

# CASE REPORT

## Acute Peripartum Cardiomyopathy after Caesarean Section: A Case Report

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### ABSTRACT:

Peripartum cardiomyopathy is an idiopathic cardiac muscle disorder which presents as heart failure secondary to left ventricular dysfunction in the last month of pregnancy or during 5 months postpartum. It is a major cause of maternal morbidity and mortality. We had a 26 years multigravida with uneventful term pregnancy delivered by caesarean section who developed cardiopulmonary failure within 8 hours of delivery. It was suspected to be either pulmonary embolism or peripartum cardiomyopathy. Echocardiography findings were suggestive of PPCM (ejection fraction <20%). She was treated with diuretics, LMW Heparin, inotropic support and ventilator support. She showed recovered cardiac function on repeat 2Decho.

Key words: *PPCM*-Peripartum Cardiomyopathy, HF-Heart Failure, Puerperium, Pregnancy, Caesarean Section, ICU management.

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**INTRODUCTION:**  
Heart failure (HF) in the setting of pregnancy has been recognized as far back as the 18<sup>th</sup> century. It was not until 1937, however, that a definitive form of postpartum cardiomyopathy was first described in a case series report by Gouley et al.<sup>1,2</sup> The report described 7 women who developed severe, clinical HF in the late months of pregnancy with histologic examination showing widespread and severe focal inflammatory reaction, often with necrosis and followed by fibrosis. In 1971, Demakis et al. described a case series of 27 women with pregnancy-associated cardiomyopathy (PACM) including those who presented with HF in the peripartum period, following which the term “peripartum cardiomyopathy” (PPCM) was first used.<sup>[3]</sup> The disease remains relatively rare, of unclear etiology, and challenging to recognize and diagnose early. The following modified criteria are used to define PPCM:

1. Development of heart failure during last trimester of pregnancy or first six months post-partum.
2. The absence of any identifiable cause for cardiac failure.
3. The absence of any recognizable heart disease prior to last trimester of pregnancy.
4. Echocardiographic criteria- Demonstrable echocardiographic proof of left ventricular systolic dysfunction. Ejection fraction less than 45%, left ventricular fractional shortening less than 30% or left

ventricular end-diastolic dimension >2.7cm/m square of body surface area.

### CASE REPORT:

A 26 year G2P1L1 with term pregnancy with previous LSCS presented to our labor room with complaints of pain abdomen. She was 40 weeks of gestation at the time of admission. On General examination her pulse was 86 bpm, BP 110/70 mmHg and all routine investigations were normal. She was delivered by caesarean section I/V/O previous LSCS with cephalopelvic disproportion. After 8 hrs of surgery patient complained of sudden onset of breathlessness, palpitations and uneasiness. The patient was shifted to ICU. On examination, she had tachycardia (110/min) and tachypnoea (24/min), BP was 100/74 mmHg and Spo2 was 80%. On auscultation bilateral fine crepitations were present. She was started on O<sub>2</sub> by mask at 6litre/min. Initially Inj. Hydrocortisone 200mg IV stat, Inj. Deriphyllin 1ampouleiv stat were given. Nebulization given with duolin and budecort. ECG showed poor progression of R wave. The Patient's condition deteriorated with PR: 160/ min, BP: 80/50 mm Hg, CVS: S1 S2 +, RS: B/L rhonchi and crepts +, SPO2: 88% with ABG suggestive of metabolic acidosis, CXR showing pulmonary edema. The patient was shifted on non-invasive positive pressure ventilation with BiPAP. Central line access of IJV was secured. Suspecting diagnosis of pulmonary embolism or peripartum cardiomyopathy, Inj Heparin, Higher antibiotics (Inj. Levofloxacin 500mg IV OD and inj. Meropenem 1 gm

IV BD, Inj. Clarithromycin 500mg BD) Inotropic support (Dopamine infusion 5ml/hr) was started. After 10 hours, Pt developed labored breathing and tachypnoea. She was intubated and shifted on mechanical ventilator (PCV MODE). A few minutes later, the patient developed ventricular tachycardia and BP was not recordable. Vitals: HR: 200/min, BP: not recordable. Synchronised Cardioversion with 30 J and 50 J Shocks given. Post cardioversion vitals were HR: 190/min, BP: 100(systolic) inotropic support started Noradrenaline 8ml/hr and Dopamine 6 ml/hr. 2Decho revealed severe left ventricular dysfunction with left ventricular dilatation and hypokinesia with LV ejection fraction <25%. As echo findings confirmed PPCM, Digoxin (0.5mg stat f/b 0.25mg od), Inj. Diltiazem 1 amp in 50ml NS (2ml/hr) and Inj. Thiamine were started. Gradually patient's condition improved, RT feeds were started and she was shifted on T-piece on POD3. Repeat 2Decho done on POD5 showed- mild global hypokinesia, moderate LV dysfunction and EF= 39%, Trivial MR The patient was extubated on POD8 and all inotropes were stopped on POD12. She was shifted to the ward on POD13. Her condition improved furthermore and her final 2Decho was done on POD17 that showed good LV contractility with EF 55%.

**DISCUSSION:**

The incidence of PPCM in India is 1:1734<sup>(4)</sup>. The overall incidence in the USA is around 1:3200 deliveries with the values in different studies ranging from 1:1149 to 1:4350 live births.<sup>(5, 6, 7, 8, 9)</sup> The varying incidence of PPCM can be attributed to multiple epidemiological factors e.g. environmental, ecological, cultural, puerperal and post puerperal practices along with diagnostic criteria and reporting standards.

Various causes have been implicated in PPCM e.g. viral myocarditis, autoimmune phenomena, genetic mutations altering prolactin production<sup>(10)</sup>, nutritional disorders and idiopathy, yet the exact cause remains unknown<sup>(11)</sup>

Risk factors associated with PPCM are increasing maternal age, multifetal gestation, multiparity, and pregnancy-induced hypertension<sup>(11)</sup> Patients with PPCM present with classical signs and symptoms of Heart failure i.e. dyspnoea, fatigue and edema that simulate a picture of pulmonary embolism<sup>(12)</sup> and ischemic heart disease as well. Therefore the diagnosis is delayed in many cases leading to increasing maternal mortality rates that can be as high as 20-50%<sup>(13)</sup>

PPCM patients are treated with fluid and salt restriction, beta blockers, diuretics and digoxin as any other congestive heart failure case. ARB and ACE are not

given as they are contraindicated in pregnancy<sup>(14)</sup>. Prognosis is poor, only around 50% patient's cardiac function returns back to normal. With every subsequent pregnancy, the left ventricular size increases and chances of recurrence are very high (30%).<sup>(15)</sup>

**CONCLUSION:**

PPCM should be considered as a differential diagnosis in all puerperal patients with dyspnoea for early intervention and better outcome.

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