Peripartum Cardiomyopathy- An Enigma in Intensive Care Unit- Case Report and Brief Discussion

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ABSTRACT
Peripartum cardiomyopathy is an idiopathic dilated cardiomyopathy, that presents with left ventricular systolic dysfunction. Early diagnosis before the advancement of pathology holds significance and chances of recovery are 100% if ejection fraction (EF) >45% at the time of diagnosis. A number of pathophysiological mechanisms have been proposed but none is clearly defined and proven. Definitive diagnosis is based on ECHO findings - bedside ECHO should be done as soon as possible. β blockers are very promising drugs. Some authors are of the view to continue these upto 1 year due to chances of relapse of left ventricular changes. Awareness and vigilance are required for diagnosis of PPCM.

Keywords: Peripartum cardiomyopathy, systolic dysfunction, β blockers

INTRODUCTION
Peripartum cardiomyopathy (PPCM) is a very rare, challenging and often misdiagnosed idiopathic dilated cardiomyopathy, that presents with left ventricular systolic dysfunction during the last trimester and upto 6 months postpartum. It mimicks the signs and symptoms of normal pregnancy and remains as the diagnosis of exclusion.

Time is the master and ‘a stitch in time saves nine’ holds true. Early diagnosis before the advancement of pathology holds significance and chances of recovery are 100% if ejection fraction (EF) >45% at the time of diagnosis.1 2 We report a case of PPCM in our ICU-

CASE REPORT
A 24 years female presents in postpartum period with dyspnea, dizziness and tachycardia with ECG changes showing arrhythmia. Vitals were- BP-130/94, pulse 130/min irregular with T wave inversion. She was immidiately intubated and put on pressure support mode (P-SIMV) on sedation with inj. Fentanyl (80mcg/hour). Cardiology opinion taken. Bedside ECHO showed left ventricular systolic dysfunction, normal ventricular size, EF 35%, wall motion abnormalities present.

Chest X RAY showed- diffuse pulmonary congestion and mild pleural effusion. ECG- T wave inversion in almost all the leads, irregular rhythm, heart rate-110/min. PPCM with severe hypokalaemia was suspected and immediately following treatment started inj magnesium 2 gm over 30 mints (diluted in 100 ml NS), inj. KCL 40 units over 4 hours, inj Ivabradin (2.5 mg BD), Tab Carvedilol through ryle’s tube (6.25 mg). Patients ECG reverted to normal after treatment. She was successfully extubated after 24 hours. She recovered completely and was shifted to ward.

DISCUSSION
PPCM is a diagnosis of exclusion, a very rare form of dilated cardiomyopathy, and aetiology is still a topic of debate. Though the definition PPCM is not clearly defined, in 1971 Demarkis et al in their study on 27 patients with cardiac abnormality (ECG changes, cardiomegaly and heart failure in peripartum period defined PPCM as:

- Development of heart failure in the last month (8 month) or within 5 months postpartum.
- No valid aetiology found
- Absence of cardiac disease before last month of pregnancy

PPCM has a high incidence of 1 in 1300 to 1 in 15000 pregnancies worldwide but the remains unsolved mainly because it disguises as normal pregnancy and postpartum changes. The major risk factors include elderly multipara, obesity, previous history of cardiac ailments like myocarditis others being smoking, use of certain drugs, PIH, preeclampsia.

PPCM is an abnormality of systolic left ventricular function. A number of pathophysiological mechanisms have been proposed but none is clearly defined and proven. Thus the management is essentially supportive. Recently the role of oxidative stress mediated cleavage of hormone prolactin into biologically active subfragment i.e. 16kDa prolactin has been proposed as the major factor initiating PPCM. Most efficient biomarker reported is NT-proBNP.

The criterion proposed by Demarkis (defined earlier) comes in very handy to help in diagnosis. The differential diagnosis of PPCM is heart failure, myocardial infarction, idiopathic dilated cardiomyopathy, pulmonary embolism, severe preclampsia, sepsis, ventricular septal defect. ECG pattern may show bundle branch block, ventricular tachycardia, atrial fibrillation, thus not diagnostic. Chest X ray may show pleural effusion, cardiomegaly and congestion. Definitive diagnosis is based on ECHO findings- bedside ECHO should be done as soon as possible. ECHO findings in our case were decreased cardiac output (EF 35%), wall motion abnormality, systolic dysfunction, no abnormal ventricular dilatation.

The most important challenge was to distinguish PPCM in preclamptic patient or any complication of preeclampsia such as (MI, heart failure). ECHO findings are heart failure with decreased EF (PPCM) and EF is maintained in preclampsia.

Various monitoring techniques have been proposed for patients of PPCM in ICU. The choice depends on the severity of PPCM. Intraarterial BP measurement is important as patients may show varied changes. Swan Ganz catheter is employed when there is severe reduction in myocardial contractility, dyspnea etc. In a study the role of continuous non invasive CO monitoring was found very useful in PPCM. It relates closely with pulmonary artery catheter and very useful in heart decompensation.

Treatment includes maintenance of ABC (Airway, breathing, circulation). Our patient presented with symptoms in postpartum period. Thus the safety of drugs for foetus was not an issue. Ventilatory support started and target was SPO2 >95%. Aims of management are decreasing preload and afterload, increasing cardiac contractile force. According to European society of cardiology guidelines the 3 neurohumoral antagonists β blockers, ACE inhibitors and mineralocorticoid antagonist should be considered in every patient showing signs of heart failure and reduction of cardiac function. Also the guidelines suggest the use of ivabradin in symptomatic patient (NYHA functional class II- IV) with sinus rhythm, HR> 70 beats per min and left ventricular ejection fraction (LVEF) <35% inspite of treatment with the above mentioned drugs. β blockers are very promising drugs. Some authors are of the view to continue these upto 1 year due to chances of relapse of left ventricular changes. Non selective β blockers are usually avoided (antitocolytic effects). β -1 selective blockers carvidolol is preferred. It is both α and β selective blocker, very effective in PPCM. Diuretics are helpful in relieving symptoms of congestion in PPCM. These reduce both pre and afterload. There are high chances of left ventricular thrombosis and embolism in PPCM patients. Thus anticoagulation should be started early in PPCM esp if EF<35%. Low molecular weight heparin is preferred that can be shifted to warfarin postpartum.

Recently bromocriptine has shown promising results. It is a dopamine antagonist and inhibits prolactin secretion. Its role in PPCM is yet to be confirmed. Ivabradin is very useful esp. when symptoms of left
ventricular failure are present. It decreases the symptoms of heart failure and improves quality of life. Another novel drug Pantoxyphyllin (blocker of TNF-α receptors) has shown improved outcomes in PPCM. But its effect requires further evaluation.

Our patient had severe hypokalemia on presentation which was effectively managed with potassium supplements and loading dose of magnesium.

**CONCLUSION**

The rarity of the disease and mimicry of PPCM with normal physiology of pregnancy often leads to its misdiagnosis. Delay in diagnosis leads to irreparable damage and increases the severity thus making the prognosis dismal. Bedside ECHO should be employed as early as possible for better outcome. All the risk factors should be kept in mind including the fact of previous heart disease. Awareness and vigilance are required for diagnosis of PPCM. Minor symptoms of dyspnea fatigue and malaise should not be overlooked. Any postpartum patient with paroxysmal nocturnal dyspnea, nocturnal cough should raise our doubt for heart failure. Adequate haemodynamic monitoring is a must. Further clinical trials are required to improve the management strategies.

**CONSENT**- A proper consent of the patient was taken for writing this case report. Patient had understanding that her initials or details of name, images etc. will not be disclosed but anonymity cannot be guaranteed.

**REFERENCES**