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PERIPARTUM PULMONARY EDEMA AND CRITICAL CARE MANAGEMENT: A CASE REPORT

Anaesthesiology

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ABSTRACT

Pulmonary edema refers to an excessive accumulation of fluid in the pulmonary interstitial spaces and alveoli which prevents adequate diffusion of both oxygen and carbon dioxide. It may occur in low risk pregnancies but one significant predisposing factor is association with pre-eclampsia. Although an uncommon event in pregnancy, it is associated with a significant high risk of maternal and fetal morbidity and mortality. Prompt diagnosis and management is thus critically important to improve maternal and neonatal outcome. We hereby are reporting a case of severe preeclamptic patient presenting as acute pulmonary edema in peripartum period and its subsequent management.

KEYWORDS

Pulmonary edema, preeclampsia, lung protective ventilation

INTRODUCTION

Pulmonary edema is infrequent in low-risk pregnancies and accounts for about 0.05% but its incidence increases up to 2.9% of pregnancies which are complicated by pre-eclampsia with 70% of cases occurring after birth.[1] This higher incidence in pre-eclamptic patients is due to presence of generalized arterial vasospasm resulting in increased systemic vascular resistance & thus afterload, reduced plasma volume & thus preload, and increased left ventricular stroke volume index. Also, additional changes like impairment of renal function, reduced serum albumin and increased capillary permeability due to endothelial cell injury predispose these patients to an increased risk of pulmonary edema.[2,3] Worsening dyspnea and orthopnea along with signs of respiratory compromise and subsequent hypoxemia is particularly concerning and justifies intensive care. Death has been reported in around 10% of cases often reflecting multiorgan failure.[4] Immediate delivery is the safest option for the parturient and the fetus when there is evidence of pulmonary edema irrespective of gestational age.

CASE REPORT

A 26 year old, primigravida with breech pregnancy, was admitted at 36+4 weeks of POG with history of gestational hypertension with GCMF baby. Ultrasonography revealed a large septate cystic lesion in the baby measuring 9x8 cm occupying almost whole abdomen suggestive of lymphangioma. On color doppler velocimetry examination, MCA/UA pulsatility index ratio of <1 MCA/UA was found suggestive of IUGR. The poor prognosis of the baby was explained. There was pallor and bilateral pitting pedal edema on examination. All other general and systemic examination findings were normal. Investigations revealed new onset proteinuria and pre-eclampsia was diagnosed. Patient was started on Tab Labetalol 200 mg tds and regular BP charting was done. After 2 days, the patient was induced with cerviprime gel and oxytocin drip was started, to which patient suddenly became drowsy. Oxygen saturation dropped to 40%, GCS recorded at the time was E2V3M5, HR-116 per minute, BP-120/86mm Hg. ABG showed pH-7.045, pCO2-76.8, pO2-75.4, HCO3-16.2, Oxygen saturation 88%. Patient was immediately attended by anaesthesiologist and bag and mask ventilation was started. Patient was shifted to OT table on Bain circuit with oxygen cylinder. Patient was then induced with inj Thiopentone 150mg i/v and Succinylcholine 75 mg i/v and airway was secured with cuffed OETT of size 6.5mm ID. Pink frothy secretions from the ETT were noticed. On chest auscultation, bilateral basal crepitations were found. Inj furosemide 20mg i/v, inj morphine 6mg i/v, inj hydrocortisone 100mg i/v, inj dexamethasone 8mg i/v were given in stat doses in view of pulmonary edema which had developed. The IUD baby was delivered. The patient was kept intubated at the end of surgery and shifted to ICU for further management. Patient was mechanically ventilated on V-CMV mode with settings as tidal volume of 300ml, PEEP of 12cmH_2O, FiO_2 of 45% for one day and then gradually weaned off to spontaneous mode. ABG showed pH-7.40, pCO_2-32, pO_2-95.6, HCO3-24.1, Oxygen saturation-96%. After patient started following verbal commands and maintaining oxygen saturation of 95-98% on T-piece trial with normal respiratory pattern, she was extubated and put on NIV mask for a day and was finally discharged on day 4 of her ICU stay.

DISCUSSION

Acute pulmonary edema causes significant morbidity and mortality in pregnant women. It is paramount to identify the at-risk patient, recognise signs of critical illness and manage these women with a skilled multidisciplinary team. The most common causes of pulmonary edema are the use of tocolytic agents, underlying cardiac disease, fluid overload and pre-eclampsia.[5] Furthermore, the physiologic changes of cardiovascular and respiratory system associated with pregnancy may predispose the pregnant patient to pulmonary edema.[6] These known changes of pregnancy need to be given due consideration when ventilating the lungs of a pregnant or recently pregnant patient. And therefore now lung protective strategies of low tidal volumes, high PEEP with low peak pressures are advocated. Pre-eclampsia is a major cardiovascular disease of pregnancy affecting multiple systems with hypertension as its main clinical manifestation.[7,8] Gradual and not sudden reduction in systolic and diastolic blood pressure should occur at a rate of approximately 30 mmHg over 3–5 min with the end target blood pressure of 140/90 mmHg. Women who suffer from severe pre-eclampsia and experience acute pulmonary edema are at increased risk of cardiovascular complications in later life, including hypertension, ischemic heart disease, stroke and renal disease and thus appropriate long-term follow-up is necessary to reduce the chance of further complications in later life.

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