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## Case report

# Peripartum Cardiomyopathy: An Unforeseen Catastrophe

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#### **ABSTRACT**

Peripartum cardiomyopathy is a form of congestive cardiac failure which is intimately related to pregnancy. The incidence is 1 in 2500-15000 live births. The patient may become symptomatic in the last month of pregnancy and the signs and symptoms may persist upto 5 months after delivery. 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 (blockage of a blood vessel by a blood clot), and even sudden cardiac death. The cause of PPCM is unknown. Here, we report a case of a 25 year old female, primigravida who presented to the HIMS emergency with complaints of amenorrhoea since 9 months and labour pains since 1 day. Her LMP was 24.10.13 and she had completed 38 weeks and 2 days on the day of admission. On examination, the patient was conscious, pulse rate was 102/min, BP was 120/80 mm hg and she was afebrile. There were petechiae and ecchymotic spots over the skin and gross pedal edema was seen. On per abdomen examination, FH was term size, uterus was irritable, cephalic, FHS was present and regular. On per vaginal examination, os was 1.5 cm dilated, 40% effaced, with vertex at -3 station. Augmentation of labour was done by oxytocin infusion. The patient had to be taken up for emergency LSCS due to meconium stained liquor, delivering an alive male baby .The patient was kept in ICU care and was managed on ionotropes and cardiotonic drugs. 16 units Platelets, 8 units FFP's and 6 units PRBC's were transfused during the treatment. The patient was discharged in satisfactory condition on cardio-protective drugs.

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## Introduction

Peripartum cardiomyopathy is defined as the onset of acute heart failure without demonstrable cause in the last trimester of pregnancy or within the first 5 months after delivery. The incidence is 1 in 2500 - 5000 live births (1).

The signs and symptoms may appear in the last trimester of pregnancy and may last upto 5 months after delivery. The risk factors are advanced maternal age, multi-parity, multifetal gestation, obesity and black race.

The Demakis and Rahimtoola diagnostic criteria for peripartum cardiomayopathy says that development of heart failure in the last month of pregnancy or upto 5 months post partum, absence of an unidentifiable cause for the cardiac failure, absence of recognisable heart disease before the last month of pregnancy, tachycardia, dyspnoea on exertion, fatigue, ankle edema, peripheral edema, embolic phenomena, atypical chest pains, haemoptysis, pulmonary rales, chest x-ray showing enlarged heart and pulmonary vascular redistribution and echocardiography showing enlargement of all chambers of heart, predominantly the left ventricle with left ventricular dysfunction(2).

### Case report

A 25 year old female , primigravida presented to the emergency of HIMS with chief complaints of amenorrhoea since 9 months and labour pains since 1 day. Her last menstrual period was on 24thOctober, 2012. The patient had completed 38 weeks and 2 days on the date of admission. The previous menstrual cycles were regular with normal flow, lasting for 4-5 days, 30 days cycle.

On general physical examination, the general condition was fair, well oriented to time, place and person. Blood pressure 120/80mmHg,pulse rate was 102/min,and respiratory rate was 18/min. She was afebrile at that time. There was no pallor/clubbing/icterus/jaundice/lymphadenopathy, peripheral oedema was seen. Thyroid examination and breast examination was normal. Cardiovascular system, respiratory system, central nervous system were normal. On per abdomen examination, fundal height was term size, uterus was irritable, cephalic, FHS was present and regular. On per vaginal examination, cervix was 1.5 cm dilated, 40% effaced and vertex was at -3 station.

On admission, the patient was investigated. The counts were, Hb was 3.9 gm%, platelets were 80,000.A high risk consent was taken, and the prognosis of the mother and the fetus was explained to the attendants. Augmentation of labour was done by oxytocin Infusion. During augmentation, 4 units platelets, 4 units FFP's and

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1 unit PRBC were transfused. Preoperatively, the counts were Hb was  $8.8~\rm gm\%$ , platelets were  $40,\!000$ . The patient had to be taken up for emergency LSCS due to appearance of meconium stained liquor.

Per-operatively, bleeding was towards the higher side, the uterus was relaxed which led to the decision of b/l uterine artery ligation. An alive male baby was delivered by vertex, weighing 2.7 kg, at 11.36 am. The baby cried immediately after birth. The Apgar score was 7/8/9.

Post-operatively, the peripheral pulses of the patient could not be felt and the patient developed hypotension (BP 80/40 mm hg) with a heart rate of 180/min. The patient was shifted to the ICU and was put on SIMV mode of ventillation. Ionotropes were started and titrated according to the patient's condition. The patient was shifted to CPAP mode of ventillation on POD-1. The counts subsequently increased to Hb rising to 11.3 gm %, total leucocytes were 24,400 and the platelets were 45,000 over the next two days. On POD-2, the patient developed b/l pneumonia with arrythmias. The chest x ray showed cardiomegaly with alveolar edema and ECG showed nonspecific ST and T wave changes, atrial or ventricular arrhythmias and conduction defects. A 2D ECHO was done which showed normal left ventricle size but severe LV systolic dysfunction, global hypokinesia of left ventricle, grade 3 diastolic dysfunction with an ejection fraction of 18-20 %. The patient developed disco-ordinate breathing. There was a fall in the counts showing Hb 8.1 gm %, , TLC of 11,300 and platelets < 20,000.

She was shifted back to SIMV mode of ventillation. The condition of the patient improved over the next 3 days and she was extubated on POD-6 and shifted to BIPAP. A total of 6 units of platelets, 6 units of PRBC's and 4 units of FFP's were transfused. Thereafter, the blood counts of the patient improved showing Hb 8.8 gm %, a total leucocyte count of 7300 and platelets 1,29,000. Catheter removal was done on POD-7. Stitch removal was done on POD-10. The patient was discharged in satisfactory condition.

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#### Discussion

50-60% patients show complete or near complete recovery within the first 6 months postpartum. There is an initial high risk period with mortality of 25-50% in the first 3 months postpartum. Patients with persistent cardiomegaly at 6 months have a reported mortality of 85% at 5 years(3). Subsequent pregnancies are associated with relapses and high risk for maternal morbidity and mortality and should be discouraged in women with PPCM who have persistent cardiac dysfunction.

Diagnosing peripartum cardiomyopathy in a mild case is by typical symptoms such as swelling in the feet and legs, and some shortness of breath can be similar to the symptoms of the third trimester of a normal pregnancy, so these symptoms may go undiagnosed. The patient may then go on to recover without further medical attention. On the other hand, severe cardiomyopathy can reveal itself if a patient becomes very short of breath and has swollen feet well after delivery as was a case in our patient. When the heart doesn't pump well, fluid can accumulate in the body, most noticeably in the lungs and the feet. An echocardiogram can detect the cardiomyopathy by showing the diminished functioning of the heart.

Diagnostic echocardiographic criteria include left ventricular ejection fraction <0.45 or M-mode fractional shortening <30% (or both) and end-diastolic dimension >2.7 cm/m2(4).

Treatment for peripartum cardiomyopathy includes conventional pharmacologic heart-failure therapies, principally diuretics, angiotensin converting enzyme inhibitors, vasodilators, digoxin,  $\beta$ -blockers, anticoagulants(5). The objective of peripartum cardiomyopathy treatment is to keep extra fluid from accumulating in the lungs and to help the heart recover as fully as possible. Mechanical support and transplantation might be necessary in severe cases as in the case reported.

The question of having additional children usually hinges on to what degree the mother has recovered from her peripartum cardiomyopathy.

If the heart does not completely recover its work capacity, another pregnancy is generally not recommended. While there is no direct risk to the baby, going through an additional pregnancy with an abnormally functioning heart can cause additional heart damage for the mother, which could in turn harm the developing foetus.

If the heart has completely recovered from the previous pregnancy, an additional pregnancy can be attempted if the heart is periodically monitored with echocardiograms and stress tests. Echocardiograms check how the heart functions at rest and stress tests measure how the heart works under strain.

There are several kinds of medications a physician can prescribe to treat symptoms. These medications include: ACE (angiotensin converting enzyme) inhibitors which help the heart use the strength that it has to work more efficiently. Beta blockers cause the heart to beat more slowly so that it has a greater chance to recover and diuretics help reduce fluid retention.

For women who breastfeed, there are medications in each of the above classes that are safer for breastfeeding than the most commonly prescribed regimens, but these are equally effective for treatment. The prognosis is best when peripartum cardiomyopathy is diagnosed and treated early. Fortunately,

despite a high risk of recurrence in subsequent pregnancies, many patients with peripartum cardiomyopathy recover within 3 to 6 months of disease onset.

A large multicenter, prospective randomized trial is currently needed to evaluate the incidence, the pathophysiology (which would include setting up a bio-repository for genetic and translational studies) and the current therapies for peripartum cardiomyopathy. Current recommended therapy remains the standard pharmacologic treatment for heart failure due to systolic dysfunction, however, outcomes on conventional therapy vary widely.

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