Recently diagnosed case of severe MS posts for elective lower Segment Cessarian Section: anaesthesia challenge

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Introduction
Rheumatic heart disease is still a major health problem associated with pregnancy in India, despite its declining trend. Rheumatic mitral stenosis forms 88% of heart diseases complicating pregnancy in tertiary referral centre in India[¹]. The mortality and morbidity are considerably reduced[²] by better perinatal care, where anaesthesiologist plays a major role in the multidisciplinary approach.

Case Presentation
Patient Smt. Heena having 9 months pregnancy with RHD with severe MS with mild MR / moderate AR / mild TR with ejection fraction 60% was diagnosed at 8 month during this (second) pregnancy posted for FTLSCS. She was operated for FTLSCS under spinal anaesthesia 3.5 years back. Intraoperatively, she developed pulmonary edema and managed for that and was admitted in ICU for treatment but relative took it to somewhat like respiratory complication.

Patient had pre-operative investigations:
- Hb -9.7 gm/dl
- Platelet- 1.59 lac/cumm
- TLC- 5200 /cumm
- FBS- 90 mg/dl
- B. Urea- 22 mg/dl
- Sr. Creatinine 0.78mg/dl
- Sr. Protein 7.18gm/dl
- Sr. Albumin- 3.46 gm/dl
- Sr. Globulin-3.72 gm/dl
- Total billirubin- 0.48 mg/dl
- Conjugated billirubin-0.22mg/dl
- Unconjugated billirubin- 0.26mg/dl
- SGOT - 22 U/l
- SGPT- 18 U/l
- S. Alkaline phosphatase-118 U/l
- S. Na. -138.6 milimol/l
- S. Potassium - 3.68 millimole/l
- BT - 2 min.
- CT -3 min. 55sec.
- Echocardiography-
- AML, PML thickened sclorised, PML fixed
MVA- 1.0 cm² (severe MS), Aortic valve- 2.4 cm² (moderate AR), TAPSE- 2.1 cm, Lt. Atrium - 4.1 cm., Lt. Ventricle - 4.4 / 3.2 cm

EF - 60 %, No clot/vegetation, Mild MR, Moderate AR, Mild TR

Impression- dilated Lt. Atrium with RHD with severe MS/ mild MR / moderate AR / mild TR with EF 60%.

Patient was on B- blocker and diuretic therapy from 10 days for cardiac disease.

Patient had pre-operative vitals- BP-126/89 mm Hg
Pulse-120/ mint.
SPO₂– 98% on room air (lungs B/L clear).

Patient was taken for elective LSCS on 24.7.2018.

We decided to give the patient epidural anaesthesia with 10 ml 0.75% ropivacaine, as patient was not on any anticoagulant therapy. After giving epidural anaesthesia, patients vitals were stable (BP- 124/82 mmHg, pulse-92 bpm, Spo2- 99%).

During LSCS, patient was comfortable and patients vitals were stable (BP- 108/74mmHg, pulse- 86 bpm, Spo2- 98%). After delivery of baby, oxytocin infusion (20Uin 500 ml NS) started but uterus was flabby, there was placenta accreta, so profuse bleeding started and suddenly BP was falled (60/36mm Hg) and then to 46/30 mmHg. Immediately patient was electively intubated and inj. Noradrenaline2 ampules+500ml NS started @ 40macrodrop per min. and one unit PRBC infused. Patient’s BP improved. Due to uncontrollable bleeding, total hysterectomy was done. Intraoperative vitals werestable (on vasopressor) and patient planned for elective ventilation as patient had severe MS and major fluid shift had happened.

Postoperatively patient's BP was 108/69mmHg (on Nor Ad support) and pulse 102 /bpm and spO₂- 100%.

Patient was shifted to ICU. In ICU, she developed tachycardia (pulse 130 per min), which was gradually increasing .Urgent ECG was done which showed AF (pulse 180bpm). Cardiologist wascalled and inj. Amiodarone 150mg stat given and amiodarone infusion was started (900mg+50ml NS) @3.4ml per hour for 6 hours and then 1.7ml per hour after that. Patient mechanically ventilated via volume control mode and ABG was donein evening after 6 hrs. In ABG, there was respiratory acidosis (pH -7.2 PCO2- 56 mmHg, PO2- 476mmHg, BE - (-) 7.4 mmol/l, HCO3- 21.3 mmol/l), so ventilator settings were changed (respiratory rate increased) and acidosis was improved (pH-7.399, PCO2-27.8mmHg, PO2-169.9mmHg, BE-(-)7.6 mmol/l HCO3-17.2 mmol/l).

Patient was sedated with inj. Fentanyl infusion (50mcg/hr) and inj. Midazolam (1mg/hr) and paralysed with inj. Vecuronium (1mg/hr).Patient’s CVP was measured and IV fluids given accordingly. Routine investigations were done and second unit of PRBC was started.

On 25.07.2018 patients routine investigations were-

Hb- 10.5gm/ dl
Platelet- 1.47 lac /cumm
TLC- 16800/ cumm
RBS - 156 mg/dl
B. Urea -36.9 mg/dl
S. Creatinine -1.81 mg/dl
S. Protein- 4.86 gm/dl
S. Albumin- 2.9 gm/dl
S. Globulin- 1.9 gm/dl
Total billirubin- 1.37 mg/dl
Conjugated billirubin- 0.69 mg/dl
Unconjugated billirubin- 0.68 mg/dl
SGOT -540 U/L
SGPT- - 610U/L
S. Alkaline phosphatase - 69.21U/L
S.sodium- 140.2 mmol/l
S. Potassium - 4.73 mmol/l.

Muscle relaxant and sedation was stopped. Patient BP - 112/72 mmHg (on Nor Ad. Support @ 10 macro drop /min.), Pulse 130bpm on amiodarone infusion, spO₂- 100% on Fio2- 30%. After retiring of spontaneous respiration, patient was reversed and extubated. After 15 min. of extubation patient BP -109/70mmHg (onNorAd.Support@10macrodrop/min.), Pulse-
130bpm (on amiodarone infusion), Spo2 - 99% (on Oxygen via venti mask). After 6 hours of extubation, ABG was done, which showed adequate oxygenation.

Patient was observed in ICU. Patient BP - 96/70 mmHg (on Nor Adrenaline 8macrodrops /min.), Pulse 98 bpm (amiodarone was stopped and tab. Metoprolol XL 25 mg OD was started), SpO2 - 99% (on O2 Support). Inj. Nor Adrenaline was tapered according to BP & then stopped.

In the next morning, ECG was done which showed sinus rhythm and patient BP- 102/67mmHg, Pulse -72 bpm and SpO2 - 98 % (on room air.), so patient was shifted to ward.

**Discussion**

Stenoticvalvular disease is poorly tolerated with advancing pregnancy, owing to ability to increase cardiac output in relation to increased plasma volume\[^3\]. When mitral valve area decreases< 2 cm2, significant gradient is developed across mitral valve. The increase in left atrial pressure increases the risk of pulmonary edema that happens more dramatically in pregnancies due to increased heart rate and intravascular volume. This progression results in pulmonary arterial hypertension that may lead to increase in right ventricular pressure and to right ventricular failure. Pulmonary hypertension was associated with high maternal and fetal mortality\[^4\].

The goals for anaesthetic management of patients with MS are maintenance of an acceptable low – normal HR, avoidance of aortocaval compression, maintenance of adequate venous return and SVR and prevention of pain, hypoxemia, hypercarbia and acidosis.

In our patient, we planned for epidural anaesthesia. Major advantages of epidural anaesthesia is that it can be administered in incremental doses and total dose could be titrated to desired sensory level. This coupled with slower onset of anaesthesia, allows maternal CVS to compensate for occurrence of sympathetic blockade, resulting in a lower risk of hypotension and decreased uteroplacental perfusion. Moreover, segmental blockade spares lower extremity (muscle pump), aiding in venous return and also decreases incidence of thromboembolic events.

General anaesthesia can be second choice, as it has disadvantage of increased pulmonary artery pressure and tachycardia during laryngoscopy and tracheal intubation. Moreover, adverse effects of positive pressure ventilation on venous return may ultimately leads to cardiac failure.

**References**