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ANAESTHETIC MANAGEMENT OF A KNOWN CASE OF PERIPARTUM CARDIOMYOPATHY POSTED FOR EMERGANCY CAESAREAN SECTION

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ABSTRACT

Peripartumcardiomyopathy(PPCM) is an idiopathic cardiomyopathy that presents with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the early months after delivery, in the absence of any other cause of heart failure.It has an incidence of one per 3500 live births and is associated with a high mortality rate (30–60%).Anaesthetic management of a patient with PPCM should be comprised of adequate preoperative optimization using a multidisciplinary approach, careful monitoring, proper use of anaesthetic techniques and vigilant postoperative care. We report a case of a 25 years old, primigravida patient with difficulty in breathing and generalized weakness at 7 months of gestation. Patient was diagnosed to have dilated peripartum cardiomyopathy. Endotracheal anaesthesia with inj. Fentanyl,inj.Pentothal and inj. Vecuronium bromide was given and maintained on oxygen and sevoflurane. Intraoperative period was uneventful. Patient was extubated, postoperatively patient was conscious, co-operative & haemodynamically stable with no pain. Patient was shifted post operatively in intensive care unit for observation for 24 hours..

Key words: Peripartum Cardiomyopathy, Cesarean Section, General Anesthesia.

INTRODUCTION

PPCM is a diagnosis of exclusion. Although the left ventricle may not be dilated, the ejection fraction is nearly always reduced below 45%.PPCM is a severe form of heart failure which was first described by Demakis and Rahimtoola1 comprising of 3 criteria. Later, another one was added by the PPCM workshop committee of the National Institute of Health (NIH).

1) Development of heart failure in the last trimester of pregnancy or within the first 5 months of postpartum
2) Absence of any determinable cause of cardiac failure
3) Any prior demonstrable heart disease
4) Echocardiographic evidence of left ventricular dysfunction

PPCM is a diagnosis of exclusion. Although the left ventricle may not be dilated, the ejection fraction is nearly always reduced below 45%.

Peripartum cardiomyopathy is associated with maternal and fetal complications.

Maternal complications –hypoxia
- Progessive cardiac failure
- Arrhythmias
-Thromboembolism

Fetal complications like fetal distress, IUGR and small for date baby are likely to occur. Fetal distress is either due to maternal hypoxia or placental hypoperfusion as a result of maternal hypovolemia or poor cardiac output [1-9].

CASE REPORT

A 25 years old female primi patient presented with 8 months of amenorrhea. Patient had developed difficulty in breathing and generalized weakness at 7 months of gestation.

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Patient was diagnosed to have dilated peripartum cardiomyopathy and put on treatment with T.lasix 40 mg OD and T.isosorbide 10 mg BD. Patient was on this treatment since one month with regular follow up weekly. Patient was admitted during routine antenatal check up one day prior and known to have breech presentation and fetal distress. On examination, patient was conscious, following verbal command, oriented to time, place and person. Temperature was normal by palpation, pulse Rate 52/min with every alternate missed beats, blood pressure 110/64 mm Hg; absence of Pallor, clubbing, cyanosis, jaundice, edema. Her lungs shows bilateral fine basal crepitations on auscultation and cardiac examination showed loud s1 and ejection systolic murmur on pulmonary area. On airway examination mouth opening was adequate with Mallampati grade-1. Her haemoglobin was 9 mg /dl, Blood sugar was 70 mg/dl, S.Sodium was 140 and S.potassium was 4.3meq/l, Blood urea was 15 mg/dl. S.Creatinine was 0.6 mg/dl. Her ECG suggestive of ventricular trigeminy with ST – T segment changes and T wave inversion in V1 - V6; and Echocardiography shows EF – 20 % with global left ventricular hypokinesia , mild PAH / mild MR and reduced left ventricular compliance, Lt atrium/Lt ventricle dilated with severe Lt entricle dysfunction. Her SPO2 – 96 % with ventimask at oxygen 4 lit / min. Written & informed high risk consent of ASA grade IV with emergency was taken. Anaesthesia work station had been checked and difficult airway cart was kept ready. Baseline monitoring like ECG, NIBP, Pulseoxymetry was applied and central venous line was secured in right brachial vein. Baseline parameters were recorded. The pre-induction pulse rate was 56/ min and blood pressure was 118/76 mm of Hg she was premedicated with Inj. Ondansetron 4 mg IV, Inj. Ranitidine 50 mg IV 10 min. before induction. Injglycopyrrolate 0.2 mg iv and Inj fentanyl 50 mcg iv were given 3 min before induction.

Intravenous Induction was carried out with Inj. thiopentone200 mg iv and Inj. Vecuronium Bromide 5 mg iv. The patient was Intubated endotracheally with portex, cuffed endotracheal tube no. 7 mm ID, BLAE checked, cuff inflated and tube fixed. Anaesthesia was maintained with 100% O2 and Sevoflurane and Neuromuscular blockade was achieved with intermittent doses of Inj. Vecuronium bromide.

- A healthy female baby weighing 2.5 kg was delievered after 5 min of induction of aneasthesia. APGAR score was 6 and 8 at 1 min and 5 min respectively. After delivery of baby, injection pitocin 20 U in500 ml 5 % dextrose was started. Injection midazolam1.5 mg IV were also given. Inj.Diclofenac 75 mg IV and inj. Lasix 20 mg IV also given. Patient had been given fluid according to cental venous pressuer maintaining around 8-10 cm H_2O.250 ml DNS and 250 ml RL was given intraoperatively. Intraoperative urine output was 500 ml. Intraoperatively patient remained stable hemodynamically throughout the procedure, which lasted for 35 min. Neuromuscular blockade was reversed with Inj. Glycopyrrolate 0.4mg and Inj. Neostigmine 2.5 mg and the patient was extubated when she had sustained spontaneous respiratory effort. Post-extubation, patient was conscious, following verbal commands with respiratory rate of 14 to 16/min, regular with adequate tidal volume and muscle tone, power. Patient was shifted post operatively in intensive care unit for observation for 24 hours. IVfluids, analgesics and urine output were major concerns postoperatively.

DISCUSSION

When peripartum cardiomyopathy occurs late in pregnancy, early delivery of the fetus is done to reduce hemodynamic stress on the maternal heart. The mode of delivery is generally based on the obstetric indications. After optimization of the mother's condition, induction of vaginal delivery can be attempted in most cases. The advantages of vaginal delivery are minimal blood loss, greater hemodynamic stability, avoidance of surgical stress, and less chances of postoperative infection and pulmonary complications. However, a vaginal delivery may increase cardiovascular stress, even when optimal pain relief is administered.

Emergency Caesarean delivery is reserved for indications such as fetal distress and primi breech failure to progress as in our case. With possible effects of haemodynamic compromise associated with sympathetic blockade, a general anaesthesia technique was felt to be the best method of providing stable analgesia and anaesthesia for emergency caesarean delivery. Regional anaesthesia may have provided an advantageous reduction in afterload but its effects on haemodynamic status were felt to be less predictable than that of a general anaesthetic using suitable agents. Epidural aneasthesia is also a safe alternative to general aneasthesia but it is not suitable for emergency Caesarean delivery as in our case.

- Management of anaesthesia for this patient includes

- Maintenance of normal to low heart rate to decrease oxygen demand
- Prevention of large swings in blood pressure
-Minimizing haemodynamic effects of general endotracheal anaesthesia
- Minimizing narcotic related neonatal respiratory depression.
- Volatile agents that preserve LV contractility without dramatic vasodilatation are desirable
- Agents that directly or indirectly increase heart rate and contractility have to be avoided or used with caution
- Blood loss has to be replaced promptly and CVP monitoring may be useful in titrating fluids

Pathophysiology

- Proper etiology of PPCM is unclear and multifactorial.
- Risk factors for PPCM are advanced maternal age,

- Malnutrition, multiparity, poor antenatal care, tobacco abuse, hypertension and preeclampsia.
- Infection, inflammation, autoimmunity, abnormal response to heamodynamic stress of pregnancy and oxidative stress induced cathepsin D are likely etiologic factors.

Clinical Features

- The severity of symptoms in patients with PPCM can be classified by the New York Heart Association system as follows:

Class I - Disease with no symptoms
Class II - Mild symptoms/effect on function or symptoms only with extreme exertion
Class III - Symptoms with minimal exertion
Class IV - Symptoms at rest

- Many presenting complaints observed in patients with cardiac disease occur during a normal pregnancy. Mild dyspnea upon exertion is particularly common in a normal pregnancy. The classic dyspnea of pregnancy is often described as the woman feeling as if she is unable to get enough air in, to get a good deep breath, or both, and it is thought to be due to the progesterone-mediated hyperventilation.
- Symptoms are the same as in patients with systolic dysfunction who are not pregnant. New or rapid onset of the following symptoms requires prompt evaluation:
    - Cough
    - Orthopnea
    - Paroxysmal nocturnal dyspnoea
    - Fatigue
    - Palpitations
    - Hemoptysis
    - Chest pain
    - Abdominal pain

Complications

Peripartum cardiomyopathy is associated with maternal and fetal complications.
Maternal complications
–hypoxia
- Progessive cardiac failure
- Arrhythmias
- Thromboembolism

Fetal complications like fetal distress, IUGR and small for date baby are likely to occur. Fetal distress is either due to maternal hypoxia or placental hypoperfusion as a result of maternal hypovolemia or poor cardiac output.

CONCLUSION

- Anaesthetic management of a patient with PPCM should be comprised of adequate preoperative optimization using a multidisciplinary approach, careful monitoring, proper use of anaesthetic techniques and vigilant postoperative care.
- When cardiomyopathy occurs late in pregnancy, early delivery of the fetus is done to reduce hemodynamic stress on the maternal heart. The mode of delivery is generally based on the obstetric indications.
- After optimization of the mother's condition, induction of vaginal delivery can be attempted in most cases. The advantages of vaginal delivery are minimal blood loss, greater hemodynamic stability, avoidance of surgical stress, and less chance of postoperative infection and pulmonary complications. However, a vaginal delivery may increase cardiovascular stress, even when optimal pain relief is administered.
- Emergency Caesarean delivery is reserved for indications such as fetal distress and primi breech /failure to progress as in our case.
- With possible effects of haemodynamic compromise associated with sympathetic blockade, a general anaesthesia technique was felt to be the best method of providing stable analgesia and anaesthesia for emergency caesarean delivery. R egionalanaesthesia may have provided an advantageous reduction in afterload but its effects on haemodynamic status were felt to be less predictable than that of a general anaesthetic using suitable agents.
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  - Minimizing narcotic related neonatal respiratory depression.
  - Volatile agents that preserve LV contractility without dramatic vasodilatation are desirable.
  - Agents that directly or indirectly increase heart rate and contractility have to be avoided or used with caution.
  - Blood loss has to be replaced promptly and CVP monitoring may be useful in titrating fluids.

The patient is advised not to become pregnant again. Because of her first pregnancy course, her family has been warned that she might not be able to tolerate another pregnancy.

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