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Anaesthesia in a Patient Undergoing LSCS with Peripartum Cardiomyopathy- A Case Report

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ABSTRACT

Peri-partum cardiomyopathy(PPCM) is an idiopathic cardiomyopathy presented as heart failure due to left ventricular systolic dysfunction towards the end of pregnancy or in the months after delivery when no other cause of heart failure. In this case report we will discuss the anesthetic mangament in a patient with PPCM posted for LSCS. The anaesthesic goal is to maintain Cardiac output with minimal effect on prelaoad and afterload. No complications were observed.

Keywords: PPCM, LSCS.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopathy which presents as acute heart failure in the third trimester of pregnancy or in early postpartum period.[1] It was first seen by Ritchie in 19th century,[2] as an idiopathic disease. Echocardiographic features of left ventricular dysfunction were added later to make the definition more precise.[3,4] Anaesthetic management of caesarean section in PPCM patients can be a challenge to the anesthesiologist.[5,6] The goals of anaesthetic management include maintenance of optimal ventricular preload and after load.[7] Understanding the nature of the disease, its pathophysiology, post op complications and the correct choice of Anaesthesia is crucial for the better outcome. Both noninvasive and invasive monitoring is essential throughout the surgery and in the postoperative period to avoid complications like arrhythmias, hypotension, hypoxemia, pulmonary edema, electrolyte imbalances, myocardial ischemia, thromboembolic episodes, and even sudden cardiac death.[5-8]

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CASE REPORT

A 29 years old multigravidae was referred to our hospital 31 weeks of gestation with complaints of increasing respiratory difficulty since four days with associated preeclampsia. On admission she was diagonosed with overt DM. She was shifted to the intensive care unit where she was diagonosed with PPCM complicated with preeclampsia based on clinical and echocardiographic findings and was put on Injection Lasix 20 mg twice daily, oral Amlodipine 5mg once daily and oral clonidine 250 mcg thrice daily. The previous two trimesters were uneventful. Echocardiography was done and had revealed dilated Cardiomyopathy with a left ventricular ejection fraction of 20% with moderate to severe MR, severe PAH (RVSP- 48 mm Hg).

Preanaesthetic examination revealed pulse rate (PR), blood pressure (BP), peripheral oxygen saturation (SpO2) and respiratory rate of 88 beats/min, 170/110 mm Hg, 96% on room air and 15/min, respectively. The chest was found to be clear on auscultation. Examination of the cardiovascular system (CVS) showed normal first and second heart sounds with no added sounds. Her routine laboratory investigation including renal function tests and coagulation profile were within normal range. LFT were deranged with AST 476 U/L, ALT 314 U/L, ALP 384U/L. ECG showed Sinus Tachycardia with short PR interval. General Anesthesia was planned and an informed written consent was taken from the patient and the husband after explaining the risk involved. Fasting blood

sugar were with in normal limits on day of surgery. Patient was brought to the operation theatre on shifting trolley in left lateral position. Patient was shifted to the operating table and a wedge was placed below the right hip. All standard monitors were placed including ECG, NIBP, SPO2 and baseline values were recorded. A 16 G iv cannula is placed on right hand under LA and left radial artery was cannulated under LA. Base line heart rate was 92 bpm, IBP was 114/74 mm Hg and Spo2 was 99% @ RA. After preoxygentaion with 100% oxygen, rapid sequence intubation was done with Thiopentone sodium 200 mg, and succinylcholine 100mg and 7.5mm ID ETT placed in trachea and confirmed by equal bilateral air entry. Central venous cannulation was done postinduction. Dopamine infusion was started at a dose of 2.5 microgm/ kg/min and titrated according to blood pressure. Hemodynamics were maintained with in normal limits throughout the procedure with CVP of 7-9 cm of H2O. Anaesthesia was maintained with intermittent titrated use of sevoflurane in oxygen and air and oxygen saturation was maintained between 97-100% and intermittent boluses of Atracurium. 5 units of oxytocin bolus was given immediately after baby delievery and 15 units of oxytocin infusion started in 500 ml NS. Along with it 100 microgms of fentanyl and 2mg of midazolam given slowly. Arterial Blood Gas was done and it was normal. At the end of the surgery the patient was extubated in deep plane of anesthesia in order to maintain hemodyanamics. At extubation PR was 102 bpm with IABP of 104/68mm Hg and saturation of 98% on venturi at FiO2 0.32. Patient was shifted to ICU for post op monitoring and on 2nd post op day patient was discharged to ward and patient was put on antibiotics, diuretics, beta blocker and digoxin.

DISCUSSION

PPCM is a form of Dilated Cardiomyopathy with left ventricular systolic dysfunction that results in signs and symptoms of heart failure. Symptoms usually occur in the 3rd trimester and diagnosis is made in the peripartum period.[3,9] Etiology is still unknown. It may be due to nutritional deficiencies, small vessel coronary artery abnormality, hormonal effects, toxaemia, maternal immulogic response to foetal antigen or mycarditis.[4] Predisposing factors include maternal age greater than 30 years, multiparous or eclamptic patients, twin pregnancy, hypertension and nutritional deficiencies. In the majority of cases there is no family history.[10] There are different opinions regarding correct method of anesthesia for LSCS in PPCM. Regardless of the anesthetic technique, hemodynamic goals include avoidance of sudden variation in heart rate and blood pressure.

Peripartum cardiomyopathy presents with symptoms that worsens cardiac failure. These include exertional dyspnoea, fatigue, pitting pedal oedema, emboli formation. The patient may present with raised CVP, tachycardia, gallop rhythm (S3), mitral regurgitation, pulmonary crackles and peripheral oedema. Chest X-ray may show cardiomegaly with pulmonary oedema and eventually there may be signs of pulmonary artery hypertension. The electrocardiogram may show non- specific ST and T wave changes, atrial or ventricular arrhythmias and conduction defects. On Echocardiography there may be enlargement of all four chambers with decrease in left ventricular systolic function, Ventricular wall motion abnormality, decrease ejection fraction and increased pulmonary capillary wedge pressure.[10,11] Hyperventilation, pedal edema, distended neck veins, loud first heart sound, splitting of the 2nd heart sound can be seen in normal pregnancy which can confuse the anaesthesiologist with respect to the overdiagonosis or underdiagonosis of the disease.[12]

During GA important things to see are

- Those volatile agents which decrease LV contractility without causing sudden vasodilatation should be used.
- Agents which deceases preload and afterload should be avoided
- Agents that directly or indirectly increase rate and contractility of heart should be avoided eg. Pancuronium, atropine, epinephrine, ephedrine.
- Replace the blood loss minutely.
- Hypotension should be treated with alpha adrenergic agonist.
- Insertion of CVP may induce atrial or ventricular dysarrhythmias.[13] So check for the changes.

In patients with an ejection fraction of <35%, chances of thromboembolic events increases. This can lead to formation of left ventricular thrombi, so patient should be kept on LMWH while pregnant and warfarin following delievery.[14] Correct choice of anaesthesia and precise titration is crucial for a favorable outcome. The challenge in either of these techniques is to avoid myocardial depression, hypovolemia, and prevent any increase in pre- and after load.[6] We opted for general anaesthesia (GA), guided with invasive monitoring in order to keep a close look of hemodynamic changes and severity of the disease. High concentrations of volatile agents, other drug like ephedrine etc and hypovolemia should be avoided as dramatic cardiac depression and uncontrolled changes in afterload and preload may be life threatening. Oxytocin was given slowly as rapid infusion can cause hypotension and tachycardia.8 Fluid management in patients with PPCM is very critical. In our case intra-operative 500 ml of ringer lactate and 500 ml of hydroxyl ethyl starch was given to prevent fluid overload. Overhydration may not be advisable as it may lead to CHF.

CONCLUSION

PPCM is a disease associated with high morbidity and mortality and can lead to maternal and fetal loss and persistent decrease in quality of life in mother. 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 abrupt changes in preload and after load and avoidance of myocardial depression, while decreasing the symptoms of congestive heart failure. Subarachnoid block can be hazardous as it can precipitate sudden and rapid reductions in systemic vascular resistance and there by preload. GA has its own disadvantages but benefits overcome the risks. In all the situations, hemodynamic monitoring and slow and titrated dose of anaesthetic drugs is important to provide a better maternal and fetal outcome.

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