

## Peripartum Cardiomyopathy - A Case Report

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**Abstract:** Peripartum cardiomyopathy is a unusual form of dilated cardiomyopathy with unknown etiology, that can be fatal in young women but with prompt diagnosis and intensive supportive measures can successfully be treated with good longterm prognosis in 90% cases. We report a previously asymptomatic women with no risk factor presented with pulmonary odema on day three of cesarean section who is now under periodic ECHO follow up to assess the recovery in cardiac function.

**Case Report:** 28 Yrs primigravida married for two years, GDM on meal plan underwent cesarean section at 40 weeks for fetal distress. Intrapartum and initial postpartum period uneventful. On third postoperative day patient developed acute breathlessness with bilateral extensive basal crepts. ECHO picture suggestive of peripartum cardiomyopathy. Patient shifted to ICU and treated with respiratory support, Inotropes, Diuretics and other supportive measures. Patient was symptomatically better on fifth postoperative day and discharged at request on fourteenth postoperative day explaining the need for regular follow up and the high risk of recurrence of peripartum cardiomyopathy in subsequent pregnancies.

**Discussion:** Though peripartum cardiomyopathy is relatively a rare disease (0.1% of pregnancies) it can lead to devastating consequences with overall morbidity mortality rates as high as 5 to 32%. The diagnosis of peripartum cardiomyopathy is challenging since most women in last month of normal pregnancy or soon after delivery experience dyspnoea, fatigue and pedal odema, (as in our case).

**Conclusion:** Hence the treating physician should have high index of suspicion and consider it when managing dyspneic patients to expedite medical treatment for this potentially lethal condition.

**Keywords:** Echocardiography, Heart failure, Peripartum cardiomyopathy

### I. Introduction

Peripartum cardiomyopathy is a unusual form of dilated cardiomyopathy with unknown etiology[1], that can be fatal in young women but with prompt diagnosis and intensive supportive measures can successfully be treated with good long term prognosis in 90% cases[2]. The diagnostic criteria of peripartum cardiomyopathy: a) development of cardiac failure in the last month of pregnancy or within 5 months after delivery, b) absence of an identifiable cause for the cardiac failure, c) absence of recognizable heart disease prior to the last month of pregnancy d) left ventricular systolic dysfunction by classic echocardiographic criteria such as depressed ejection fraction or fractional shortening along with a dilated left ventricle. [3] We report a previously asymptomatic women with no risk factor presented with pulmonary odema on day three of cesarean section who is now under periodic ECHO follow up to assess the recovery in cardiac function.

### II. Case Report

Mrs.x, 28 yrs old, married for two years, Primi, GDM on meal plan, came to us for safe confinement. Booked and immunized outside. First visit to SreeBalaji Medical College & Hospital was at 40 weeks. Menstrual H/O- Age at menarche-14yrs, regular cycles, 3/30days, not associated with clots & pains. Marital H/O: Married for two years, Non consanguineous marriage. Obstetric H/O: 1<sup>st</sup> Trimester: patient was started on Tab Susten which was taken till 34 weeks, Rest of the trimester uneventful. 2<sup>nd</sup> Trimester: OGCT=155mg/dl, Therefore patient was started on meal plan, Rest of the trimester Uneventful. 3<sup>rd</sup> Trimester: h/o Tab Susten was taken till 34 weeks, Rest of the trimester uneventful. Past H/O: Nil significant. Personal H/O: Normal bladder & bowel habits. Family H/O: Nil significant. On examination: Gc Fair, afebrile, not pale, B/L pitting pedal odema+, no cyanosis, not icteric, no clubbing CVS: S1S2 + RS:NVBS +, P/A- Uterus Term, Not Acting, head unengaged, FHS- Good, P/V-Cx mid position, Ext OS patulous, IntOS admits two finger, Membranes present, vertex at brim can be pushed down, pelvis adequate. Investigations: Haemoglobin-10.8gms, Urine albumin & sugars -Nil OGCT =155mg/dl, FBS-75mg/dl, PPBS-119mg/dl, HbA1c-5.5%, Serology-negative, TSH 2.87uIU/ml Blood Group-B positive, USG on 26/06/2015- SLIUG GA= 38-39 wks, AFI=7-8cm, placenta posterior grade III, FL-7.6cm, EFW-3.59 kg. Cervix prime Induction was done on 26/06/2015 at 5.00pm as patient was on her due date with oligohydramnios. After 6hrs of induction, patient spontaneously ruptured her membranes. P/V-Cx 50% effaced, Os 2 cm dilated, membranes absent, vertex at -3

station, moderate meconium stained liquor draining pv .Patient was taken up for emergency LSCS in view of Meconium stained liquor& fetal distress.Patient delivered an alive male baby on 26/06/2015 at 11.50pm with B.wt 2.8kg with goodapgar 8/10,9/10.On 3<sup>rd</sup> POD at 9.35am Patient c/o acute breathlessness.O/E- patient dyspneic,Tachypneic, mild pallor+ , B/L pedal odema+ CVS:S1S2+ RS: B/L coarse extensive crepitations+,R.R-40/min,P.R-140/min,B.P-170/130mmHg, Spo2= 60-70 % in room air,PATIENT WAS SHIFTED TO ICU FOR FURTHER MANAGEMENT,patient was started on Inj.Lasix60mg I.V stat,Inj.Morphine 5mg I.V given,ECHO shows features suggestive of peripartumcardiomyopathy with moderate to severe LV dysfunction, [FIGURE -1,2,3] ECG shows Sinus Tachycardia,Chest X-ray: B/L homogenous opacity more on right side Patient was on NIPPV with Fio2 0.5 &Cpap8/15mmHg,patient was treated with the following drugs: Inj.Lasix 3mg/hr infusion, Tab.Lanoxin0.25mg ½ OD,Tab.Flavedon MR 35mg BD,Tab.Neurokind LC BD,Tab.Ivabrad5mg TDS, Tab.Envas 2.5mg ½ OD,Along with Inj.Taxim1gm I.V BD as post operative antibiotics . Patient was symptomatically better &was shifted back to ward from ICU on 5<sup>th</sup> POD . She was on the following medications ,and she was covered with Inj.Heparin5000 units S/C BD for 5 days. Fluids were restricted to 800ml/day. She was given Duolin Neb 8<sup>th</sup>hrly. She was on O2with nasal prongs 2to4litres(sos).Tab.Lasix 40mg 1 OD, On (7<sup>th</sup> POD),Tab.Envas was stopped &Tab.Metoprolol 25mg ½ BD was given.Tab.Lasix40mg ½- ½ - 0, Tab.Ivabrad 5mg reduced to BD dose. On (10<sup>th</sup> POD)-fluids restricted to 1.5L/day, Tab.Lasix40mg ½-0-0, alternate suture removal was done . On (11<sup>th</sup> POD)-complete suture removal was done .On (14<sup>th</sup> POD)-patient was discharged at request.Patient was adviced to do repeat ECHO after one week. Patient was adviced to continue the following drugs on discharge, Tab.Metoprolol 25mg 1/2 BD, Tab.Lanoxine 0.25mg ½ OD,Tab.Lasix 40mg ½ OD,Tab.Enalapril 2.5mg ½ BD.

**DEPARTMENT OF CARDIOLOGY**

**ECHO CARDIOGRAPHY REPORT**

Aorta : 3.5cms  
Left Atrium : 3.3cms

LIVDD	3.83cms	EDV	63.09ml
LVIDS	3.27cms	ESV	43.30ml
IVSD	1.11cms	EF	31.00%
LVPWD	1.11cms	FS	14.00%
RVID	cms	SV	19.79ml

**B - MODE**

Mitral valve normal in thickness and mobility.  
Aortic valve tricuspid,no systolic doming.  
pulmonary valve normal in morphology.No evidence of PHT.  
Tricuspid valve normal.  
RA,RV normal in dimensions.

**GLOBAL HYPOKINESIA OF LV WITH PRESERVED WALL THICKNESS.**

Ascending aorta appears normal,no evidence of CoA.  
Pulmonary Artery normal in dimensions.  
Septae IAS / IVS : Intact  
Venous drainage : Normal.  
**MINIMAL PERICARDIAL EFFUSION.**  
No clot  
No Vegetation

FIGURE – 1 Echo report suggestive of LV dysfunction

**COLOR FLOW AND DOPPLER STUDY**

**Mitral Flow**

E-Wave: 0.17M/sec  
A-Wave: 0.54M/sec  
**MITRAL REGURGITATION (MILD).**

**Tricuspid Flow**

TR PEAK : 278.0 CM/S.  
TR PG : 30.9MMHG.  
**TRICUSPID REGURGITATION (MILD).**

**Aortic Flow** : 0.65M /sec.

**Pulmonary Flow**: 0.52M /sec .

**IMPRESSION:**  
**GLOBAL HYPOKINESIA OF LV WITH PRESERVED WALL THICKNESS.**  
**MODERATE TO SEVERE LV SYSTOLIC DYSFUNCTION.**  
**GRADE I DIASTOLIC DYSFUNCTION.**  
**MITRAL REGURGITATION (MILD).**  
**TRICUSPID REGURGITATION (MILD).**  
**NO PULMONARY HYPERTENSION.**  
**MINIMAL PERICARDIAL EFFUSION.**

FIGURE – 2 Supportive report of peripartum cardiomyopathy

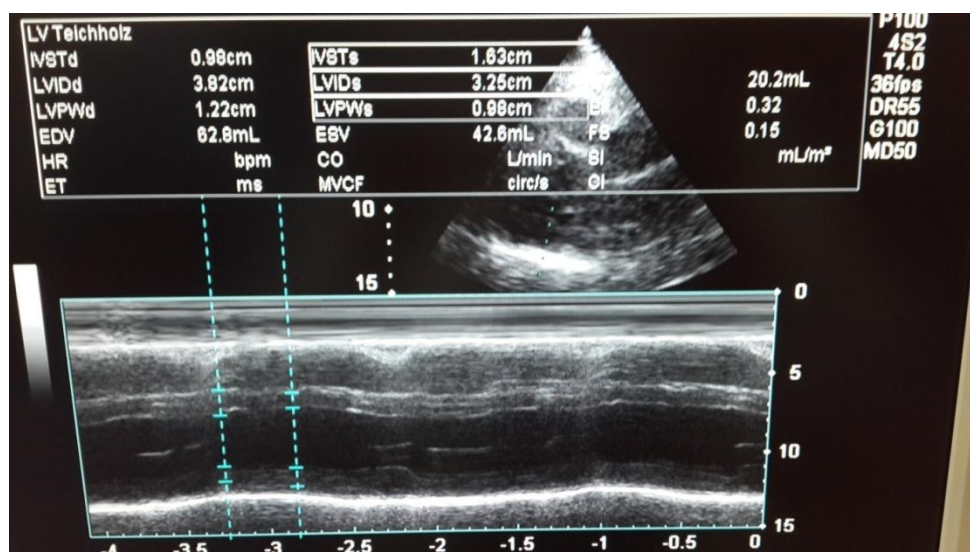


FIGURE- 3 Echo picture of peripartum cardiomyopathy with moderate to severe LV Dysfunction

### III. Discussion

Though peripartum cardiomyopathy is relatively a rare disease (0.1% of pregnancies) it can lead to devastating consequences with overall morbidity mortality rates as high as 5 to 32%. It is estimated that the incidence of peripartum cardiomyopathy is between 1 in 2500 to 1 in 15,000 live births.[3] Etiology remains unknown other potential causes a) viral myocarditis b) cardiovascular stress of pregnancy c) inflammatory response in pregnancy – elevation of TNF Alpha & IL-6 , d) pathologic autoimmune response to fetal cells that lodge in the maternal circulation & cardiac tissue. e) nutritional deficiencies - selenium [3] . Risk of Peripartum cardiomyopathy can occur in woman ( age of parity ) either young or elderly gravida , number of pregnancies , multiple pregnancy , pre-eclampsia , gestational hypertension , oral tocolytic therapy like beta adrenergic agonists. [1] . Symptoms usually include one or more of the following: orthopnea (difficulty breathing while lying flat), dyspnea(shortness of breath on exertion), pitting edema in lower extremities (swelling), cough, frequent night-time urination, excessive weight gain during the last month of pregnancy (1-2+ kg/week; two to four or more pounds per week), palpitations(sensation of racing heart-rate, skipping beats, long pauses between beats, or fluttering), and chest pain.[5] The shortness of breath is often described by patients as the inability to take a deep or full breath or to get enough air into the lungs. Also, patients often describe the need to prop themselves up over night by using two or more pillows in order to breathe better. These symptoms, swelling, and/or cough may be indications of pulmonary edema(fluid in the lungs) resulting from acute heart failure . Unfortunately, patients and clinicians sometimes dismiss early symptoms because they appear to be typical of normal pregnancy. Yet early detection and treatment are critically important to the patient with Peripartum cardiomyopathy . Delay in diagnosis and treatment are associated with increased morbidity and mortality. For these reasons, it is paramount that clinicians hold a high suspicion of Peripartum cardiomyopathy in any peri- or postpartum patient where unusual or unexplained symptoms or presentations occur . Treatment for Peripartum cardiomyopathy is similar to 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) only after delivery. Diuretics, preferably furosemide, help the body to get rid of excess water weight and also lower blood pressure. ACE-I and B-B improve blood circulation and contribute to the reversal of the immune system dysfunction . If ACE-I is not well tolerated by the patient, it can be replaced by angiotensin receptor blockers (ARB). Hydralazine with nitrates may replace ACE-I in breastfeeding mothers or before delivery ; .If EF is less than 35%, anticoagulation is indicated, as there is a greater risk of developing left ventricular thrombi It is important that the patient receives regular follow-up care including frequent echocardiograms to monitor improvement or the lack. 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. Antiviral therapy, immunoabsorption, intravenous gamma globulin, or other immunomodulation therapy may then be considered accordingly , it is still recommended that both ACE-I and B-B be continued for at least one year after diagnosis. 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 patients improve with treatment. over 50% of patients experience complete recovery of heart function (EF 55% or greater) [6]. Once fully recovered, if there is no subsequent

pregnancy, the possibility of relapse or recurrence of heart failure is minimal. Subsequent pregnancy should be avoided when left ventricular function has not recovered and the EF is lower than 55% , the risk for recurrence of heart failure in recovered Peripartum cardiomyopathy patients as a result of subsequent pregnancy is approximately 21% or better [7]. The chance of relapse may be even smaller for those with normal contractile reserve as demonstrated by stress echocardiography [8]. 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.

#### **IV. Conclusion**

Though peripartum cardiomyopathy is relatively a rare disease (0.1% of pregnancies) it can lead to devastating consequences with overall morbidity mortality rates as high as 5 to 32% . The diagnosis of peripartum cardiomyopathy is challenging since most women in last month of normal pregnancy or soon after delivery experience dyspnoea , fatigue and pedal odema , [2],(as in our case) . Hence the treating physician should have high index of suspicion and consider it when managing dyspneic patients to expedite medical treatment for this potentially lethal condition . [4]

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