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An interesting case report of peripartum cardiomyopathy

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Abstract

27 year old female P1L1 post Lower Segment Cesarean Section who presented with complaints of breathlessness 3-4 hours post her emergency LSCS which had settled with Oxygen via nasal cannula. Patient was asymptomatic on room air after which she was discharged on day 3 of LSCS. Patient presented in the emergency department of our hospital on day 4 post LSCS with complaints of breathlessness at rest since 6 hours. Patient was immediately admitted, evaluated and diagnosed as a case of Peripartum Cardiomyopathy. She was stabilized and managed conservatively.

Keywords: Cesarean section, breathlessness, peripartum cardiomyopathy

Introduction

PPCM is a dilated cardiomyopathy defined as systolic heart failure in the last month of pregnancy or within 5 months of delivery.

It includes four criteria

- Development of cardiac failure.
- Absence of an identifiable cause of heart failure.
- Absence of a recognizable heart disease before last month of pregnancy.
- Left ventricular dysfunction (EF<45%)^[1]

Incidence: 1 in 3000-4000 live births^[1].

Risk factors: Black race, multiparity, advanced maternal age, multiple pregnancy^[1, 2] (Our - 3, 4)
Symptoms include fatigue, dyspnea at rest, edema^[3].

Differential diagnosis for this condition can be Pulmonary Thromboembolism, amniotic fluid embolism or eclampsia^[3]. Mortality rate is very high (20-25%) and recurrence rate in subsequent pregnancy is also very high (50%).

Case Report

A 27 year old Asian woman presented for the first time in the emergency department of our hospital on day 4 postpartum with complaints of breathlessness at rest and fatigue since 12 hours. She was found to be dyspneic and hypoxic with saturation on room air below 90%. Patient was admitted for further evaluation and management. Patient had a similar episodes 3 hours post her emergency LSCS in the hospital where she had delivered. According to her case files, she was managed at that hospital with 8L of nasal O₂ which gradually tapered off and then stopped after which she was maintaining O₂ saturation on 98% on room air and she was discharged on Day 3.

Her antenatal period was uneventful. She underwent emergency LSCS in view of fetal distress. Her intraoperative and immediate post operative period involved no complications.

During examination, General condition of the patient was fair, she was afebrile, had a pulse rate of 110 bpm, blood pressure of 110/70 mm of Hg, respiratory rate of 20 per minute and oxygen saturation was 90% on room air.

On auscultation, respiratory system examination was normal and air entry was bilaterally equal, no abnormal breath sounds heard. Jugular venous pressure was not raised. Her extremities were non edematous. She had no calf tenderness. Patient was admitted subsequently. Electrocardiography was normal, Chest X ray showed cardiomegaly. Urine analysis was negative for protein. Plasma levels of D-Dimer was 1500. Circulating level of NT Pro BNP was 9313 pg/mL.

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A Transthoracic Echocardiography was done after admission which showed Left Ventricular Ejection Fraction of 20-25%, mild dilated Left Ventricle, grade 1-2 Mitral regurgitation, Global hypokinesia, Grade 2 Diastolic Dysfunction.

She was given Injectable Furosemide 20mg IV for diuresis. She was started on Low molecular weight Heparin (Injectable Clexane) 0.6mg, Ivabradine 5mg, Carvedilol 3.125mg. She improved symptomatically with treatment and was able to maintain saturation on room air. Her fatigue and dyspnoea reduced significantly.

Patient was discharged on Tablet Torsemide 10mg, Tablet Ramipril 25mg, Tablet Bisoprolol, Tablet Spironolactone 25mg, Tablet Bromocriptine 5mg.

Discussion

Approximately 60-70% of women experience a sensation of dyspnea during the course of normal pregnancy. Risk factors for PPCM include multiparity, advanced maternal age, multifetal pregnancy, pre-eclampsia and gestational hypertension [4]. However, contemporary trends show that there is an increasing incidence (24%-37%) in young primigravida and white women [4]. The exact etiology of PPCM is still unknown. However, a number of possible causes have been still proposed for PPCM which include myocarditis, abnormal immune response to pregnancy, maladaptive immune response to hemodynamic stresses of pregnancy, stress activated cytokines and prolonged tocolysis. There have been few reports of familial PPCM, raising the possibility that some cases are actually familial dilated cardiomyopathy unmasked by the stress of pregnancy [5].

Recent data suggest a central role of unbalanced peri/post partum oxidative stress that triggers the proteolytic cleavage of the nursing hormone prolactin into a potent antiangiogenic, pro-apoptotic and pro-inflammatory 16-kDa PRL fragment. This notion is supported by the observation that inhibition of Prolactin secretion by bromocriptine, a dopamine D2 receptor agonist [6].

Clinical Features and Diagnosis

Many symptoms of PPCM overlap with the normal symptoms in the last month of pregnancy like dyspnea, fatigue and pedal edema. Therefore, PPCM can go unrecognized leading to underestimation of incidence [7].

The differential diagnosis includes accelerated hypertension, diastolic dysfunction, systemic infection, pulmonary embolism and obstetric complications such as pre-eclampsia, eclampsia and amniotic fluid embolism.

Imaging studies include electrocardiogram, chest radiography and echocardiography. Electrocardiogram findings are often normal but can include sinus tachycardia, non specific ST and T wave abnormalities and voltage abnormalities. Chest radiographs can show signs of pulmonary congestion, cardiac enlargement and even pleural effusions [7].

The diagnosis of PPCM rests on the Echocardiographic identification of new left ventricular systolic dysfunction during the period surrounding parturition.

Management

Therapy regimens include diuretics to reduce preload and to treat pulmonary congestion or peripheral edema. Diuretics like Furosemide and Hydrochlorothiazide are safe during pregnancy [8].

Inotropic agents: Drugs like Dopamine and Dobutamine are used only in cases of low urine output.

Beta blockers: Beta1 selective blockers like Carvedilol are preferred for long term systolic dysfunction.

Anticoagulants: Cardiac failure and pregnancy are independent risk factors for thromboembolism. Low Molecular Weight Heparin (LMWH) antepartum and LMWH/warfarin post partum are recommended to be used when Ejection fraction is <30% [8].

Mechanical device circulatory support and transplantation:

In critical cases, mechanical support and cardiac transplantation might be needed. In cases of fulminant PPCM, LVAD can be used in the interim period as PPCM usually resolves within 3-6 months [9].

Non-pharmacological management: Salt and water restriction are important in patient management.

Bromocriptine reduces the production of Prolactin by dopamine agonists. It is supposed to improve outcomes in patients of peripartum cardiomyopathy by eliminating the cleaved form of prolactin [6].

Immunosuppressive therapy can be if myocardial biopsy indicates myocarditis and if no therapy works after two weeks of standard heart failure [10].

Prognosis: Recovery from PPCM is defined as improvement of LVEF>50% or increase in EF by 20%. It takes 3-6 months post partum for recovery. Even after complete recovery risk of recurrence in subsequent pregnancies remains high and EF may worsen again [1]. Therefore, subsequent pregnancies must be avoided and if cannot be avoided should be managed in collaboration with a high risk perinatal center.

Conclusion

We have presented the above case report to highlight Peripartum Cardiomyopathy which is a rare cause of dyspnea in pregnancy and how it is crucial to differentiate it from other causes of dyspnea in pregnancy. The above case demonstrates a typical presentation of PPCM with hypoxia, breathlessness, elevated NT pro BNP levels, reduced ejection fraction on Echocardiography. Thus it is important for Obstetricians and Physicians to be familiar with PPCM as a potential diagnosis and therefore consider it while diagnosing a dyspneic antenatal and postnatal patient to hasten medical treatment for a potentially fatal condition.

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