



## Case Report

# **Anaesthetic Management of Pregnant patient with severe mitral stenosis and Severe Pulmonary Artery hypertension for cesarean section**

Authors

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## **Introduction**

Cardiac disease in pregnancy poses a unique challenge to the obstetrician and anaesthesiologist. Rheumatic mitral stenosis forms 88% of heart diseases complicating pregnancy in tertiary referral centres in India. The mortality and morbidity are considerably reduced by better perinatal care, where anaesthesiologist plays a major role<sup>1</sup>. The case report of a patient with severe mitral stenosis (MS) and pulmonary artery hypertension (PAH) for elective caesarean section is being presented.

## **Case Report**

A 25 year lady (gravida 2, para 1, living 1) with MS and PAH (underwent balloon mitral valvotomy six years ago) at 36 weeks of gestation was planned for elective LSCS. She had a previous normal vaginal delivery four years ago which was uneventful. However during the present pregnancy at 33 weeks of gestation, she had dyspnoea at rest and was found to have severe residual stenosis of mitral valve. She was admitted at 34 weeks of gestation with worsening dyspnoea and bilateral pedal oedema suggestive of heart failure. She was managed conservatively with diuretics, beta blockers and cardiac glycosides in

the intensive care unit. On examination she was dyspnoeic, heart rate – 68/ min, blood pressure- 100/60 mm hg and Spo<sub>2</sub> of 98% on room air. Crepitations was present in left apical region. Hematology and biochemistry investigations were within normal limits. Electrocardiogram showed normal sinus rhythm. Transthoracic echocardiogram revealed severe mitral stenosis, tricuspid regurgitation and severe pulmonary artery hypertension (mitral valve orifice – 0.8 cm<sup>2</sup>, pulmonary artery systolic pressure -64 mmhg), mitral Valve pressure gradient – 41 mmhg, ejection fraction- 60%). She was on Tab Furosemide 40 mg BD, Tab Digoxin 0.125 mg OD, Tab Metoprolol 12.5 mg BD and Tab Penicillin G 400 mg BD. She was electively posted for caesarean section at 36 weeks of gestation and assessed under ASA III.

In addition to her routine medication, tab ranitidine 150 mg was given on the night before and at 6 am on the day of surgery. In the operating room, standard hemodynamic monitor of heart rate, non-invasive blood pressure, Spo<sub>2</sub> and temperature was instituted. Left radial artery cannulation was done with 20 G cannulae. After preoxygenation with 100% oxygen for 3 minutes, Induction was done with Inj Thiopentone 250 mg

and inj succinylcholine 75 mg was given to facilitate endotracheal intubation. Inj Esmolol 5 mg I V was given prior to laryngoscopy to avoid intubation response. After endotracheal intubation was with ETT 7 ID tube anaesthesia was maintained with oxygen, air, Isoflurane and intermittent doses of inj Vecuronium. A Live female baby was delivered with 1 minute APGAR score of 8/10 and 5 minute APGAR score of 10/10. Following the delivery, patient was given Inj furosemide 40 mg I V to prevent overload due to autotransfusion which could cause fluid overload and pulmonary oedema. Inj Fentanyl 100 mcg I V was given for pain relief and Inj oxytocin 20 I.U is given as continuous infusion. Methylergometrine was avoided to prevent sudden increase in afterload which could precipitate pulmonary oedema. Intraoperative period was uneventful. Heart rate and blood pressure was maintained within normal limits. Duration of surgery was 90 minutes. Neuromuscular blockade was reversed with Inj glycopyrolate 0.5 mg and Injection Neostigmine 2.5 mg I.V. Inj Esmolol 5 mg was given before extubation to prevent the hemodynamic response to extubation. Following extubation patient was shifted to Intensive care unit for observation. Heart rate, Blood pressure and saturation was continuously monitored. Postoperative pain was managed with Paracetamol and Fentanyl IV. After 48 hours of observation she was shifted to ward in a hemodynamically stable condition.

### Discussion

Mitral stenosis in pregnancy is generally less well tolerated. In Normal adult, mitral valve has an area of 4-6 cm<sup>2</sup>.<sup>2</sup>

In normal adult, mitral valve area measures 4-6 cm<sup>2</sup>. With significant mitral stenosis the increase in blood volume and cardiac output is less tolerated in pregnancy. When mitral valve is reduced to less than 2 cm<sup>2</sup>, a pressure gradient develops across the mitral valve. The magnitude of this gradient depends on the stenosis severity and the amount of blood flow across the valve. As

cardiac output increases, the gradient increases. This increase in left atrial pressure is reflected back into pulmonary vessels and increases the risk of pulmonary oedema. If untreated, this progression results in pulmonary arterial hypertension, which leads to increase in right ventricular pressures and right ventricular failure. A mitral valve area greater than 1.5cm<sup>2</sup> usually does not cause symptoms at rest. As severity of stenosis increases patients develop decreased exercise tolerance, orthopnoea, cough, paroxysmal nocturnal dyspnoea and pulmonary edema.<sup>3</sup>

Pregnancy aggravates MS because, increased heart rate decreases diastolic filling time through narrow ostium, Increased Cardiac output causes more blood to flow through the narrow orifice, Increased pulmonary blood volume causes the rise in pulmonary capillary pressure, that exceed the colloid osmotic pressure thereby increasing the chances of pulmonary oedema and autotransfusion and following the delivery of baby can convert compensatory stage into decompensatory stage.<sup>4,5</sup>

The goals for anaesthetic management of patients with mitral stenosis are to maintain low to normal heart rate (60-80/min), sinus rhythm, adequate venous return and systemic vascular resistance and prevention of pain, hypoxemia, hypercarbia and acidosis.

Problems encountered by the anaesthesiologist are as follows:

- 1) Fluctuations in hemodynamics during labour, delivery and immediate postpartum period may result in congestive heart failure and pulmonary edema. Following delivery autotransfusion may result in pulmonary edema.
- 2) Interaction with cardiac medications such as Digoxin, calcium channel blockers, diuretics and anticoagulants.<sup>6</sup>

There is no single technique which is exclusively indicated or contraindicated. The primary concern is to manage the pathophysiological changes that exacerbate the disease. Both Epidural anaesthesia and general anaesthesia has been given. Epidural anaesthesia has been tolerated by patients with moderate stenosis.<sup>7</sup> General anaesthesia is

beneficial in severe stenosis<sup>2,8</sup>. The choice of drugs used for general anaesthesia should avoid sudden drop in systemic vascular resistance. Any drop in systemic vascular resistance may be managed with phenylephrine hydrochloride.<sup>6</sup>

Opioids with a low concentration of volatile agent is a good combination for intraoperative management. Nitrous oxide can aggravate the pulmonary artery hypertension and precipitate right heart failure. So nitrous oxide was avoided. Vecuronium a cardio stable neuromuscular blocking agent is useful in these patients. Opioids can be used for induction but can cause respiratory depression of the foetus. Hence is safe to give opioids after the delivery of the baby and is beneficial in avoiding the increase in sympathetic response due to pain.

After the delivery of the baby, oxytocin 10 -20 U in 1000 ml of crystalloid should be administered at the rate of 40-80 mu/min. Oxytocin can lower the systemic vascular resistance as well as elevate the pulmonary vascular resistance, resulting in drop in cardiac output. Care must be taken during its administration. Methylergometrine or 15-methylprostaglandin F<sub>2α</sub>, produces severe hypertension, tachycardia and increased pulmonary vascular resistance. Hence it should be avoided in this patients.<sup>9</sup>

As these patients are at risk of developing circulatory overload due to autotransfusion which may precipitate pulmonary hypertension, pulmonary oedema and cardiac failure. Intensive monitoring and therapy should be continued in the postpartum period, till the haemodynamic parameters return to normal.<sup>10</sup>

### Conclusion

Rheumatic mitral stenosis complicating pregnancy is still a frequent cause of maternal death. Understanding the physiological changes due to pregnancy and the pathological state of mitral stenosis with the management of patient using a multidisciplinary team helps in avoiding perioperative complications.

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