Journal of Cardiovascular Disease Research

ISSN: 0975-3583, 0976-2833 VOL 14, ISSUE 10, 2023

EMERGENCY LSCS OF A PATIENT HAVING MODERATE MITRAL VALVE REGURGITATION COMPLICATED BY PREECLEMPSIA- CASE REPORT

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ABSTRACT

Cardiac disease in pregnancy is a important fector responsible of maternal and fetal morbidity and mortality.(1) Pre-eclampsia is a serious complication seen in pregnancy that is characterized by high blood pressure. Problems arising due to this condition are fetal growth restriction, low birth weight, preterm birth, stillbirth. Eclampsia, increased risk of stroke, cardiovascular diseases, damage to kidneys, liver, eyes, lungs, and heart can be seen in the mother. A 24 year old female who was 1st gravida and 35 week pregnant came to hospital. Due to fetal distress emergency lscs was planned. The anaesthesia management used in a case of an emergency LSCS in a patient with moderate MR with preeclampsia is discussed at length in this article.

Keywords- LSCS, mitral regurgitation, preeclampsia

INTRODUCTION

Cardiac disease in pregnancy is a important fector responsible of maternal and fetal morbidity and mortality. (1). Pregnancy and the peripartum period represent a physiologic burden that may worsen symptoms in even moderate degrees of cardiac disease. Consequently, many women are first diagnosed with cardiac disease during pregnancy. Cardiac disease in pregnancy is associate in 1-4% of pregnancies in developing countries(2)

Pregnancy induced hypertension constitutes a major cause of morbidity and mortality in developing nation and it complicates about 6-8 % of pregnancies. It has long been established that a placenta, but not a fetus, is required, and that the syndrome eventually resolves once the placenta is removed. Hence, in terms of pathogenesis it is primarily a placental disorder. Complications includes late spontaneous miscarriage, abruptio placentae, fetal growth restriction (FGR), pre-term rupture of the membranes, and premature delivery.(3). The lack of spontaneous pre-clinical animal models for these conditions has limited our understanding, but the recent advances in “omics technologies”(4) and the derivation of organoid cultures of the endometrium(5) and placental trophoblast(6,7) create new opportunities for systematic research.

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CASE REPORT:

A 23year old 55kg weighed primigravida came to hospital at 35 weeks with pedal oedema and headache. She also complained dyspnoea on moderate exertion for 4 months. Physical examination revealed an arterial blood pressure of 151/101mmHg, a regular heart rate of 87 beats/min, and a respiratory rate of 22 breaths/min. Due to PIH she was admitted in hospital and further investigated. All routine investigations were send. BP was monitored 4 hourly and it was always more than 150/99mmhg. Initial diagnosis was made by obstetric department – 35 week 4 days primigravida with preeclampsia. And she was given 1 dose of betamethasone for risk of premature birth (for better lung functioning of fetus). And she was catheterised for better urine output monitoring. A refrence for PAC was send to anaesthesia department. During PAC on examination pt was found pale, dyspnoea on moderate exertion. On auscultation respiratory system was ok with no added sound but murmer was heared and ecg was also showing left atrial enlaergment. So 2d echo was advised.

INVESTIGATIONS:

PARAMETER	**VALUE**	**PARAMETER**	**VALUE**
HB	9.5 g/dl	S. urea	1.04 mg/dl
TLC	25.02 ths/ul	S. creatinine	31 mg/dl
Platelet count	298* 1000/uL	Total bilirubin	0.3 mg/dl
PCV	31.4%	SGOT	72 U/L
TSH	**9.11 uIU/ml**	SGPT	53 U/L
T3	1.31 ng/dl	TOTAL PROTIEN	5.4 g/dl
T4	13.8 ug/dl	ALBUMIN	**2.0 g/dl**

On urine examination **albumin and red blood cells** were seen in urine.

Viral markers were negative for HBSAG, HIV, HCV

VDRL negative

PT, INR, BT, CT was with in normal limits.

2d ECHO report :

Concentric LVH with normal systoloic function,

LVEF was 55%,

DIALAED LA,

Moderate MR , starry sky appearance of septum.

Mild pericardial effusion.

MANAGEMENT: Multidisciplinary meetings were held to discuss her management and included cardiology, high-risk obstetrics, and cardiac surgery, as well as cardiac and obstetric anesthesiology, nephrology, endocrinology and paediatrics department.

Tab eltroxin 75 mcg od (empty stomach) was started by endocrinologist for abnormal TFT.

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Tab labetalol 200 mg tds was started for PIH. Hourly bp monitoring and urine output monitoring advised with continue fetal heart rate monitoring.

After 24 hours sudden sudden blood pressure increased suddenly and urine output was decreased. Injection labetalol 20mg given and injection Lasix 20 mg also given as adviced by nephrologist. Bp was 170/110 mg even after labetalol injection, infusion labetalol started (50mg labetalol in 50 ml solution) @ 5ml/hr. Fetal heart rate went below 110 beat/min so emergency lscs was planned. Inj MGSO4 4g iv slowly given. Inj ranitidine and metoclopramide was given iv. Iv tranexamic acid 500mg was given, inj ceftriaxone 1gm given and pt was shifted to operation theatre. Paediatrician was informed.

All consents were checked, and patient was taken into ot at 3:10 pm. two 18 gz cannulas were secured in both forearm, basic monitoring attached like BP, SPO2, ECG, Temperature monitoring. Labetalol infusion was continued,Surgeon and assistants were asked to srub and paint drape the patient.

Base line BP- 170/102 (on labetalol infusion), HR – 88, SPO2 -99% on room air, URINE OUTPUT 20 ml (last 2 hrs), 100% 0xyegn was given for preoxygenation. General anaesthesia was planned.

At 3:20 pm patient was induced with injection fentanyl 50 mcg IV, inj thiopentone sodium 250 mg IV, injection rocuronium 35 mg IV after 1minute cuffed endotracheal tube 7.0 mm ID inserted under dicert laryngoscopy with rapid sequence induction. B/L air entry were checked tube fixed at 20 cm mark. Etco2 also confirmed tube placement. Simultaneously surgery was started. incision was made at 3:22 pm. After intubation vitals was BP 160/100mmhg, HR 92 /MINUTE, SPO2-100%, ETCO2 -28. Inj paracetamol 1gm was given for analgesia. At 3:29 PM female child was delivered with meconium stained liquor. cord was clamped and cut. Baby cried immediately after birth. Injection oxytocin 3 IU was given as IV bolus slowly and 7 units were injected in iv fluid. 10 units of oxytocin was given IM. Uterus was well contracted and closure was started. At 4:15 pm surgery was completed. Vaginal toileting and tab. misoprost 600mg intravaginally inserted. Inj. ondansetron 4mg given. After spontaneous respiration achieved injection myopyrrolate 5ml (inj. neostigmine 25mg + inj. glycopyrrolate 5mg) given. Patient was well oriented, followed command and all limb movement and neck holding was present so gently extubated. All vitals were normal except BP which was between 140 -170mmhg systolic and 95-105mmhg diastolic throughout the surgery. Postoperative BP was 160/100mmhg on labetalol infusion, HR was 90/min, spo2 was 99%. No complaint of pain was present. Input was 600ml RL and output was 15ml urine. Patient was shifted to ICU for better monitoring.

Baby weight was 2.2kg. APGAR score at 1min was 7 and at 5 min it was 9. Baby did not required oxygen support but shifted to NICU for monitoring as meconium was passed just before delivery. Baby was discharged from NICU after 24 hours.

At postoperative day1 tab nicardia 20mg BD (for 1 day only as adviced by cardiologist) was given to mother and labetalol infusion was decreased to 2ml/hr from 5ml/hr as BP started to decrease. Iv fluid (D5 + sodium bi carbonate 75ml+ 1 ml KCL) was given @ 30ml/hr alternate with inj. Kabilyte as advised by nephrologist. At postoperative day 2 labetalol was stopped. All vitals were normal, urine output was around 30ml/hr, iv fluid RL, NS, DNS were

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given alternatively @60ml/hr. both mother and baby was ok and discharged at post operative day 7 with oral medications.

DISCUSSION:

Pre-eclampsia is a multisystem disease of unknown cause, characterised by hypertension and (BP >140/90) and proteinuria (>300 mg within 24 hours) after 20 weeks of pregnancy (8). Severe pre-eclampsia is characterised by blood pressure >160/110 mmHg at rest, severe proteinuria (>300 mg/day) and oliguria (8). The main aim of management of these patient is to prevent HELLP syndrome by control rise in blood pressure, adequate supply of oxygen to mother and prevent uteroplacental insufficiency [9]. The anaesthetist should also be cautious regarding development of pulmonary oedema and coagulopathy [9]. Choice of anaesthesia in such patients depends on overall condition of the patient as general anaesthesia as well as regional anaesthesia both have effects on mother and baby. But in our case there is mitral regurgitation is also present so we preferred general anaesthesia.

Normal pregnancy results in dramatic changes to the cardiovascular system. Pregnancy produces a 30 –50% increase in blood volume and cardiac output with physiologic anaemia as a result of a greater increase in blood volume than red cell mass (10-11). The increase in cardiac output is primarily the result of an increase in stroke volume with a smaller contribution from an increase in heart rate. Pregnancy reduces systemic vascular impedance. Anaemia decreases blood viscosity with resultant decrease in systemic vascular resistance. During peripartum and postpartum period gush of blood return to circulation and increase preload so further deteriorate the valvular condition. So aim should be optimise vitals before labour and one should maintain vitals within normal range in peripartum as well as in postpartum period and general anaesthesia is perfect choice for lscs in this scenario with minimum hemodynamic changes.

Kuczkowski et al. (12) published recommendations for peripartum anaesthesia for pregnant women with valvular disease. Anaesthetic options for Cesarean section include incrementally dosed lumbar epidural or general anesthesia. In general, an incrementally dosed lumbar epidural will provide the least amount of hemodynamic alteration (12). General anaesthesia may provide very stable hemodynamics if the sympathetic stimulation associated with laryngoscopy and intubation are attenuated either by use of anaesthetic agents or beta blockade. But in our case there was an emergency situation so we preferred general anaesthesia. General anaesthesia provides the advantages of definitive airway control. A number of case reports have described the use of general anaesthesia with good maternal and fetal outcomes. Opioid-based techniques are often recommended for anaesthesia in patients with moderate to severe valvular disease because they have a minimally depressive action on the cardiovascular system and provide excellent analgesia. In the case of Cesarean section, however, this technique could result in prolonged neonatal respiratory depression. We used minimum dose of fentanyl but patient had given mgso4 before induction so it helped in attenuation of laryngoscopy response as well as decresed demand for opioid analgesia. We used isoflurane but did not required in large concentration.

Perioperative use of pulmonary artery catheters is controversial. The most recent practice guidelines for pulmonary artery catheterizations published by the American Society of Anesthesiologists (13) concluded that pulmonary artery catheterization should be reserved for patients and procedures (1) where there is a high risk of significant hemodynamic

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disturbances, and (2) in practice settings where there are competent and experienced practitioners to insert the catheter and interpret the data derived from it. Although many practitioners continue to use catheterization routinely or in specific situations (14) its use has been called into question for failure to show benefit—and even a potential for increased mortality (15). We did not use pulmonary catheter as we already discussed it was an emergency situation.

After 48 hour of placenta removal in postpartum period patients vitals were in normal range urine output was maintained because pre-eclampsia is basically disease of placental origin.

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