lower than 55% [1,18]. The chance of relapse may be even smaller for those with normal contractile reserve as demonstrated by stress echocardiography [18-20] in any subsequent pregnancy, careful monitoring is necessary. Where relapse occurs, conventional treatment should be resumed, including hydralazine with nitrates plus beta-blockers during pregnancy, or ACE-inhibitors plus beta-blockers following pregnancy.

Conclusion

Peripartum cardiomyopathy mimics changes occurring in normal pregnancy. It should be treated like any other cardiac failure along with anti coagulant therapy. Epidural anesthesia is preferable with close observation and monitoring in ICU.

Availability of Data and Materials

Not applicable.

Ethical Approval

Not required.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and/or any accompanying images.

Consent

The patient is consented.

Competing Interests

I declare that I have no competing interests.

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Author Contribution

A. AJ has written the case report and he is the doctor in charge of the patient.

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