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Case Report

Anaesthesia Section

Anaesthetic Management of a Peripartum Cardiomyopathy Patient undergoing Caesarean Section

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ABSTRACT

Peripartum Cardiomyopathy (PPCM), is an uncommon form of heart failure that occurs during the last month of pregnancy or upto five months after delivery. In this condition, the heart chamber enlarges and the muscle weakens. This case report is about a 30-year-old primigravida, with oligohydramnios, at 32.3 weeks of gestation. She visited for a caesarean section, and was diagnosed with PPCM on the basis of clinical findings of growing fatigability, troubled breathing and severe dyspnoea and on echocardiography ejection fraction of 64% with mild mitral regurgitation, moderate dilated left ventricle, tricuspid regurgitation was found. These patients require vigilant anaesthetic intervention for management of painless labour and/or either vaginal or operative delivery. The basic haemodynamic goals should always be kept in mind for favorable maternal as well as foetal outcome while selecting the drug dose and mode of anaesthesia. Various studies showed that both general and regional anaesthesia can be used with comparable outcomes in PPCM patients undergoing caesarean section. The index patient, who needed a caesarean section, was managed with Combined Spinal-Epidural (CSE) anaesthesia, weighing the risk of increase mortality due to general anaesthesia.

Keywords: Epidural anaesthesia, Heart failure, Primigravida, Spinal

CASE REPORT

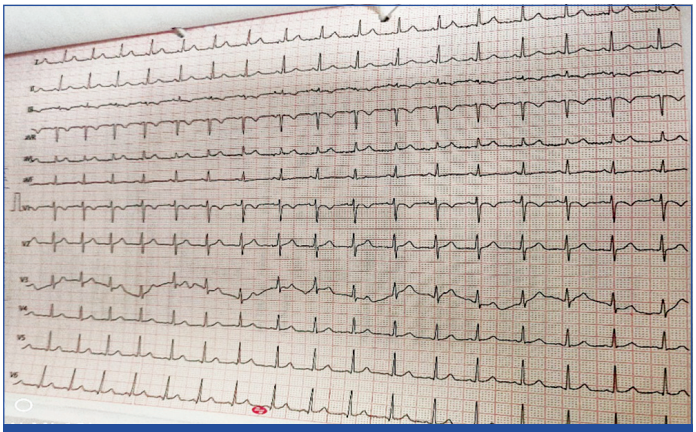
A 30-year-old primigravida with oligohydramnios at 32.3 week gestational age arrived with complaints of growing fatigability, troubled breathing, and severe dyspnoea on minimal physical exertion for five days, and pain in bilateral lower leg since three days. She had never been on any medications before. She was never diagnosed with hypertension, asthma, or any other co-morbidity.

On examination, the patient was afebrile, with a pulse rate of 120 beats/minute, and blood pressure of 140/88 mmHg. Auscultation revealed bilateral basal murmur. In both lower lobes of lungs, air entry was reduced. Patient had 92% saturation on room air, and 97% saturation on 12 litre oxygen. Respiratory rate was 20 beats/minute. Arterial Blood Gas (ABG) revealed no metabolic or respiratory acidosis. In both legs, she had minor pitting pedal oedema. Temporomandibular joint mobility was normal, and mouth opening was 6 cm. The patient's laboratory investigations are shown in the [Table/Fig-1].

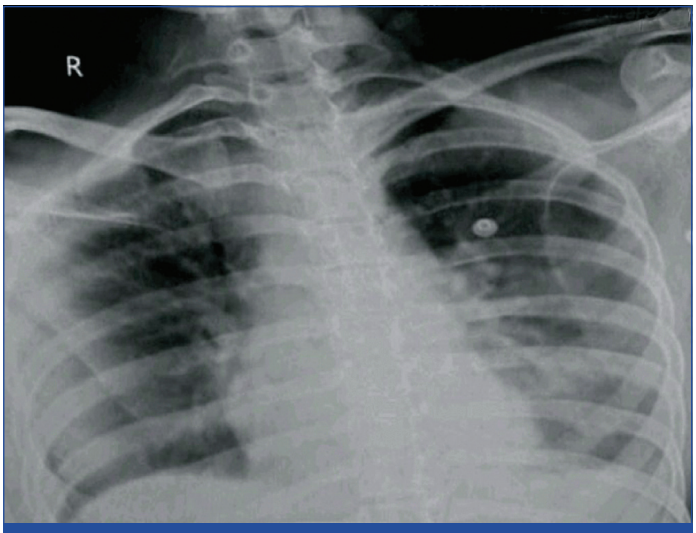
Investigations	Findings	Normal range
Haemoglobin (gm%)	10.2	12-15
White blood cells (/cumm)	15,300	4000-10,000
Platelet count (lakh/cumm)	2.2	1.5-4.1
PT(sec)/INR	12.5 (1.05)	11-13.5 (0.8-1.1)
D-dimer (ng/mL) (third trimester)	2729	483-2256
NT-Pro BNP (pg/mL) (third trimester)	615	<150
CK-MB (IU/mL)	9	5-25
hs.Troponin(I) (ng/mL)	18.3	0-0.04
Random blood sugar (mg/dL)	104	70-110

[Table/Fig-1]: Laboratory findings.
PT: Prothrombin time; CK-MB: Creatine kinase-MB; NT-Pro BNP: N-terminal (NT)-pro hormone BNP

Thyroid profile, liver and renal function tests were all within normal limits. A bilateral lower limb arterial doppler was performed as well, and the veins appeared to be normal. An ejection fraction of 64% was found on echocardiography, as well as mild mitral regurgitation. With a moderately dilated left ventricle, tricuspid regurgitation. There was sinus tachycardia on the Electrocardiogram (ECG) [Table/Fig-2]. On the right side of the chest, an X-ray revealed pericardial effusion with basal haziness [Table/Fig-3].



[Table/Fig-2]: ECG showing sinus tachycardia.



[Table/Fig-3]: Chest radiograph- right side of the chest revealed pericardial effusion with basal haziness.

Once the patient was diagnosed with Peripartum Cardiomyopathy (PPCM) she was started on frusemide 5 mg and metoprolol 10 mg were given once every 24 hours for preoperative optimisation. The patient was secured with an 18G Intracath on her right hand.

She was also put on inj. ceftriaxone 1 gm i.v. 12 hourly (antibiotic coverage), inj. dexamethasone 12 mg i.v. 12 hourly, and 4 hourly budecort nebulisation. Prior to caesarean section, antiaspiration prophylaxis was administered with Inj. ondansetron 4 mg i.v. Inj. metoclopramide 10 mg i.v, as well as Inj. pantoprazole 40 mg i.v. Once the patient was shifted in operation theatre another 18G interacath was secured in her left hand and given 500 mL of ringer lactate at a rate of 4 mL/kg/hr. The oxygen saturation (SpO_2) probe, ECG, non invasive blood pressure monitor, and chest auscultation for crept and Ronchi was done. The anaesthetic protocol included spinal and lumbar epidural anaesthesia, as well as titrated dosages of local anaesthetic with an opioid. Gradual epidural anaesthesia is recommended to avoid haemodynamic impairment.

Because the woman was in active labour, emergency Lower Segment Caesarean section (LSCS) was performed under spinal anaesthetic with narcotic fentanyl after initial stabilisation and gaining high-risk permission. She was given spinal anaesthesia with 0.5% bupivacaine 1.5 mL (heavy) 23G using a Quincke's spinal need leat the level of L3-L4 Level, T8 (dense) level was reached, and an epidural catheter was inserted in the L2-L3 space, all while in a sitting posture and under aseptic conditions.

After 15 minutes, adequate analgesia was observed upto T8 level. She was then positioned supine with a wedge inserted beneath her to minimise aortocaval compression. Intraoperative blood pressure was kept between 120 and 100 mmHg systolic and 80 and 70 mmHg diastolic, with a pulse rate of 90-100 beats per minute. Blood loss was around 850 mL, and intraoperative monitoring, fluid input, andoxytocic dosage were performed. The patient was given 500 mL of restricted fluid and one unit of colloid (500 mL) intraoperatively, with a urine output of 350 mL. In addition to Inj. dexamethasone 12 mg, Inj. hydrocortisone 100 mg i.v. was given. Epidural fentanyl mixed with local anaesthetic was given as10 mL of 0.12% percent bupivacaine with 50 µ fentanyl after half-hour (intraoperative). An appropriate level of analgesia was attained.

A2, 800 gm female child was born and was shifted to the postoperative intensive care unit for 24 hours of observation. Epidural top-up was also used in the postoperative period Inj. bupivacaine 0.125%+25 µg fentanyl given after three hours. The patient's vital signs were normal, and she had no complaints.

Head-up posture, nebulisation, deep breathing exercises, chest physiotherapy, and Deep Vein Thrombosis (DVT) prophylaxis with low molecular weight heparin, enoxaparin 40 mg once every 24 hours were all used to avoid PPCM after surgery. A liquid diet was restarted after a brief hiatus.

DISCUSSION

Peripartum cardiomyopathy is a rare illness that affects women during their third trimester and for upto five months after giving birth [1]. Antenatal individuals with a history of PPCM or who are suspected of having PPCM should be closely watched and treated for heart failure as soon as possible, if necessary. Viral infections, selenium insufficiency, autoimmune, and genetic predisposition are all risk factors for peripartum cardiomyopathies. Chronic hypertension, numerous pregnancies, advanced maternal age, assisted reproductive technologies, and other variables are among the others [2]. A majority of the patients have exertional dyspnea (NYHA-III or IV), and some of them have a history of complicated arrhythmias, pulmonary embolic events, or cardiac arrest [3]. A part from ECG and 2D echo, all standard examinations, thyroid function tests, and serum electrolytes should be performed for the management of cardiomyopathy patients. Brain Natriuretic Peptide (BNP) or N-Terminal pro-Brain Natriuretic Peptide (NT pro BNP) levels values are highly sensitive to screening for adverse maternal events related to cardiomyopathy and to assess risks of heart failure [4].

In this case, continuous ECG, invasive blood pressure monitoring, pulse oximetry, and right internal jugular vein cannulation were all used for intraoperative monitoring. Urine output was monitored while the patient was catheterised. Intraoperatively, restricted intravenous fluids were provided in accordance with the therapy of heart problems during pregnancy [4]. A bilateral lower limb arterial doppler scan was done to rule out venous thrombosis risk.

The treatment of PPCM is identical to that of heart failure and is caused by other causes like- hypertension, valvular heart disease, uncontrolled arrhythmia, myocarditis, constrictive pericarditis [5]. Heart failure can be acute or persistent in pregnant patients. Patients with a known history of heart disease may present in a stable condition early in pregnancy, and their therapy consists primarily of routine monitoring for cardiac breakdown and prescription adjustments.

Patients with decompensated cardiac state or in the peripartum phase during pregnancy may have a history of heart illness or develop it during the pregnancy, such as PPCM. The most common finding in patients with acute decompensation is acute pulmonary edema. A careful assessment of the severity of decompensation is required in the treatment of these patients [6,7].

The index patient was in the acute decompensation stage. Preoperatively, she was stabilised with frusemide, head-up posture, 100% oxygen through face mask, nebulisation with budesonide four times a day, and inj. In the calculated dose, metoprolol was utilised to regulate heart rate. Acute pulmonary edema with respiratory failure in pregnant patients requires intubation with suxamethonium with ventilator support, with (PEEP) of 8 cm H_2O.Cardiotocography is done for foetal monitoring at frequent intervals. After stabilising the vitals of the patient then only the decision of termination of pregnancy by LSCS should be undertaken [8].

In these individuals, the major goal of treatment is to improve haemodynamics and cardiac contractility. Controlling hypertension or hypotension, managing cardiac arrhythmias and pulmonary congestion, and avoiding thromboembolic events could all help. Digoxin is safe to take throughout pregnancy. If the salt limitation is insufficient, diuretics can be administered. In individuals with PPCM, beta-blocker-like drugs (metoprolol, carvedilol) improve left ventricular function, although ACE inhibitors are the therapy of choice in postpartum PPCM [9]. Preoperatively, Inj. metoprolol was given to the patient in order to optimise heart rate. Furosemide, a diuretic, was used to help avoid heart failure and reduce pulmonary congestion.

In situations of PPCM, class III antiarrhythmics (amiodarone) are the best option for treating ventricular arrhythmias [10]. Invasive cardiac monitoring should be used to guide IV therapy with inotropes like dobutamine, adrenaline, and milrinone. Because pregnancy is a hypercoagulable state, anticoagulants must be used in patients with severe PPCM [11]. Unfractionated or low molecular weight heparin is given before delivery, while war far in is utilised during the postpartum phase [12].

Pregnancy can deteriorate with illness progression, increasing the risk of hypertension, repeated abortions, thromboembolism, and other complications. It raises the chances of maternal death and morbidity, as well as foetal problems. It can result in foetal problems such as Intrauterine growth restriction, intrauterine death, and congenital heart disease, among others [Table/Fig-4] [13].

Causes of maternal heart failure	Adverse neonatal outcome
• Cardiomyopathy • Hypertension/Preeclampsia • Multiple gestation • Smoking during pregnancy	• Low birth weight, SGA • Infant Respiratory Distress Syndrome (IRDS) • Lower APGAR score • Prematurity • Neonatal death

[Table/Fig-4]: Adverse Neonatal outcomes associated with maternal heart failure [13].

Adverse events such as heart failure and/or ventricular tachycardia, atrial fibrillation, and transient ischaemic attack might develop as a result of the increased haemodynamics train on the heart [14]. In the present case, a multidisciplinary approach was used to manage

this patient, which included an obstetrician, cardiologist, physician, radiologist, and anaesthesiologist.

Preoperative optimisation, perioperative management, and post operative stabilisation are all part of the PPCM patient's anaesthetic management. Anaesthesia can be administered in two ways: general and localised. The type of anaesthesia used is determined by the urgency of the delivery and the patient's haemodynamic stability. The basic goal of anaesthesia is to maximise cardiac output by maintaining preload and lowering afterload. Maintaining myocardial contractility is also important, as is avoiding medicines that can reduce cardiac contractility further.

The PPCM is a significant challenge for anaesthesiologists. The use of anaesthetics is determined by the urgency of the delivery and the patient's physiological condition. To avoid drops in cardiac output, coronary perfusion must be maintained [15,16]. Vaginal birth is preferred in patients with compensated PPCM [17].

In caesarean sections with severe cardiac decompensation, where even minor sympathetic blocking might result in heart failure, particularly in patients taking anticoagulants, general anaesthesia is preferred as an option. Rapid general anaesthetic induction should be avoided since it can cause a drop in systemic vascular resistance, which can compromise coronary perfusion.

If general anaesthesia is chosen as the plan of therapy for decompensated cases, the induction should be smooth to reduce the risk of both hypotension and hypertension. Opioids, such as alfentanil or remifentanil, can minimise the laryngoscopy press or reaction and intubation [18]. In patients scheduled for a non emergent caesarean section who have a generally stable haemodynamic status, incremental epidural dose should be employed. Early labour epidural analgesia is indicated to reduce cardiovascular stress. This also decrease after load and provide a means of achieving surgical anaesthesia [19].

In PPCM patients, regional anaesthetic treatments (epidural, CSE) can be a superior choice [20]. The insertion and removal of the neuraxial catheter in patients taking anticoagulants should be coordinated with the timing of anticoagulation treatment. The main benefit of epidural is that it allows for unrestricted titration of the local anaesthetic dose to obtain the optimum motor and sensory block level without triggering rapid hypotension and decompensation in these patients [21].

Regional anaesthetic procedures increase cardiac function and reduce heart work load, whereas epidural insertion aids in achieving appropriate postoperative analgesia.

In the present case report, the patient was in the latent phase of labour, with no signs of advancement. The LSCS was performed under subarachnoid and epidural anaesthesia, with 1.5 mLof 0.5% bupivacaine used for the spinal block (Heavy). After the test dose, 6 mL of lignocaine 2% with fentanyl 50 µg was delivered through an epidural catheter inserted in the L2-L3 region. There were no difficulties during the procedure, and appropriate analgesia was established afterward.

CONCLUSION(S)

In order to avoid unintended intraoperative problems, a thorough preoperative examination is necessary. According to the patient's health, the choice of anaesthetic approach should be carefully considered before surgery. In the present case report, a subarachnoid block was employed to induce the case, with the focus on coronary perfusion and epidural anaesthesia. A combination strategy was selected, taking care not to compromise the patient's haemodynamics intraoperatively and to give good postoperative pain management.

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