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ANAESTHETIC MANAGEMENT OF SEVERE PREECLAMPSIA WITH PULMONARY OEDEMA AND PERIPARTUM CARDIOMYOPATHY IN A 33-WEEK DICHORIONIC DIAMNIOTIC GESTATION UNDERGOING EMERGENCY CAESAREAN SECTION: A CASE REPORT

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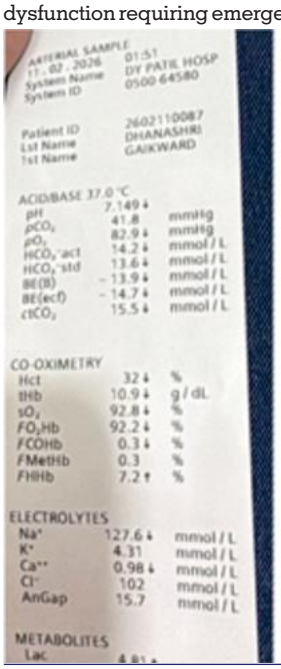
ABSTRACT

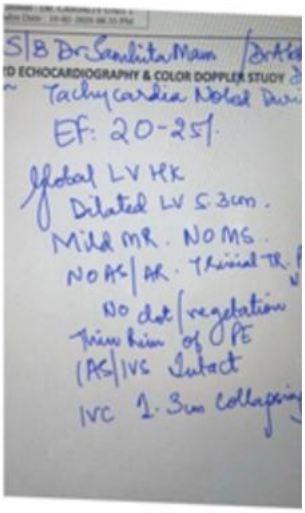
Background: peripartum cardiomyopathy is a life-threatening obstetric emergency, and presence of severe preeclampsia along with pulmonary oedema and twin gestation lead to further complications and gives significant anaesthetic challenges in a peri operative period. **Case:** 32 yr old primi at 33 week dichorionic diamniotic gestation, presented with breathlessness and bilateral lower limb swelling, after examination patient was diagnosed with severe preeclampsia, peripartum cardiomyopathy and suspected pulmonary oedema, EF was 20-25%and ABG analysis showed metabolic acidosis. patient was kept on NIV in a Preoperative period, then intubated with endotracheal tube with help of rapid sequence induction method and with use of etomidate Intraoperative management included controlled ventilation with help of rapid sequence intubation along with cricoid pressure, and fluid restriction along with intermittent suctioning for pulmonary oedema, postoperatively, patient was managed in icu with mechanical ventilation, diuretics and supportive care, which lead to significant improvement of patient **Conclusion:** coexisting of peripartum cardiomyopathy with severe preeclampsia and pulmonary oedema significantly increases perioperative risk for mother and babies. Early diagnosis, Anesthetics techniques, strict fluid managements, and multidisciplinary care important for favourable outcomes

KEYWORDS : Peripartum Cardiomyopathy; Severe Preeclampsia; Pulmonary Oedema; Twin Gestation; General Anesthesia; Perioperative Management.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of systolic heart failure that develops towards the end of pregnancy or in the early postpartum period in women without pre-existing structural heart disease. It is characterised by left ventricular systolic dysfunction and may present with acute heart failure, pulmonary oedema, arrhythmias, thromboembolic events, or cardiogenic shock. Established risk factors include preeclampsia, multifetal gestation, advanced maternal age, and hypertensive disorders of pregnancy. The coexistence of severe preeclampsia and PPCM presents unique perioperative challenges because both conditions contribute to significant haemodynamic instability and increased maternal morbidity. We report the successful anaesthetic management of a parturient with twin gestation, severe preeclampsia, and newly diagnosed peripartum cardiomyopathy presenting with acute pulmonary oedema and severe left ventricular dysfunction requiring emergency caesarean section.





Case Report

A 32-year-old primigravida at 33 weeks of gestation with a

dichorionic diamniotic twin pregnancy presented with complaints of progressive breathlessness and bilateral lower limb swelling for the past 3 days. She was a known case of severe preeclampsia.

On admission, her blood pressure was 180/120 mmHg, heart rate ranged between 120 and 150 beats/minute, respiratory rate was elevated, and oxygen saturation was 90–92% on room air. Respiratory examination revealed bilateral basal crepitations and tachypnoea, suggestive of pulmonary oedema with impending respiratory failure. In view of respiratory distress, non-invasive ventilatory support was initiated.

2D echocardiography was suggestive of severe global left ventricular hypokinesia with an ejection fraction of 20–25%, dilated left ventricular chambers and mild mitral and tricuspid regurgitation. Arterial blood gas analysis revealed a pH of 7.149, $PaCO_2$ of 41.8 mmHg, PaO_2 of 82.9 mmHg and bicarbonate of 14.7 mmol/L, consistent with severe metabolic acidosis likely secondary to tissue hypo-perfusion.

Based on clinical, echocardiographic, and laboratory findings, a diagnosis of peripartum cardiomyopathy complicated by severe preeclampsia and pulmonary oedema in a twin gestation was established. Following multi-disciplinary discussions, an emergency caesarean section was planned.

In the operating room, standard ASA monitors were attached, and two wide-bore intravenous cannulae were secured. Considering the presence of acute pulmonary oedema, impending respiratory failure, severe left ventricular dysfunction (ejection fraction 20–25%), and the urgent requirement for delivery, general anaesthesia was selected for induction of the case.

Following pre-oxygenation, rapid sequence induction was performed using intravenous etomidate and succinylcholine with application of cricoid pressure to minimise the risk of aspiration. Endotracheal intubation was achieved successfully on the first attempt, and correct tube placement was confirmed by bilateral chest auscultation and continuous capnography. Bilateral coarse crepitations were noted on auscultation following intubation, consistent with ongoing pulmonary oedema.

Mechanical ventilation was instituted using volume-controlled ventilation. Peak airway pressures were noted to be elevated, necessitating gradual optimisation of positive end-expiratory pressure (PEEP) to improve alveolar recruitment and oxygenation. Repeated endotracheal suctioning was required throughout the procedure as significant amounts of pink frothy secretions were intermittently aspirated through the endotracheal tube, further confirming severe pulmonary oedema. Intravenous vecuronium was administered for long-acting neuromuscular blockade. Intravenous hydrocortisone was administered to reduce airway inflammation and facilitate pulmonary management.

Haemodynamic goals included strict control of hypertension while preserving adequate uteroplacental and systemic perfusion. Intravenous labetalol 5 mg was administered following induction, resulting in satisfactory reduction of blood pressure. Intraoperative management focused on optimisation of oxygenation, maintenance of haemodynamic stability, minimisation of cardiac workload, and meticulous fluid restriction. Anaesthesia was maintained with low concentrations of sevoflurane in an oxygen-air mixture while avoiding excessive myocardial depression. Intravenous fluids were administered judiciously, with a total crystalloid volume of 200 mL given during the procedure. Owing to severe metabolic acidosis with evidence of ongoing haemodynamic compromise and tissue hypoperfusion, sodium bicarbonate was administered to facilitate correction of the acid-base disturbance. Diuretic therapy was intentionally deferred until after delivery and stabilisation of maternal haemodynamics. After delivery of both twins, injections fentanyl 100mcg was administered followed by intravenous furosemide in titrated boluses to facilitate diuresis and reduce pulmonary congestion.

Uterotonic therapy consisted of oxytocin 20 IU administered intravenously as a controlled infusion along with 10 IU administered intramuscularly. Intravenous tranexamic acid 1 g was administered to minimise perioperative blood loss. Estimated blood loss was approximately 1000 mL, for which one unit of packed red blood cells (250 mL) was transfused. Strict input-output monitoring was maintained throughout the procedure.

Both twins were delivered successfully with satisfactory neonatal outcomes. Maternal haemodynamics remained stable throughout the surgery without any major intraoperative complications. Considering the severity of pre-existing cardiopulmonary dysfunction and ongoing requirement for ventilatory support, the patient was transferred intubated to the Surgical Intensive Care Unit. She was electively sedated and mechanically ventilated overnight with a planned strategy for gradual weaning and extubation following optimisation of respiratory and cardiovascular status.

DISCUSSION

Peripartum cardiomyopathy is an uncommon but serious cause of maternal morbidity and mortality. The present case was particularly challenging because multiple recognised risk factors for PPCM were present, including severe preeclampsia and twin gestation. The coexistence of these conditions likely contributed to the rapid development of acute decompensated heart failure with pulmonary oedema and severe left ventricular systolic dysfunction.

The primary anaesthetic objectives in PPCM include optimisation of oxygenation, reduction of cardiac workload, maintenance of adequate organ perfusion, avoidance of fluid overload, and prevention of further myocardial depression. Although neuraxial anaesthesia is frequently preferred in haemodynamically stable patients, the presence of severe pulmonary oedema, respiratory distress requiring non-invasive ventilatory support, severe metabolic acidosis, and an ejection fraction of only 20–25% necessitated immediate airway control and ventilatory support in our patient. Under these circumstances, general anaesthesia provided the safest approach for both maternal stabilisation and urgent fetal delivery.

Induction and maintenance of anaesthesia in patients with severe left ventricular dysfunction require careful selection of drugs to minimise myocardial depression and abrupt haemodynamic changes, while taking into consideration of them crossing the placenta. Etomidate was chosen because of its relative cardiovascular stability. Vecuronium was preferred because of its ability to provide reliable neuromuscular blockade while preserving haemodynamic stability and minimal histamine-releasing properties, making it particularly suitable in the presence of severe left ventricular dysfunction and acute pulmonary oedema. Strict fluid restriction was maintained throughout the procedure, and haemodynamic management focused on controlling severe hypertension while preserving systemic and uteroplacental perfusion. Following delivery, administration of fentanyl and furosemide facilitated reduction of sympathetic stress and pulmonary congestion. The use of controlled oxytocin infusion rather than rapid bolus administration further helped avoid sudden cardiovascular compromise.

The presence of severe metabolic acidaemia (pH 7.15) reflected significant tissue hypoperfusion secondary to acute heart failure. Correction of the underlying haemodynamic disturbance remained the cornerstone of management; sodium bicarbonate was administered because profound acidaemia may further impair myocardial contractility and reduce responsiveness to endogenous and exogenous catecholamines.

Postoperative intensive care was an essential part of the management because haemodynamic deterioration may continue even after delivery due to auto-transfusion from the contracting uterus and postpartum fluid shifts. Elective postoperative ventilation in our patient allowed gradual optimisation of cardiopulmonary function before extubation.

CONCLUSION

Peripartum cardiomyopathy complicated by severe preeclampsia, pulmonary oedema, and twin gestation represents a rare but extremely high-risk obstetric emergency associated with significant maternal and fetal morbidity. Successful management requires early recognition of cardiac decompensation, prompt optimisation of oxygenation and haemodynamics, meticulous fluid management, and timely delivery. In the present case, the use of general anaesthesia with rapid sequence induction, controlled mechanical ventilation, judicious fluid administration, and elective postoperative ventilatory support facilitated a favourable maternal outcome despite severe left ventricular dysfunction. Successful management of such patients necessitates a multidisciplinary approach involving obstetricians, anaesthesiologists, cardiologists, Intensivist, and neonatologists. This case highlights that meticulous haemodynamic management, aggressive treatment of pulmonary oedema, appropriate selection of cardiostable anaesthetic agents, and planned postoperative critical care can contribute to favourable maternal and neonatal outcomes even in the presence of severe ventricular dysfunction.

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