ANAESTHETIC MANAGEMENT OF PERIPARTUM CARDIOMYOPATHY: A CASE REPORT

Prabhu B. G[1], Shashidhar D. T[2], Harshitha H. Patil[3], Hassaan Muhammed[4]

[1]Professor, Department of Anaesthesiology & Critical Care, J. J. M. Medical College, Davangere.
[2]Post Graduate, Department of Anaesthesiology & Critical Care, J. J. M. Medical College, Davangere.
[3]Post Graduate, Department of Anaesthesiology & Critical Care, J. J. M. Medical College, Davangere.
[4]Post Graduate, Department of Anaesthesiology & Critical Care, J. J. M. Medical College, Davangere.

ABSTRACT

Anaesthetic management for caesarean section of a patient with peripartum cardiomyopathy (PPCM) can be challenging. These patients require analgesia/anaesthesia for normal delivery or caesarean section. In this case report we describe the anaesthetic management of a 20-year-old patient at 37 weeks of gestation, with peripartum cardiomyopathy, heart failure and pulmonary oedema. She was scheduled for emergency caesarean section because of a threat to mother's life and fetal distress. GA was induced with Etomidate and fentanyl safely. No adverse outcome on mother or new born was observed.

KEYWORDS
Peripartum cardiomyopathy, Caesarean section, General Anaesthesia, Etomidate.

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INTRODUCTION: Peripartum cardiomyopathy (PPCM) is an unusual form of dilated cardiomyopathy which manifests as acute heart failure in last trimester of pregnancy or early postpartum period.(1) Its diagnosis is based on exclusion of other identifiable causes.(1,2) Anaesthetic management is directed towards optimisation of myocardial contractility, with maintenance of ventricular preload and afterload. A vigilant monitoring is essential throughout the surgery to avoid any complications like arrhythmias, hypotension, hypoxemia, pulmonary oedema, myocardial infarction, thromboembolism and sudden death.(3-8)

CASE REPORT: A 20-year-old female patient at 37 weeks of gestation presented with signs and symptoms of congestive cardiac failure mainly being generalized oedema since 15 days, chest pain and palpitations 7 days, breathlessness grade 4 since 3 days, persistent cough 2 days and history of generalized weakness and easy fatigability. General physical examination revealed pallor, bilateral pitting pedal oedema, and pulse rate 132 bpm (regular), BP 150/100 mmHg. Systemic examination revealed jugular venous dilation and bilateral basal crepitation. She reported regular antenatal check-ups during 1st and 2nd trimester with no previous history of preeclampsia or any other co-morbid condition. Her previous pregnancy one year back ended in a spontaneous abortion at 2½ months of gestation, check scan was done.

Her ECG showed sinus tachycardia and echocardiogram findings of dilated left ventricle, global LV hypokinesia, mild MR, AR, TR, mild PAH, LV systolic dysfunction (EF 40%). Chest X-ray showed cardiomegaly with basal haziness.

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Corresponding Author:
Dr. Prabhu B. G,
Professor, Department of Anaesthesiology & Critical Care,
JJM Medical College, Davangere-577004.
E-mail: prabhubethur@gmail.com
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She was admitted to the ICU and medically managed on Inj. Furosemide 40mg, T. Labetalol 200mg tid, T. Methyldopa 500mg BD, T. Carvedilol 3.125 mg BD, LMWH 0.4 mg BD.

Her routine blood investigations were Hb 10.7 gm%, TC 13,550 (N-71%, L-20%), PCV 34.4%, platelet count 2.84 lakhs/cumm, and ESR 08mm/hr. Peripheral blood picture revealed – Microcytic hypochromic anaemia with neutrophilic leucocytosis.

Because of worsening maternal condition and foetal distress, the attending obstetrician recommended emergency caesarean section. Patient was transferred to the operating room with severe respiratory distress, orthopnoea and sinus tachycardia (HR 120). Patient was connected with SPO2 probe, NIBP, ECG, temperature probe. Patient was premedicated with Inj. Glycopyrrolate 0.2 mg, Inj. Metoclopramide 10 mg. General anaesthesia was induced with intravenous etomidate 12mg; Inj. Fentanyl 100 µg, and relaxed with succinylcholine 75mg. Patient was intubated using Sellick's manoeuvre (7.5 mm/ET cuffed). The position of the ETT was confirmed by auscultation and Et CO_2 graph and fixed. Anaesthesia was maintained with O_2 + Air mixture + IPPV and intermittent Inj. Vecuronium. Mechanical ventilation consisted of FiO_2 0.7, PEEP 5 mmHg, TV 400 ml, RR 14 breaths/min, peak airway pressure of 32 mmHg. Systolic BP was maintained about 140-150 mmHg and oxygen saturation was maintained between 96-98%. 10 min after induction a term male new born was born (Wt. 2.4 kg) and APGAR score of 8 and 9 in 1st and 5th post-delivery minutes respectively. After the delivery of the baby, an infusion of oxytocin was started with 10 units in 500 ml of normal saline and Inj. Furosemide 20mg IV was given intravenously. Operation lasted for 80 min at the end of the procedure patient was shifted to ICU for ventilator support. Shifting vitals were, HR- 98b/min, BP- 128/70 mmHg, SPO_2- 95%. ABG was with in normal limits. She was weaned off the ventilator and extubated 6 hours later uneventfully. Post extubation, she was conscious, oriented and maintaining

SPO_2 of 96% on facemask and blood pressure of 110/65 mmHg. She was managed with antibiotics, LMWH, loop diuretic and beta-blockers. She was discharged to the ward on the third- postoperative day with stable haemodynamics and is now on regular follow-up after discharge form the hospital.

DISCUSSION: PPCM is characterized with onset of acute congestive heart failure without any demonstrable cause in the last trimester of pregnancy or within the first 6 months after delivery.[9,10] The incidence of PPCM is reported as 1:300 to 1:15000 in the western literature; however, incidence in Indian scenario is lacking due to paucity of data.[10,11]

• Development of heart failure in the last month of pregnancy or within 5 months postpartum. • Absence of an identifiable cause of heart failure. • Absence of recognizable heart disease before the last month of pregnancy. • Left ventricular systolic dysfunction demonstrated by left ventricle ejection fraction of less than 45%, fractional shortening of less than 30%, or both, with or without a left ventricle end-diastolic dimension less than 2.7 cm/m2 of body surface area.
*As defined by Workshop convened by NHLBI and NIH Office of Rare Diseases.[12]
Table 1: Criteria for diagnosis of peripartum cardiomyopathy*

It is usually a diagnosis of exclusion, as one need to exclude differential diagnosis like other types of cardiomyopathies, valvulopathies, pulmonary embolism, infections, metabolic or ischemic causes of myocardial dysfunction which are amenable to therapy.[13,14]

Despite clearly outlined criteria, the diagnosis of PPCM presents a challenge because many normal parturients in the last month of pregnancy experience dyspnoea, fatigue, and pedal oedema and moreover the initial symptoms could mimic mild upper respiratory tract infection. A high index of suspicion of heart failure should be kept in mind for symptoms like paroxysmal nocturnal dyspnoea, orthopnoea, chest pain, persistent cough, new regurgitant murmurs, pulmonary crackles or crepts, elevated jugular venous pressure, hepatomegaly, and postural hypotension reflecting low cardiac output. [15-17]

The aetiology of PPCM remains elusive, however, viral, autoimmune, hemodynamic stress of pregnancy, cytokine-mediated inflammation, Gq-related myocyte apoptosis, oxidative stress-induced Cathepsin D production, selenium deficiency, and idiopathic causes are being hypothesized. Other possible aetiologies include nutritional deficiencies, small-vessel coronary artery disease, excessive salt intake, and peripartum fluid shifts. Maternal age more than 30 years, multiparity, eclampsia, obesity, racial origin (African descent), hypertension, malnutrition, and prolonged tocolysis are predisposing factors.[18-23]

Management is mainly symptomatic and aims to reduce after load and preload and enhance myocardial contractility, reduce arrhythmogenicity; hence, includes bed rest, digitalis, loop diuretics, vasodilators, β-blockers, anticoagulants, and ionotropic support. If supportive treatment fails, cardiac transplantation may be indicated. Early delivery is the major consideration to avoid deleterious effects on parturient either by instrumental vaginal or caesarean section. Close liaison with the patient's cardiologist, intensivist, and obstetrician is essential.[24,25]

Anaesthetic management for caesarean delivery in women with PPCM should incorporate measures to optimally reduce preload and after load while maintaining an increase in myocardial contractility correct choice of anaesthesia and precise titration is crucial for a favourable outcome. Various case reports describe both general anaesthesia and regional anaesthesia techniques like combined spinal epidural anaesthesia (CSE), sequential CSE anaesthesia or epidural anaesthesia or continuous spinal anaesthesia. The challenge in either of these techniques is to avoid myocardial depression, hypovolemia, and prevent any increase in pre- and after load. The use of invasive monitoring is also recommended to guide anaesthetic management, though successful outcomes have been reported with non-invasive monitoring as well. Anticoagulation is indicated as PPCM increases the risk of thromboembolic events.[26-29]

We opted for general anaesthesia (GA), due to the urgent nature of surgery, presence of anticoagulation and severity of PPCM. Tough performing a rapid sequence induction on a patient with compromised cardiac function can be very challenging, a smooth titrated induction and maintenance of anaesthesia obviated the stress of laryngoscopy and intubation and diminished the sympathetic responses. Avoidance of high concentrations of volatile agents, other drugs like atropine, ephedrine, etc. and hypovolemia prevented dramatic cardiac depression and uncontrolled changes in after load and preload. As boluses of oxytocin can cause hypotension and tachycardia, we used infusion of oxytocin. We also avoided the stress of extubation by planning elective ventilation postoperatively followed by gradual weaning and extubation.[30,31]

In elective conditions, a carefully administered regional anaesthetic technique like CSE or sequential epidural has been found to be beneficial as it avoids the stress of GA and produces changes in preload and after load which mimic the goals of hemodynamic variability in these patients thereby improving myocardial performance by reducing LV after load.[6,8] However, a subarachnoid block can induce catastrophic hemodynamic instability secondary to reduction in systemic vascular resistance (SVR) and limited cardiac reserve.

In the course of the disease, cardiac function recovers in around half the women with PPCM, but in those with persistent left ventricular dysfunction even after 6-12 months, a mortality of up to 85% has been reported, indicating irreversibility and is an absolute contraindication for future pregnancies. Those with documented poor recovery might be considered for cardiac transplants.[19]

CONCLUSION: PPCM is a rare disease of unknown cause that affects women in the child bearing years. The diagnosis of PPCM is challenging and that of exclusion which requires a high index of suspicion. Choice of anaesthesia needs to be guided based on the urgency of lower segment caesarean section (LSCS) and severity of PPCM. The primary anaesthetic goal should be to avoid major swings in preload and after load and avoidance of myocardial depression, while alleviating the symptoms of congestive heart failure.

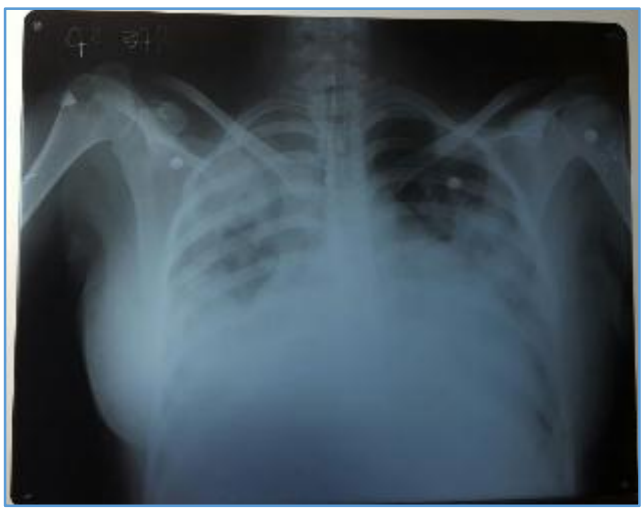
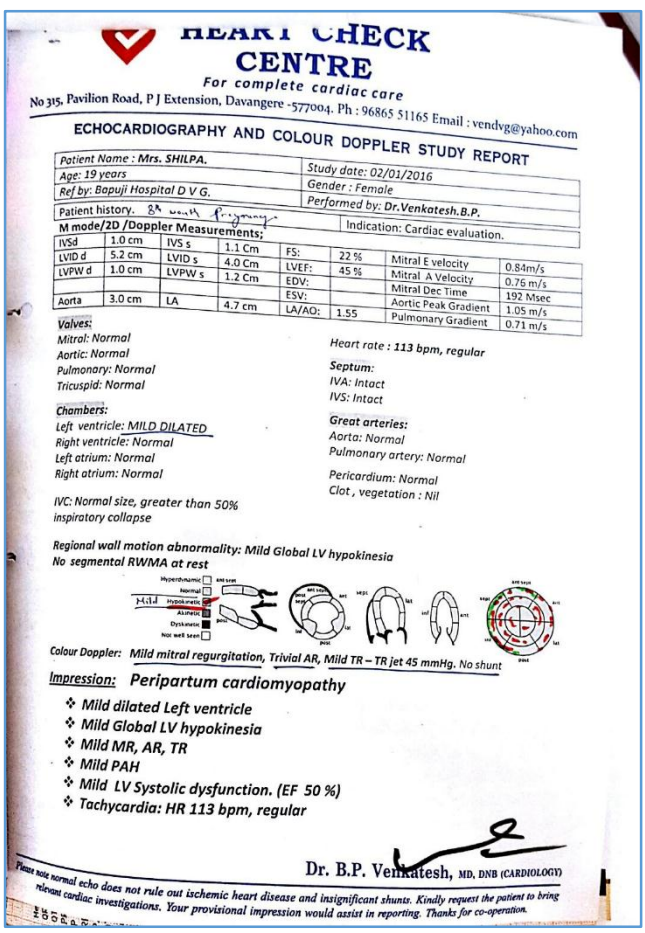


Figure 1



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ECHOCARDIOGRAPHY AND COLOUR DOPPLER STUDY REPORT

Patient Name : Mrs. SHILPA. | Study date: 02/01/2016
Age: 19 years | Gender : Female
Ref by: Bapuji Hospital D V G. | Performed by: Dr.Venkatesh.B.P.
Patient history. [illegible] | Indication: Cardiac evaluation.

M mode/2D /Doppler Measurements;

IVSd	1.0 cm	IVS s	1.1 Cm	FS:	22 %	Mitral E velocity	0.84m/s
LVID d	5.2 cm	LVID s	4.0 Cm	LVEF:	45 %	Mitral A Velocity	0.76 m/s
LVPW d	1.0 cm	LVPW s	1.2 Cm	EDV:		Mitral Dec Time	192 Msec
				ESV:		Aortic Peak Gradient	1.05 m/s
Aorta	3.0 cm	LA	4.7 cm	LA/AO:	1.55	Pulmonary Gradient	0.71 m/s

Valves:
Mitral: Normal
Aortic: Normal
Pulmonary: Normal
Tricuspid: Normal

Heart rate : 113 bpm, regular

Septum:
IVA: Intact
IVS: Intact

Chambers:
Left ventricle: MILD DILATED
Right ventricle: Normal
Left atrium: Normal
Right atrium: Normal

Great arteries:
Aorta: Normal
Pulmonary artery: Normal

Pericardium: Normal
Clot , vegetation : Nil

IVC: Normal size, greater than 50% inspiratory collapse

Regional wall motion abnormality: Mild Global LV hypokinesia
No segmental RWMA at rest

Colour Doppler: Mild mitral regurgitation, Trivial AR, Mild TR – TR jet 45 mmHg. No shunt

Impression: Peripartum cardiomyopathy

- Mild dilated Left ventricle
- Mild Global LV hypokinesia
- Mild MR, AR, TR
- Mild PAH
- Mild LV Systolic dysfunction. (EF 50 %)
- Tachycardia: HR 113 bpm, regular

Dr. B.P. Venkatesh, MD, DNB (CARDIOLOGY)

Please note normal echo does not rule out ischemic heart disease and insignificant shunts. Kindly request the patient to bring relevant cardiac investigations. Your provisional impression would assist in reporting. Thanks for co-operation.

Figure 2

Figure 3

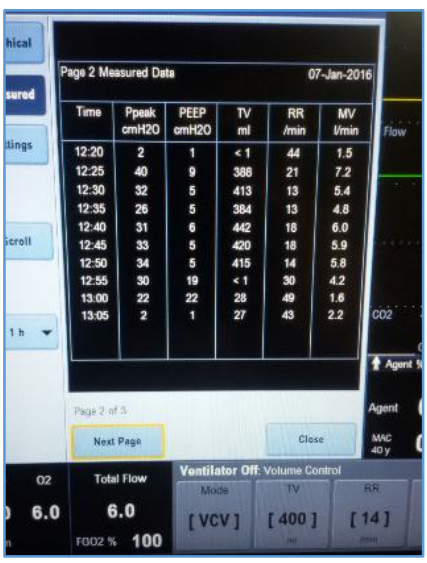

Figure 4

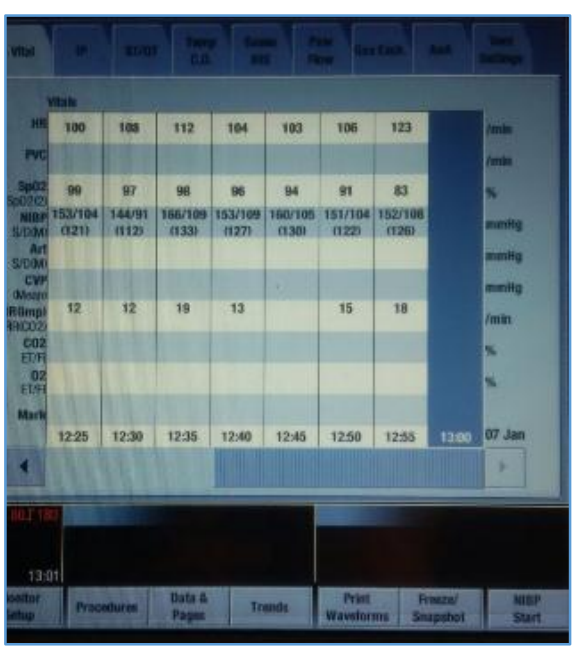

Figure 5

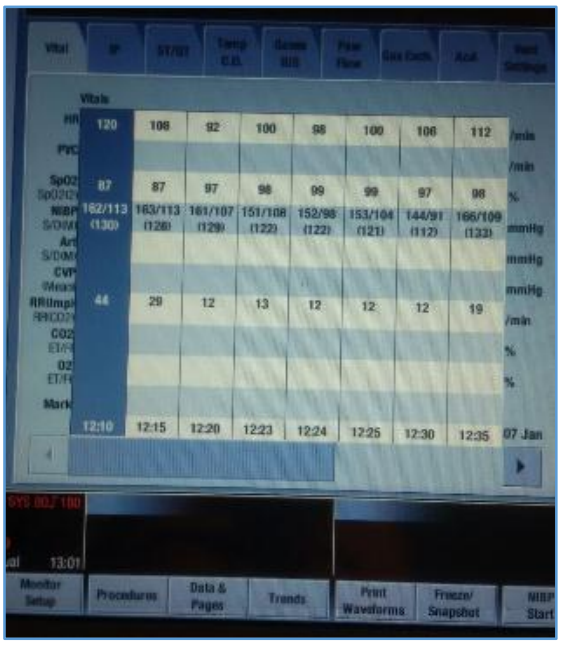

Figure 6

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