

# General Anaesthesia for Emergency Caesarian Section in a Patient with Eisenmenger's Syndrome and Pre-eclampsia

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## Abstract

Pregnancy is poorly tolerated in patients with Eisenmenger syndrome (ES) with maternal mortality of 30-50%. Physiological changes of pregnancy decreases systemic vascular resistance that further aggravates the bi-directional or right to left shunt associated with ES. When it occurs with eclampsia, the morbidity and mortality are even higher. We report a case of 30 weeks pregnant woman with ES, who underwent emergency caesarian section because of pre-eclampsia. The intra-operative course was uneventful but she died on the second post-operative day. Post-operatively she was managed by the cardiologist in the coronary care unit. The probable cause being that she was over transfused, as the fluid status was not assessed by any invasive monitoring (like CVP). It was concluded that patients should be monitored closely in the post-operative period in the intensive care unit with complete invasive monitoring for up to a week to prevent factors resulting in worsening of the shunt (such as fluid balance) and thromboembolic phenomenon.

## Introduction

Eisenmenger's syndrome includes any condition in which communication exists between the systemic and pulmonary circulations, resulting in pulmonary vascular disease characterized by elevated pulmonary vascular resistance and right to left shunting of blood through the systemic to pulmonary circulation connection.

The risk of maternal death is significantly higher (40%), in those who choose to continue with pregnancy compared to the 7% mortality that is associated with elective termination.<sup>1</sup>

Pre-eclampsia is a type of hypertension in pregnancy associated with high maternal and perinatal morbidity and mortality.<sup>2,3</sup>

We report a case of 30 weeks pregnant woman with Eisenmenger's Syndrome associated with ASD who underwent emergency caesarian section because of pre-eclampsia. Although a high risk case, it was successfully managed intra-operatively but as she was not closely monitored after surgery and probably over-transfused, it resulted in reversal of shunt and mortality on the second post-operative day. The aim of presenting this case is to raise awareness on the man-

agement of such cases.

## Case Report

A 38 years old, primigravida with Eisenmenger's Syndrome, visited the antenatal clinic for routine check in the 30<sup>th</sup> week of gestation. She was found to have a blood pressure 160/100 with albumin 3+ in urine. She was admitted after being diagnosed with pre-eclampsia for emergency caesarean section.

On pre-operative assessment, she was severely dyspnoeic, sitting propped up in bed and unable to talk because of breathlessness. Pulse rate was 92 /min, blood pressure 160/90 mmHg and respiratory rate 28/min. She was cyanosed and had clubbing of fingers, engorged neck veins, raised JVP and oedema feet. Chest was clear on auscultation.

Echocardiography showed enlarged and hypertrophied right ventricle, normal left ventricle size and function, large atrial septal defect II (ASD) (35 mm) and bi-directional shunt at ASD mostly right to left. Pulmonary arterial pressure was 85/17mmHg (normal=25/8 mmHg).

Oxygen saturation detected by pulse oximetry (SpO<sub>2</sub>) was 72% on room air and improved to 88% with the use of oxygen at a flow rate of 5 L/min via face mask. Her Hb was 16.55 gms/dl, haematocrit 49.2%, platelets 164x10<sup>6</sup>/L and urine protein 3+.

The blood pressure was controlled by intravenous hydralazine infusion at the rate of 5-10 mg/hr.

She was shifted to the operation theatre at 22.00 hours, propped up in bed. General anaesthesia was planned with invasive arterial pressure, ECG, oxygen saturation and urine output monitoring. In the operating room her BP was 130/70 mmHg, heart rate 90/min, respiratory rate 26/min and SpO<sub>2</sub> of 88%. Arterial blood gases on 5 litre O<sub>2</sub> by mask showed pH 7.450, pCO<sub>2</sub> 25.4mmhg, pO<sub>2</sub> 48.5, bicarbonate 17.8, base excess -3.9 and SaO<sub>2</sub> 86.6%. Pulmonary artery catheter was not placed as she was dyspnoeic and positioning of the patient was not possible.

With the patient in the sitting posture, after pre-oxygenated with 100% oxygen for 5 minutes, Rapid sequence induction with cricoid pressure was done with etomidate and rocuronium. She was then tilted to the supine posture for intubation. Controlled mechanical ventilation was instituted

and anaesthesia was maintained with isoflurane and 100% oxygen. After the delivery of a live baby Fentanyl 150 µg was administered and FiO<sub>2</sub> was decreased to 0.5 in air. Nitrous oxide was avoided. Throughout the operation haemodynamic parameters were maintained and O<sub>2</sub> saturation was maintained at 90-95%. At the end of the surgery pulmonary arterial (PA) catheterization was performed and parameters were CVP 8 mmHg, Pulmonary artery pressure (PAP) 60/43 and pulmonary capillary wedge pressure (PCWP) 18 mmHg.

Patient was shifted to the recovery room at 1.00 hour and ventilated overnight on a control mode of ventilation with FiO<sub>2</sub> of 0.7, respiratory rate of 12/min, and tidal volume of 500 ml. Atracurium 30 mg /hr, morphine 1 mg per hour and midazolam 0.5 mg/hr infusions were administered. Fluids were transfused to maintain CVP around 10-12 mmHg. Arterial blood gases 1 hour post-operatively showed pH 7.379, pCO<sub>2</sub> 32.4 mmHg, pO<sub>2</sub> 70.1 mmHg, bicarbonate 19.2, BE-4.3 and O<sub>2</sub> saturation 93.75. She was haemodynamically stable without any pharmacological support.

At 7.00 hours FiO<sub>2</sub> was decreased to 0.4 and sedation and muscle relaxant were held. Her O<sub>2</sub> saturation was 95-96 % on 0.4 FiO<sub>2</sub> and ABG showed pH 7.388, pCO<sub>2</sub> 34.2 mmHg, pO<sub>2</sub> 81.1 mmHg, bicarbonate 20.7, BE-2.9 and O<sub>2</sub> saturation 95.9%. She was extubated at 11.00 hours and after monitoring for three hours she was shifted to the Coronary Care Unit (CCU).

Further management was done by the cardiologist in CCU and at 14.30 hours, pulmonary arterial catheter was removed as according to the cardiologist it was misleading due to pulmonary hypertension. ECG, oxygen saturation and blood pressure were monitored. Oxygen was given 6L/min by facemask. Pethidine infusion (1 mg /ml) was used to relieve pain at a rate of 10 -12 mg per hour.

She remained stable on 1<sup>st</sup> post-operative day and maintained O<sub>2</sub> saturation between 90-95% on oxygen at 6L/min by facemask. Her labs showed Hb levels of 14.7g% with a haematocrit of 45.1% and platelet 81x10E/L. Her total fluid intake during 17 hrs after admission to CCU was 4759 ml whereas output was 732 ml. Her urine output had decreased to 20-30 ml per hour.

After 36 hours of surgery her Hb and Haematocrit levels decreased (10gm% and 30.9 respectively) but there was no obvious source of bleeding. She became dyspnoeic with respiratory rate of 23-28 per minute, BP 120/70 mmHg, and pulse 110/min and fine crepitations at both lungs bases. At this time arterial blood gases showed pH 7.351, pCO<sub>2</sub> 28.3 mmHg, PO<sub>2</sub> 29.2 mmHg, bicarbonate 15.7, BE-7.7 and O<sub>2</sub> saturation 53.9%. She was given O<sub>2</sub> at the rate of 15 l/min via face mask but her O<sub>2</sub> saturation ranged between 60-80%. Lasix 20 mg and 300 ml of packed cells were administered. O<sub>2</sub> saturation remained below 70% and dyspnoea aggravated.

She had 3 episodes of dyspnoea and severe cyanosis and on the suspicion of thromboembolism Heparin injection was given. Endotracheal intubation was performed but oxygen saturation rapidly fell despite giving 100% oxygen, followed by cardiac arrest. Cardiopulmonary resuscitation was done but was unsuccessful. The patient died 60 hours after the surgery with thromboembolism being the probable cause of death. Postmortem examination was not conducted.

## Discussion

The goal of patient management with Eisenmenger's syndrome is the maintenance of systemic vascular resistance to prevent increase in right to left shunt.<sup>4</sup> Some authors have used general anaesthesia<sup>5</sup> while others have suggested epidural anaesthesia.<sup>6</sup> Cole PJ et al.<sup>7</sup> have used incremental spinal anaesthesia for caesarian section.

General anaesthesia was planned because of two reasons. Firstly to avoid fall in systemic vascular resistance with regional anaesthesia. Secondly the patient was very dyspnoeic and was unable to lie supine for surgical positioning.

Etomidate was used to minimize the risk of fall in systemic vascular resistance. Rocuronium was instituted for rapid sequence induction as the patient was overnight ventilated. Nitrous oxide was avoided because it is a potent pulmonary vasoconstrictor.

The value of a pulmonary artery floating catheter in the management of patient with pulmonary hypertension is controversial<sup>8</sup> but it allows the clinician to detect and treat fluid overload, early changes in cardiac output, pulmonary arterial pressure and shunt fraction.<sup>9</sup>

Although, thromboembolic phenomenon has been associated with 43% of all maternal deaths<sup>10</sup>, there was no conclusive evidence for such phenomena in our patient (as no ventilation/perfusion scan was done). Thromboembolic phenomenon was suspected by the lack of improvement in O<sub>2</sub> saturation with O<sub>2</sub> therapy. Heparin was started around 30 hours after delivery with no beneficial or untoward effect.

Our patient's general condition deteriorated on the 2<sup>nd</sup> postoperative day. Her Hb decreased from 14.7 to 10.5 gm %, most likely due to haemodilution as a result of over transfusion of fluid. There was no obvious source of bleeding to account for fall in Hb.

The cause of deterioration could in all probability be over-transfusion of fluid (4759 ml intake with 732 ml output) (resulting in reversal of shunt) not managed till she developed signs of cardiac failure. This was accompanied by a rapid fall in oxygen saturation between 55-70% despite increase in inspired O<sub>2</sub> concentration.

It is concluded that in a parturient with Eisenmenger's syndrome, intra-operative course may be uneventful but these

patients should be managed during the post-operative period closely in an intensive care unit with complete invasive monitoring for upto a week.

Although the pulmonary arterial catheter does not give accurate measurements of pulmonary arterial pressure, it is useful to assess sudden changes in volume and pressure; we therefore recommend its use in the management of these patients.

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