

Peripartum Cardiomyopathy After Caesarean Section in the Post-Operative Period: A Report of Two Cases with Review of Literature

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ABSTRACT

Peripartum cardiomyopathy presenting as heart failure in patients in the post-partum period is a diagnosis of exclusion. We report two cases presenting in the post-operative period almost eight hours after Caesarean section. The diagnosis and subsequent management of this entity in the post-operative period requires a clinician to be aware of the clinical presentation of PPCM. One patient recovered well but the other one succumbed to the illness.

Key words: Peripartum Cardiomyopathy, Caesarean Delivery, Heart Failure.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is defined as heart failure in the last month of pregnancy upto five months post-partum, in the absence of an identifiable cause of heart failure and in the absence of evidence of heart failure before the last month of pregnancy. It is supported with echocardiographic (2D-Echo) evidence of Left ventricular systolic dysfunction with ejection fraction (EF) <45%, reduced Fractional shortening (FS) <30% and increased Left ventricular end diastolic dimensions (LVIDed) >2.7 cm/m².¹

This article aims to alert obstetricians and anaesthesiologists who as perioperative physicians need to diagnose PPCM in the postoperative period after caesarean section (LSCS) as early diagnosis and institution of aggressive treatment measures can lead to improved outcome.

CASE 1


A 26 year old female, second gravida, who had an uncomplicated full term pregnancy with previous LSCS was posted for emergency LSCS for scar tenderness with a history of pre-eclampsia in the first pregnancy. General examination revealed height (Ht) 146cm, weight (Wt) 46kg, Pulse 70/minute and Blood pressure (BP) 120/80 mmHg in supine position. Systemic examination was normal. We proceeded for the surgery under sub-arachnoid block. Patient was pre-medicated with Inj. Metoclopramide 10mg intravenous (IV) and Inj. Ranitidine 50mg IV and pre-loaded with 500ml of Ringer Lactate IV. Baseline readings of vitals were recorded and a subarachnoid block was established at L2-L3 space in sitting position with Inj. Bupivacaine (0.5% heavy) 12mg to achieve a sensory level of T6. Five minutes later, a male baby weighing 2.4 kg, was delivered, with APGAR

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score of 8/10 at 1 minute (min) and 10/10 at 5 min. Inj. Midazolam 1mg IV, Inj. Pentazocin 15mg IV, Inj. Oxytocin 10 IU IV infusion over 30 min and Inj. Methergin 0.2mg IM was given. Rest of surgery was uneventful. In the post-operative period, 8 hours later she developed breathlessness. Respiratory rate (RR) was 30/min, Pulse 110/min, BP 100/70mmHg, SPO₂ 80% and auscultation of chest revealed bilateral crepts, uterus was well retracted, no increase in abdominal girth and no excessive per vaginum bleeding. By the time she was shifted to ICU, pulse was 180/min, BP had dropped to 80/50mm Hg and SPO₂ with O₂ by mask @ 6 lit/min was 88%. She was intubated and put on volume control ventilation with tidal volume 500ml and rate 12/min. A differential diagnosis of pulmonary embolism and PPCM was considered. ECG, Chest Radiograph and bedside 2D Echo was done. Echo showed severe LV Dysfunction with an EF-25%, FS%-12.5 and LVIDed-5.35cm which confirmed the diagnosis of PPCM. Central venous access was established through Right (Rt) Internal Jugular Vein (IJV) and CVP was 12 cm H₂O. Post central line chest X-Ray was normal. She was started on Inj. Dobutamine infusion 5mcg/kg/min, Inj. Diltiazem infusion 5mg/hour, Tab. Digoxin 0.5mg through Ryle's tube and low molecular weight heparin 0.6 ml subcutaneous 12 hourly. Inj. Fentanyl 50mcg/hr and Inj. Midazolam 1mg/hr infusion was used for sedation. Total IV infusions were restricted to 1000ml/day. Her vitals gradually returned to normal by Day 3. On the 3rd post-operative day (POD), a Rt sided pneumothorax was noted on Chest X-Ray after which an Intercostal drain (ICD) was inserted in Rt 5th intercostal space in mid axillary line. On POD 7, ICD was removed after lung expansion noted radiographically, inotropic support stopped and extubated on POD 8. Repeat 2D-Echo on POD 13 showed a

recovery in systolic function with EF-55%, and improvement in FS-28.4% and LVIDed- 3.90 cm. She was continued on treatment with frusemide, dioxin and ACE inhibitors and asked to follow up regularly with the physician.

CASE 2

A 21 year old female, Primigravida with 32.4 weeks of gestation, twin pregnancy with severe Pre-eclampsia detected 3 days earlier on treatment with Tab.Labetalol 100mg TID was posted for emergency LSCS for severe Pre-eclampsia with impending signs of eclampsia. She was conscious, oriented, Ht- 160cm, Wt-78kg, bilateral pedal edema, Pulse 110/min, BP 160/100 mmHg, RR 18/min. After premedication and noting baseline parameters, a subarachnoid block was placed with 14mg of Inj. Bupivacaine 0.5% (heavy) to achieve a sensory level of T6. A male baby, 1.1kg with an APGAR score of 9/10 at 5min and female baby, 1.6 kg were delivered with APGAR score of 9/10 at 5 min, and shifted to NICU for observation. An infusion of 10 Units Oxytocin was administered over 30 minutes. Surgery was uneventful.

Loading dose of Inj. Magnesium sulphate, Four grams IV and 5 grams Intramuscular in each buttock followed by IV infusion 1gram/hour for 10 hours (total dose 24 grams) was given.

12 hours after surgery, patient developed breathlessness, Pulse-134/min, BP-90/60 mmHg, RR-35/min, SPO₂- 80% on room air with bilateral crepts on auscultation. The patient was intubated and Volume control ventilation instituted. We started Inj. Fentanyl (1mcg/kg/hour) and Inj. Midazolam (1mg/hour) infusion. Central Line was inserted for CVP monitoring in Rt. IJV which was 14 cm of H₂O. 2D-Echo showed Global hypokinesia with EF- 41%, LVIDed- 5.45 cm, FS- 20.7%, confirming the diagnosis of PPCM with LV dysfunction. The patient was treated with Inj.Frusemide 20 mg IV QID, Tab. digoxin 0.25 mg OD through Ryle's tube, Low molecular weight heparin and Inj.Dobutamine infusion 7mcg/kg/min. BP improved 110/ 70mmHg and urine output 1000 ml/day with vasopressor support. She had two early morning intermittent episodes of bradycardia and hypoxia which responded to Inj.Atropine 0.6mg IV and adjustment of ventilatory parameters. On POD 3, patient had a sudden episode of severe bradycardia Pulse-30/min, BP- 60mmHg, SpO₂- 70%. The endotracheal tube was checked which was in situ and patent. Temporary pacing and Noradrenaline infusion was instituted. We did not have facilities for Intra-aortic balloon pump. However patient developed asystole after 20 min. Standard CPR protocol continued for next 45 minutes, however patient could not be revived.

DISCUSSION

Clinical diagnosis of PPCM requires a high index of suspicion in pregnancy as the symptoms of normal pregnancy mimic those of heart failure like fatigue, dyspnoea and pedal edema.² Orthopnea and paroxysmal nocturnal dyspnoea should alert the physician to look for signs of heart failure like new regurgitant murmurs, pulmonary rales, hepatomegaly and elevated jugular venous pressures.³ Both our patients presented initially with dyspnoea. Though, PPCM is postulated to have various etiologies like auto-immune mediated, viral myocarditis or a vascular element similar to pre eclampsia, there is no exact supporting evidence for it. Risk factors for the development of PPCM include pre-eclampsia, hypertensive disorders of pregnancy, multiparity and multiple gestation.⁴ The authors observed that the prevalence of pre

eclampsia quadrupled in the study in PPCM cases as compared to general population.⁴ The secretion of potent anti-angiogenic factors in circulation is postulated to be the etiology of both pre-eclampsia and PPCM. Our second case had two risk factors of pre-eclampsia and multiple gestation. However PPCM may occur in the absence of pre-eclampsia in more than 80% cases⁴ as was seen in our first case report. Treatment of heart failure includes measures to reduce pre-load, afterload and improve cardiac contractility using diuretics, nitrates and digoxin. Cardiogenic shock requires the use of inotropes, intensive haemodynamic monitoring and non-invasive or invasive ventilation.³ ACE Inhibitors can be used in the postpartum period and beta blockers can be added once the condition stabilises.²

In a case series on PPCM, the authors noted that six out of sixteen patients developed PPCM in the postnatal period.⁵ Unexplained cardiac arrest has occurred after administration of spinal anaesthesia in undiagnosed PPCM.⁶ Jae Jun Lee reports a case of PPCM occurring in the recovery room after caesarean section conducted under combined spinal epidural anaesthesia.⁷ However though symptomatic treatment was instituted immediately, a diagnosis of PPCM was established on echocardiography findings done on POD2 by the authors. Both our cases had a late presentation nearly 8 hours after caesarean section in the ward and echocardiographic evidence of PPCM was obtained within few hours. In spite of aggressive initial therapy, one patient succumbed. Though there are higher chances for spontaneous recovery in patients of PPCM (23-54%) than from other forms of cardiomyopathies^{8,9}, mortality for PPCM ranges from 1.4 to 30%.²

Fett et al did not find any statistical difference in the left ventricular echocardiographic findings between deceased patients and those who survived (mean end-diastolic dimension (6.2 vs 5.8 cm; P=.08), ejection fraction (22% vs 25%; P=.12), and fractional shortening (16% vs 15%; P=.46).^[8Fett] Another study however noted that, a fractional shortening value less than 20% and a left ventricular end diastolic dimension 6 cm or greater at the time of diagnosis was associated with a more than 3-fold higher risk for persistent left ventricular dysfunction.¹⁰

CONCLUSION

Cardiac failure in the post-operative period must be diagnosed and treated promptly and aggressively. The perioperative physician should include PPCM in the differential diagnosis and plan the subsequent management.

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