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Anaesthetic Management Of Peripartum Cardiomyopathy – A Case Report

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Abstract

Peripartum cardiomyopathy (PPCM) is an uncommon form of heart failure with no identifiable cause that happens during the last month of pregnancy or upto 5 months postpartum. Some authors believe this to an abnormal cardiac response to hemodynamic changes during pregnancy that endangers the life of mother as well as fetus. The hallmark of the disease is onset of decreased ejection fraction either in late pregnancy or in early puerperium.

This case report describes the anaesthetic management in a patient who presented with signs and symptoms of decompensated heart failure (raised Jugular Venous Pressure, anasarca pitting type, severe hypertension) in labour at 39.6 weeks of gestation undergoing Emergency Lower Segment Caesarian Section (E-LSCS). After exclusion of all possible causes and correlating with 2D Echogram findings, the heart failure was attributed to peripartum cardiomyopathy. We concluded that early identification and intervention leaded to a successful outcome for the mother as well as the fetus.

Keywords: Primigravida, low ejection fraction, Peripartum cardiomyopathy, general anaesthesia, Lower segment Cesarean section

Introduction

The clinical presentation of the Peripartum cardiomyopathy is similar to that of dilated cardiomyopathy due to any other cause, but the pregnant or lactating state of the mother should be considered at all times. Pregnancy is a state of high blood volume, approximately 40-50% above normal range which increases the preload and in turn increase the cardiac output. A physiologically normal heart has enough reserve to cope with these changes, whereas a damaged heart might not be able to compensate, pushing the patient towards a decompensated heart failure. A vigilant monitoring, both noninvasive and invasive is essential throughout the surgery and in the postoperative period to avoid complications like arrhythmias (atrial fibrillation), hypotension, hypoxemia, pulmonary edema, electrolyte disturbances, myocardial ischemia, thromboembolism, and even sudden death[(1)].

This case report describes the anaesthetic management in a case of peripartum cardiomyopathy.

Case Report

A 23 year old female, primigravida, by date 39.6weeks and by scan 41.1weeks came to MGM Kalamboli Women and Children Hospital with shortness of breath and pain in abdomen since 2 hours.

The patient had visited a public health centre for routine antenatal checkups once in first and once in second trimester. Neither of the two times was anything abnormal noticed in her examination or investigations. Patient presented to MGM hospital casualty in labour, in a decompensated state with vitals as follows:

Pulse – 110/minute, blood pressure – 190/100 mm Hg, oxygen saturation – 84% on room air. Anasarca was present, pitting type. The Jugular Venous Pressure was raised. On auscultation, Bilateral coarse crepts in lower zones and pan systolic murmur was present. She was given Inj. Labetalol 20 mg and Inj. Furosemide 10 mg stat and the patient was shifted to operation theatre for Emegency Lower Segment Caesarian Section (LSCS).

Patient was taken into the OR and all standard monitors including saturation probe, blood pressure cuff, electrocardiogram leads were attached. The patient was disoriented with Glasgow coma score of $E_2V_1M_4$ with pre induction vitals as follows: pulse – 100/minute, BP – 160/100 mm Hg, oxygen saturation – 92% on 15 litre oxygen by non rebreathing mask.

Patient was pre-oxygenated with 6 litre/min and premedicated with Inj. Glycopyrrolate 0.004mg/kg body weight. Inj. Propofol 2mg/kg body weight was given. Rapid sequence induction was done with Inj. Succinyl choline 2mg/kg body weight. She was intubated with Endotracheal tube number 7, air entry bilaterally equal confirmed on auscultation and tube was fixed at 20 lip mark. Baby was delivered within 3 minutes of induction. After delivery of baby, patient was given inj. Oxytocin 20 units in 500 ml Ringers Lactate intravenous at the rate of 60ml/hr. Intraoperatively, the plane of anaesthesia was maintained by sevoflurane 1%w/w, oxygen with high flow of 6L/min` and Inj. Atracurium as per maintenance dose of 0.5mg/kg body weight. There was 1 episode of sudden desaturation to a minimum of 80% on mechanical ventilation. intraoperatively which was managed with adequate suctioning, Inj. Hydrocortisone 200mg and Inj. Dexamethazone 8mg. An arterial blood gas was sent which depicted mixed respiratory and metabolic acidosis with pH - 7.23, pCO2 – 40 mm Hg, pO2 – 49mm Hg, HCO3 – 16.3mmol/L, SpO2 – 88%. Post operatively, patient was shifted to intensive care unit sedated and paralysed on continuous mechanical ventilation delivering a tidal volume of 400ml, respiratory rate of 14breaths/minute, positive end expiratory pressure of 5 mm Hg and 100% FiO_2. Chest X ray depicted multiple homogenous opacities in all lung fields suggestive of pulmonary edema secondary to heart failure or severe Pneumonia leading to acute respiratory distress syndrome. A screening 2D Echogram was done which revealed right ventricular dilataion, inferior vena cava was congested, global left ventricular hypokinesia and ejection fraction was approximately 25%. As no obvious cause of heart failure was detected, the diagnosis of peripartum cardiomyopathy was made. The cardiologist advised to take Tab. Furosemide 20 mg BD, Tab. Metoprolol 25mg OD and strict blood pressure charting with addition Angiotensin converter enzyme inhibitor for control of hypertension if required. She was weaned off the ventilator and extubated 7 hours post operatively. After a 48 hours stable course in ICU, patient was shifted to cardiology ward for further management and then discharged at stable hemodynamics after 10 days and is now on regular follow up.

Discussion

Peripartum Cardiomyopathy occurs in 1:300 to 1:15000 pregnancies[3]. Originally, it was thought to appear after parturition and hence it was named 'postpartum cardiomyopathy' or 'puerperal heart failure'. It was later discovered that the onset of signs and symptoms can occur anytime in last month of pregnancy upto 5 months post partum. The etiology is still not known but it is supposedly attributed to nutritional deficiencies, small vessel coronary artery abnormality, hormonal effects, toxaemia, maternal immune response to fetal antigen or myocarditis[3]. Some other hypothesized causes are viral, autoimmune, hemodynamic stress of pregnancy, cytokine- mediated inflammation, Gq-related myocyte apoptosis, oxidative stress-induced Cathepsin D production, selenium deficiency[1]. Risk factors include advanced maternal age (>30 years), multiparity, multifetal gestation, obesity, hypertension, eclampsia and black race.

The diagnostic criteria for PPCM are as follows:

1. Development of heart failure in last moknth of pregnancy or upto 5 months post partum.
2. The absence of an identifiable cause for cardiac failure.
3. No recognizable heart disease before the last month of pregnancy.
4. Left ventricular systolic dysfunction shown by echocardiographic criteria.

These patients usually present with signs and symptoms of decompensated heart failure due to dilated cardiomyopathy. Pulmonary edema is the

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most common presentation of PPCM. Rarely, it may also present as thromboembolism or cardiac arrhythmias. It is crucial to differentiate preeclampsia and PPCM inorder to start early management which is beneficial for the mother as well as the fetus. A high index of suspicion of heart failure should be kept in mind for symptoms like paroxysmal noctural dyspnoea, orthopnea, chest pain, persistent cough, new regurgitant murmurs, pulmonary crackles or crepts, elevated jugular venous pressure, hepatomegaly, and postural hypotension reflecting low cardiac output[(1)].

Anesthesia management is similar to those for any patient with a dilated cardiomyopathy. The management of PPCM needs a multidisciplinary action. In general, unless there is deterioration in the maternal or fetal well- being, there is no need for urgent or emergent delivery and the pregnancy is allowed to progress to term[(2)]. The patient may be allowed to deliver the baby vaginally or operatively according to obstetric parameters or patient's wish[(2)]. However, this needs to be done with continuous hemodynamic monitoring and invasive blood pressure monitoring if required.

In case of an emergency, if cesarean section is required the analgesics and anaesthetics are used judiciously so as to prevent further cardiac depression. Combined epidural and spinal anesthesia is preferred since it avoids stress of general anesthesia and causes its peripheral vasodilation which eventually leads to decrease in LV afterload. Although it may also lead to hemodynamic instability due to reduction in systemic vascular resistance and limited cardiac reserve[(2)]. However, if a patient comes in a severely decompensated state, like in our case, general anesthesia is considered to be a safer and quicker method of induction. Inj. Etomidate was used for induction due to its property of providing better hemodynamic stability as compared to other induction agents. Etomidate has proved to be safer in patients with heart disease. After delivery of baby, Inj. Fentanyl and sevoflurane was used for maintenance of anaesthesia. Opioid- based anaesthesia provides good hemodynamic control and obtundation of response to endotracheal intubation but may require postoperative ventilatory support for both mother and neonate[(2)]. Thus, it is obvious that favorable maternal and fetal outcome is not dependent on anaesthetic technique, but strict hemodynamic control and meticulous cardiovascular monitoring with close coordination between various involved specialists[(2)]. Patient was shifted to ICU intubated, sedated and paralysed and an elective extubation was planned to avoid response that would increase cardiac stress and worsen the prognosis. The main aim of management in PPCM is to reduce cardiac preload and afterload and maintaining an increase in myocardial contractility.

Bed rest, digitalis, diuretics, anticoagulant and antihypertensive therapy are most important part of perioperative management. Levosimendan is a novel cardiotropic agent that improves cardiac output by increasing the response of myofilaments to intracellular calcium is found to be beneficial in PPCM[(2)]. Levosimendan is used as an intravenous infusion at the rate of 0.1–0.2 μg/kg/min in cardiac failure with or without a loading dose of 3–12 μg/kg over 10 minutes[(2)].

The mortality rate is found maximum within the first 3 months. However, as many as 60% patients show near complete recovery of cardiac function, usually within 6 months. Patients with persistent cardiomyopathy at 6 months have a reported mortality of 85% at 5 years and subsequent pregnancies are associated with relapses and high risk for maternal morbidity and mortality[(6)]. Patients who recover normal left ventricular function and have normal left ventricular contractile reserve after dobutamine challenge may undertake another pregnancy safely, but they should be warned of the risk of recurrence even with fully recovered left ventricular function[(4)].

Conclusion

Peripartum cardiomyopathy causes excessive morbitity and mortality in the mother. Any delay in detection or treatment can cause lethal complications in the mother as well as the fetus. Early detection and intervention along with regular follow ups post operatively leads to a positive outcome. In any case, a prudent titration of anaesthetic techniques and drugs maintaining the cardiac contractility along with continuous hemodynamic monitoring leads to a better maternal and fetal outcome.

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