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Management of Perioperative Pulmonary Edema in a Case of Preeclampsia for Cesarean Section

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Abstract

Although rare, pulmonary edema is a life-threatening complication occurring in preeclampsia. It is also a significant cause of maternal and perinatal morbidity and mortality. Hence, it is imperative to identify the at-risk parturient, recognize signs of critical illness and manage these patients with a skilled multidisciplinary team. We herein describe successful management of a parturient who developed pulmonary edema in a perioperative period. We emphasize the importance of a thorough pre-operative assessment, vigilance and early intervention in managing parturient with the history of preeclampsia.

Keywords: Cesarean delivery, preeclampsia, oligohydramnios, acute pulmonary edema, general anesthesia.

Introduction

Acute pulmonary edema complicating pregnancy and the puerperium results in serious maternal morbidity. It is a leading cause of death in women with preeclampsia and a frequent cause for admission to an intensive care unit (ICU) [1, 2]. Pulmonary edema may occur in up to 2.9% of women with preeclampsia with only 30% of cases occurring before delivery [3]. We describe a case report of a parturient who developed pulmonary edema as a consequence of preeclampsia and was successfully managed under general anesthesia for the emergency cesarean section.

Case Report

A 32 weeks primigravida with a history of preeclampsia, gestational diabetes mellitus was scheduled for the emergency lower segment cesarean section (LSCS) in view of severe oligohydramnios (Amniotic fluid index=4) and a poor fetal heart rate (HR) variability. She was on regular treatment with Tablet Methyldopa 250 mg thrice a day, Tablet Labetalol 150 mg twice a day, and Tablet Glucophage 500 mg thrice a day. The patient complained of a mild cough with difficulty in breathing in lying down position. Her body weight was 105 kg with a body mass index of 42 and had puffiness of the face. There was no history suggestive of obstructive sleep apnea or sleep disorder related to obesity. Her preeclampsia profile (complete blood cell count, complete metabolic profile, liver function tests, and coagulation tests) was normal. On examination, her HR was 104/min with blood pressure (BP) of 150/90 mm Hg and her 12 lead electrocardiogram (EKG) showed sinus tachycardia with mild ST depression (EKG 1.). She had bilateral pedal edema, minimal basal crepitations in both the lungs and oxygen saturation (SpO2) of 93% on room air which improved to 97% with oxygen supplement. Airway examination revealed a short neck and high Mallampati grade for which a difficult airway cart was kept ready. Due non-reassuring fetal HR, the patient was accepted for emergency LSCS without further workup and was immediately wheeled in the operating room (OR) in the lateral decubitus position. After shifting in OR, the patient started complaining of increasing breathlessness and was unable to lie down on the operating table. She had a pulse rate of 130/min, B.P. of 160/100, SpO2 of 88%, and bilateral crepitation was heard all over the lungs. Immediately high flow oxygenation by mask initiated, intravenous (IV) furosemide 20 mg was given and 30° lateral tilt provided. Cardiologist and intensivist were summoned for urgent help. Within next few minutes, the patient appeared to be severely dyspneic and cyanosed. Her oxygen saturation rapidly fell to 54% followed by a progressive drop in HR up to 35 beats/min. Immediately bag and mask ventilation commenced with 100% oxygen and rapid sequence induction was performed using IV 150 mg of propofol and IV 100 mg of succinylcholine. Direct laryngoscopy revealed frothy secretions bubbling from the glottic opening. Trachea was intubated with a 7.0 mm 'endotracheal tube(ETT)', and controlled ventilation with 100% oxygen was started which resulted in improved vitals. (HR, 136/min BP, 140/92mm Hg; SpO2, 84%; EtCO2 of 56 mm Hg; and peak airway pressure of 33 mmHg). Intermittent suctioning was done for clearing of frothy secretion. Cesarean delivery was commenced immediately for which anesthesia was maintained with 1.5% sevoflurane in 100% oxygen and IV Rocuronium 50 mg. Following delivery of baby IV fentanyl 100 µg, Midazolam 1.5 mg, and injection Oxytocin 3 IU IV were

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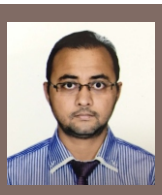
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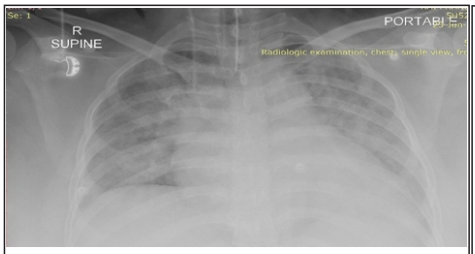


Figure 1: Bilateral pulmonary infiltrates, s/o pulmonary edema.

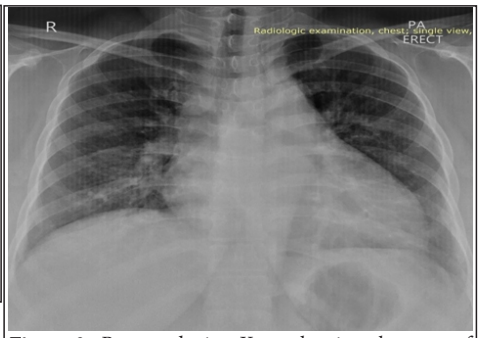
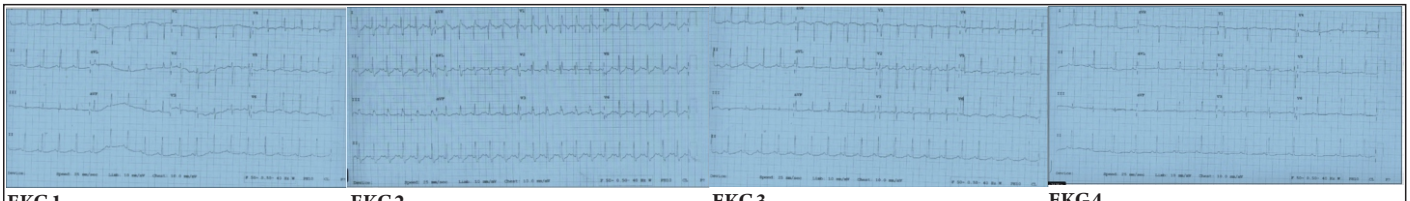


Figure 2: Post-extubation X-ray showing clearance of pulmonary infiltrates.

administered. Oxytocin infusion was maintained at 0.1 IU per min rate. Post-delivery, her peak airway pressure reduced to 25 mm Hg and SpO2 improved to 95% with a noticeable reduction in tracheal secretions. Left radial artery was cannulated for invasive BP monitoring. Her estimated blood loss was approximately 300 ml and urine output of 400 ml which was compensated with 500 mL of Ringer lactate. Initial arterial blood gases showed respiratory acidosis with hypoxemia (pH, 7.12; paO2, 68 mm Hg; paCO2, 65 mm Hg; SpO2, 78%; bicarbonate, 18 mEq/L; and base excess, −8 mEq/L) which gradually improved after controlled ventilation (pH, 7.25, paO2: 88 mm Hg, paCO2: 38 mm Hg, SpO2, 93%, HCO3 12 mEq/L, and BE – 9.8 mEq/L). The infant was intubated for a low Apgar score and shifted to neonatal intensive care for definitive care. After the completion of cesarean section, the patient was not extubated and transferred to the ICU for advanced ventilatory support. She was put on pressure control ventilation (Respiratory rate - 16/min; pressure support - 15 mm of Hg, FiO2 - 0.8 and PEEP-6 mm Hg). Initial EKG in ICU showed sinus tachycardia with right bundle branch block pattern (RBBB) (EKG 2.). Her echocardiography revealed hyperkinetic circulation, fair left ventricular systolic function - 51%, no left ventricular regional wall motion abnormality, right ventricular dysfunction with mild pulmonary arterial hypertension, and Grade 1 LV diastolic dysfunction. Troponin I assay showed initial surge (0.769 ng/ml at 3 h) followed by gradual reduction (0.627 ng/ml at 9 h and 0.552 ng/ml at 15 h) which favors a non ischemic cause for its elevation. Repeat EKG after 4 h showed sinus tachycardia, ST depression but devoid of RBBB (EKG 3). Changes in ST-T waves were corresponding with tachycardia which settled down with the reduction of HR (EKG 4). Her chest radiogram showed bilateral opacities (pulmonary infiltrates) suggestive of pulmonary edema (Fig. 1). The patient was sedated using fentanyl and midazolam IV infusion. Further management in ICU included seizure prophylaxis with magnesium infusion and nitroglycerine infusion for managing BP and reduction of pulmonary edema. IV fluids were restricted to 1–1.5 ml/kg/h. She also received diuretics, antibiotics, and prophylactic anticoagulation. Serial EKG's, cardiac markers, Echocardiography, and CT pulmonary angiography were done to rule out pulmonary embolism and cardiomyopathy. She was kept on ventilatory support for 24 h and gradually weaned off and extubated (Fig. 2). Non-invasive intermittent, continuous positive airway pressure and chest physiotherapy were provided following extubation. Both mother and neonate were discharged from hospital on the 10th post-operative day.

Discussion

Preeclampsia remains a leading cause of maternal and fetal morbidity and mortality. Pulmonary edema is now recognized as the most common final cause of death in women with complications of hypertension [4]. It may occur as a result of left ventricular dysfunction secondary to high systemic vascular resistance, iatrogenic volume overload in the face of contracted intravascular space, decreased plasma colloid oncotic pressure, or pulmonary capillary membrane injury [5]. The reduction in colloid osmotic pressure after delivery may result from excessive blood loss, fluid shifts secondary to increased capillary permeability, or excessive crystalloid infusion. Such changes help to explain at least in part why 70–80% of cases of pulmonary edema in the setting of preeclampsia develop after delivery [6, 7]. Associated clinical symptoms include breathlessness, orthopnea, agitation, cough, and signs such as tachycardia, tachypnea, crackles and wheeze, cardiac S3 gallop rhythm and murmurs, and decreased oxygen saturation. Typical chest X-ray features include upper lobe redistribution, Kerley-B, and pulmonary infiltrates. Recent studies have shown that lung ultrasound is more accurate than chest radiography in the diagnosis of pulmonary edema. It carries the advantage of avoiding irradiation in the obstetric population. Lung ultrasound can aid rapid identification of the cause of acute dyspnea and accurate assessment of pulmonary interstitial edema in the pre-eclamptic parturient [8]. Further, arterial blood gases (decreased PaO2), ECG and echocardiography may help establish the diagnosis. Management strategies for acute pulmonary edema include; reduction of left ventricular preload and afterload, maintenance of adequate oxygenation, ventilation for clearance of pulmonary edema, and prevention myocardial ischemia. Use of non-invasive ventilation avoids the complications associated with tracheal intubation in parturient who are hypertensive, such as intracerebral hemorrhage [9]. Treatment includes oxygen, diuretics, fluid restriction to achieve reduction of preload and afterload, and intermittent positive pressure ventilation [4, 5]. A multimodal treatment which ensures optimal oxygenation and stable hemodynamics with the lowest amount of fluid seemed to be justifiable. In addition,



EKG 1 EKG 2 EKG 3 EKG 4

consideration needs to be given to expedite delivery of the fetus. Women who suffer from severe preeclampsia and experience acute pulmonary edema are at increased risk of cardiovascular complications in later life, including chronic hypertension, ischemic heart disease, stroke, and renal disease [10]. They should be closely monitored with control of BP until resolution of the initial disease process and then followed up regularly, with observation for the long-term complications of the disease.

Conclusion

Managing acute pulmonary edema in antenatal period is a challenging task for anesthetist as it is associated with increased maternal and perinatal morbidity and mortality. Goals for hemodynamic optimization include reduction of the left ventricular preload and afterload. In addition, early intervention to maintain adequate oxygenation, positive pressure ventilation, and use of venodilators leads to a successful outcome.

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