

# Perioperative management of a caesarean section in a patient with isolated pulmonic stenosis and severe pre-eclampsia

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The anaesthetic management of a parturient with isolated pulmonic stenosis and severe pre-eclampsia is particularly challenging. Each of this needs to be evaluated as a separate entity and managed in unison. The challenges that we faced were compounded by the urgent nature of Caesarean section in view of equivocal NST with meconium stained liquor. This provided us with little opportunity to evaluate the present

cardiac status and the spectrum of manifestations of pre-eclampsia. Central venous and arterial access was promptly established to enable monitoring of fluid and hemodynamic status peri-operatively. Management of pre-eclampsia was initiated by the use of intra-venous antihypertensive and magnesium sulphate. General anaesthesia with modified rapid sequence intubation was expeditiously administered and this facilitated early delivery of a healthy neonate. The mother was extubated uneventfully following assessment and optimisation of the cardiac status in the ICU.

**Key Words:** Isolated pulmonic stenosis; Severe pre-eclampsia; Caesarean section; Labetalol; Magnesium sulphate.

## CASE REPORT

A 25 year old primigravida, at 36 weeks of gestation presented to the labour room with severe abdominal pain, leak per vaginum and history of breathlessness. There was a marked limitation in physical activity due to breathlessness, even during less than ordinary activity, corresponding to New York Heart Association (NYHA) class three. She was a known case of isolated moderate pulmonic stenosis diagnosed during the first antenatal check (ANC).

Physical examination revealed pallor and bilateral pitting pedal oedema. A heart rate of 110/min, blood pressure of 240/146 mm of Hg, respiratory rate of 25/min and room air saturation of 96% was recorded. On inspection, precordial pulsations were present; on auscultation, normal first heart sound, a widely split second heart sound and a systolic ejection murmur in the left upper sternal border was appreciated. There was no evidence of right heart failure and the rest of the systems were unremarkable. She was posted for an urgent LSCS in view of equivocal NST with meconium stained liquor.

Peripheral venous access, radial arterial monitoring and central venous access were established in the labour room. Initial Invasive BP (IBP) and CVP were recorded at 238/140 mm Hg and 11 cm H<sub>2</sub>O respectively. Intravenous labetalol was started to control the blood pressure and infusion was titrated accordingly. Intravenous magnesium sulphate (MgSO<sub>4</sub>) loading dose was given. Routine and relevant investigations were sent. Laboratory reports and 2D-Echocardiography couldn't be obtained in view of the urgent nature of the surgery. The patient was pre-medicated with Inj. Ranitidine 50 mg and Inj. Metoclopramide 10 mg. Inj. Ceftriaxone 1 gm intravenously was given as antibiotic prophylaxis.

After obtaining written informed consent, the patient was directly wheeled into the OT with a 15° wedge placed for left uterine displacement and NST being continuously monitored. ECG, IBP, SpO<sub>2</sub> and temperature monitors were connected.

A modified rapid sequence induction was performed with propofol in titrated doses and 1.5 mg Kg<sup>-1</sup> of succinyl choline. Airway was secured with a 7.0 sized oral cuffed endotracheal tube and ventilated on volume control mode. Anaesthesia was maintained with a 50:50 oxygen-air mixture, isoflurane at 0.5-0.8% and atracurium bolus of 0.5 mg Kg<sup>-1</sup> followed by top-ups as required.

A live female baby weighing 2.6 Kg was delivered in 3 min, with an APGAR score of 8 at 1' and 9 at 5'. Inj. oxytocin 10 units, fentanyl 2 mcg kg<sup>-1</sup> and midazolam 0.025 mg Kg<sup>-1</sup> were administered at this point. Following the delivery, IBP reduced to 160/96 mm of Hg and CVP was maintained at 13 cm H<sub>2</sub>O. Labetalol infusion was titrated accordingly and the infusion of MgSO<sub>4</sub> was continued.

Intra-operatively, 1500 ml of crystalloid was infused. The urine output was measured to be 550 ml and blood loss was estimated at 600 ml. Post-operatively, patient was electively ventilated in the Surgical Intensive Care Unit (SICU), in view of uncontrolled hypertension and the need for a formal cardiac evaluation.

2D-Echo cardiography showed severe PS (PPG of 65 mm of Hg), mild mitral regurgitation, mild tricuspid regurgitation, normal pulmonary artery pressure and ejection fraction of 60%. In view of her labile BP, she was initiated on nasogastric amlodipine 5 mg with prazosin 5 mg and labetalol was tapered. Post-operative arterial blood gas, haematocrit, serum magnesium and calcium were within normal limits. Vitals, urine output and deep tendon reflexes were monitored continuously.

The patient was ventilated for two hours and was extubated following reversal of neuromuscular blockade with Inj. Neostigmine 0.05 mg Kg<sup>-1</sup> and Inj. Glycopyrrolate 0.01 mg Kg<sup>-1</sup>. Analgesia was provided with paracetamol and morphine-patient controlled analgesia (PCA). Magnesium infusion was stopped after 24 h. Subsequent stay was relatively uneventful and BP was well controlled. She was discharged from the ICU the next day.

## DISCUSSION

Pulmonary stenosis refers to a dynamic or fixed anatomic obstruction to flow from the right ventricle to the pulmonary arterial vasculature [1]. Although most commonly diagnosed and treated in the paediatric population, individuals with complex congenital heart disease and more severe forms of isolated pulmonary stenosis are surviving into adulthood and require on-going assessment and cardiovascular care [2]. A critical component of the care of women of reproductive age involves knowledgeable preconception counselling and skilful management throughout pregnancy and delivery [3]. Such care requires both familiarity with the congenital lesions and close collaboration with high-risk obstetrical services.

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Pulmonic stenosis is relatively uncommon in occurrence as an isolated congenital defect, occurring in approximately 5/10,000 live births and accounting for 2% of CHD with a slight female predominance [1]. It is graded based on peak pressure gradient (PPG) across the pulmonary valve into mild (<36 mm Hg), moderate (36-64 mm Hg) and severe (>64 mm Hg) [4]. Many patients remain asymptomatic throughout childhood and early adulthood and pose a challenge as they present at a later stage of progression of the disease [5].

Pulmonic stenosis presents a right ventricular (RV) outflow obstruction which leads to RV hypertrophy. This is particularly important during pregnancy as these patients may be sensitive to the volume overload that ensues and may be expected to have a risk for right heart failure, atrial arrhythmias, miscarriages and pre-term birth [6,7]. There is a dearth of literature to guide the anaesthetic management of parturients with severe pulmonary stenosis.

This patient was diagnosed with isolated moderate pulmonic stenosis in her first trimester when she presented with breathlessness on moderate exertion corresponding to NYHA 2. She was advised monthly follow-up with 2D Echocardiography for monitoring the PPG with a plan to undergo palliative balloon pulmonary valvuloplasty (PBPV) if she experienced breathlessness corresponding to NYHA 3 in her second trimester. However, she failed to follow up for any subsequent ANC's.

The management of this case was particularly challenging as we were dealing with a known case of moderate pulmonary stenosis with unknown cardiac status at the point of contact. To further compound the challenge, the patient presented to us with severe pre-eclampsia with an equivocal NST and meconium stained liquor which necessitated a class 2 (urgent) caesarean section.

Anaesthetic management of a parturient with cardiac valvulopathy requires a thorough understanding of the physiology of the lesion and factors maintaining cardiac compensation. Pulmonary valve stenosis increases right ventricular work and dramatically impairs left ventricular output. This is of special concern in a parturient where an increase in cardiac output associated with labour may result in acute cardiac decompensation and right sided heart failure [5]. Also, excessive fluid administration can precipitate acute right heart failure and atrial arrhythmias. Thus, a precarious balance of volume status is required to ensure that the fixed right ventricular output state caters to an adequate left ventricular output. It is particularly important in a patient with severe pre-eclampsia where the afterload is increased and volume status is contracted. Also, while dealing with a gravid uterus preload may further decrease by the resulting aortocaval compression.

An arterial line and central venous access was established in the labour room to ascertain the volume and hemodynamic status. A 15° lateral tilt was maintained at all times to prevent aortocaval compression. It is recommended that central neuraxial blockade is best avoided in isolated PS to maintain adequate preload and also in severe preeclampsia due to a propensity to cause sudden hypotension [8]. General anaesthesia was chosen in view of urgent nature of the surgery, full stomach status and unknown cardiac and coagulation status.

The principles of haemodynamic management in pulmonary stenosis are maintenance of (a) right ventricular preload (b) right ventricular contractility (c) left ventricular afterload (d) normal sinus rhythm and (e) avoiding increase in pulmonary vascular resistance [8]. Anaesthetic management should focus on factors reducing pulmonary vascular resistance such as hypothermia, hypercarbia, acidosis, hypoxia and avoiding high ventilator pressures [8].

The goals of anaesthetic management were met by giving intravenous induction drugs in titrated doses. Anaesthesia was maintained by isoflurane in an air and oxygen mixture and nitrous oxide was avoided as it could increase pulmonary vascular resistance. Stress response to intubation was blunted by Labetalol infusion which was on-going for control of blood pressure. Analgesia was provided intra-operatively with fentanyl and post-operatively with paracetamol and morphine PCA. EtCO<sub>2</sub> levels fluctuated between 22-26 cm of H<sub>2</sub>O which was as expected in PS.

Ringer's Lactate was administered to maintain CVP at 8-12 cm of H<sub>2</sub>O. In pulmonary stenosis, fluid management is of utmost importance for maintenance of preload but care should be taken to avoid overload leading to right heart failure and acute pulmonary edema, especially in this setting of associated pre-eclampsia [6].

Management of severe pre-eclampsia was commenced concomitantly. The patient presented to us with an elevated BP recording of 240/146 mm Hg with no symptoms suggestive of impending eclampsia. A urine protein dipstick of three plus was subsequently obtained. A diagnosis of severe pre-eclampsia was made and invasive blood pressure monitoring established. Hypertensive emergency was controlled with infusion of labetalol with a plan to deliver the fetus promptly. Loading and maintenance dose of MgSO<sub>4</sub> was given as seizure prophylaxis.

Severe pre-eclampsia is a hypertensive disorder of pregnancy defined as systolic pressure more than 160 mm Hg or diastolic pressure more than 110 mmHg with proteinuria more than 5 g/24 h (urine dipstick more than three plus). Systolic blood pressure more than 180 mm Hg is defined as a hypertensive crisis and requires immediate and effective treatment in order to decrease maternal morbidity and mortality [9].

Drugs that can be safely used include labetalol (oral or intravenous), nifedipine (oral) and hydralazine (intravenous) [9]. Continuous fetal heart rate monitoring should be employed until the blood pressure is stable [9]. Particular care should be taken to avoid precipitous falls in blood pressure, which may induce maternal or fetal complications, as a result of falling below critical perfusion thresholds. Elevated blood pressure should be lowered to levels of systolic 140-150 mm Hg/diastolic 80-100 mm Hg at a rate of 10-20 mm Hg every 10-20 min.

Magnesium sulphate is recommended as prophylaxis for eclampsia in women with severe pre-eclampsia [10]. The dose of magnesium sulphate is derived from the Collaborative Eclampsia Trial regimen - 4 g bolus over 10 min followed by 1 g/h infusion until 24 h after delivery. This maintenance dose is stopped or decreased to 0.5 g/h if the patient is oliguric or if the serum magnesium levels are higher than the therapeutic range. Monitoring of MgSO<sub>4</sub> should utilise clinical parameters of urinary output, respiratory rate, oxygen saturation and patellar reflexes. Serum magnesium levels should be measured if toxicity is suspected [10].

## CONCLUSION

This report highlights the management of a known case of moderate pulmonic stenosis and severe pre-eclampsia with a favourable maternal and fetal outcome. The patient presented to us with an indication for an urgent Caesarean section. This provided us with little scope for evaluation of the cardiac status and the pre-eclamptic disease continuum. The authors recommend the following principles of management.

A thorough understanding of the dynamics of adult isolated pulmonic stenosis in pregnancy and the effects that hypertensive diseases of pregnancy may superimpose.

(b) Early and prompt establishment of invasive lines for fluid and hemodynamic status assessment with cautious interventions on maintenance of preload and gradual afterload reduction. Continuous cardiac output monitoring is recommended in such a case which provides data on systemic and pulmonary vascular resistance.

(c) Management of severe Pre-Eclampsia with expeditious control of hypertensive crisis through the use of intravenous anti-hypertensives and magnesium sulphate with an emphasis on the early delivery of the fetus.

(d) Continuous monitoring of the fetal NST with an emphasis on factors maintaining left ventricular output and uteroplacental perfusion in turn.

(e) Precise management of fluid status for maintenance of preload in PS with care being taken to avoid overload leading to right heart failure and acute pulmonary oedema, especially in this setting of associated pre-eclampsia.

(f) Central neuraxial blockade is best avoided in isolated PS to maintain adequate preload and also in severe preeclampsia due to a propensity to cause sudden hypotension.

(g) General anaesthesia is preferred especially while dealing with urgent nature of the surgery, full stomach status and unknown cardiac and coagulation status. Anesthetic management should focus on factors reducing pulmonary vascular resistance such as hypothermia, hypercarbia, acidosis, hypoxia and avoiding high ventilator pressures.

### COMPETING INTEREST

No external funding and no competing interests declared.

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