Acute Pulmonary Edema With Severe Pre-Eclampsia In Twin Pregnancy – A Catastrophic Situation

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DOI: 10.47750/pnr.2023.14.S01.90

Abstract

Acute pulmonary edema is an indicator of significant pessimism and may lead to mortality in pregnant women. It is paramount to identify the at-risk patient, recognise signs of critical illness and manage these pregnancies. Estimated reported cases in pregnancy are variable, ranging from 0.08% to 1.5%. It may occur in low-risk pregnancies but should be rule out in case of dyspnea in a pregnant woman, especially in the context of pre-eclampsia. It is a life-threatening condition of which, co-existing hypertensive disorders of pregnancy, Multiple gestation contribute to maternal and perinatal complications. Early pin point of probable cause, early risk reduction strategies along with interdisciplinary approach helps in reduction of maternal morbidity and mortality. We managed a 24-year-old G2P1L1 woman at 36 weeks DCDA twin gestation with severe pre-eclampsia presenting in emergency with acute pulmonary edema and mild pulmonary hypertension.

Key words: Acute, Pulmonary Edema (PE), Twin pregnancy, pre-eclampsia.

INTRODUCTION

Acute pulmonary edema (PE) is rare but critical issue with high feto-maternal mortality and morbidity. This is due to superimposed physiological changes in pregnancy and the presence of fetus, as well as contributory effect of poorly understood patho-physiology of pregnancy-related conditions such as pre-eclampsia [2], multiple gestation and /or underlying undiagnosed cardiac disease [3] which is associated with significant morbidity and mortality for mother and baby. Persisting hypoxemia is concerning, justifies intensive care. Death occurs mainly due to multi-organ failure. Multidisplinary approach and prompt delivery are safest options for feto-maternal outcomes, when there is evidence PE irrespective of gestational age.

CASE

A 27-year-old G2P1L1, booked ANC, 36 weeks DCDA twin with severe pre-eclampsia presented at the labour room with complaints of breathlessness at rest, fever, cough for 5 days. On examination her general condition was fair, afebrile, pulse-76 bpm, BP-170/100 mm Hg, SpO2-88% on room air,96% on 2 Litre of Oxygen. Pedal oedema was present. On auscultation of respiratory system, bilateral crepitations were present. Her cardiovascular examination was normal. She did not give history of any disease or treatment in the past.

All routine investigations were under normal limits. Patient was evaluated by the obstetrician, cardiologist, intensivist and the patient was taken for LSCS in view of Previous LSCS with DCDA twin gestation with severe pre-eclampsia with PE with mild pulmonary hypertension. Intraoperative period was uneventful. Post operatively patient was shifted to the Surgical ICU and her SpO2 maintained at 90% on 4 litre of Oxygen and was started on Antibiotics, anti-hypertensives and diuretics .All the above findings simulated severe pre-eclampsia complicated with PE with pulmonary hypertension. Further investigations revealed

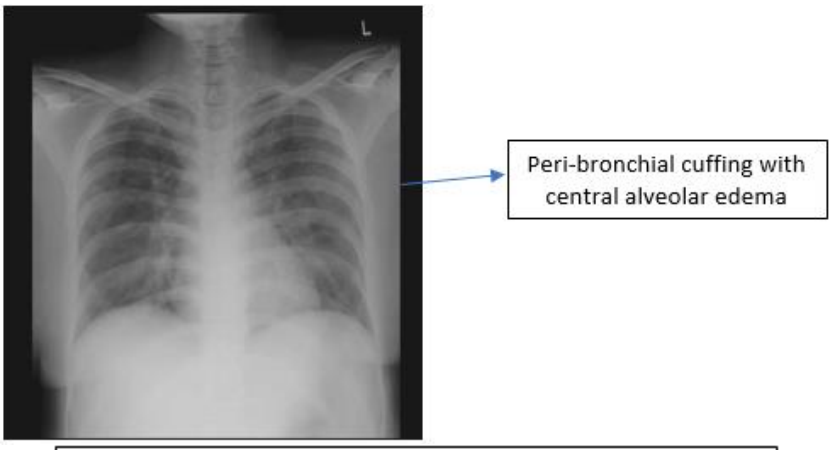

Figure-1 X RAY – Cephalization of pulmonary vessels, peri-bronchial cuffing with central alveolar edema suggestive of PE

2D ECHO- Moderate Mitral regurgitation (grade-2), Mild pulmonary artery hypertension
Patient was stabilized and was shifted on postoperative day 10 to ward and discharged.

DISCUSSION

In our case, we downrightly investigated her keeping in mind the differential diagnosis of pre-eclampsia, pulmonary embolism, sepsis, and cardiac condition as the cause for PE . Our initial suspicion was PE as a presentation of atypical pre-eclampsia. Multidisciplinary input was sought and on liaison with anaesthetics, Physician, intensivist, cardiologist and radiologist the above were excluded after normal pre-eclampsia blood and sepsis markers and the diagnosis of PE was done.

Acute PE which is a severe disease, is a leading cause of death in women with pre-eclampsia and is a main cause of admission in intensive care unit. Only 30% of cases of PE in pre-eclampsia occurs before delivery[4]. Cardiogenic and non-cardiogenic PE can be widely categorised. Early increase in cardiac output during pregnancy are followed by a peak in the postpartum period. Preload increases as a result of plasma volume expansion brought on by salt and water retention, while afterload decreases as a result of vasodilation [5].Gestational physiological changes increase woman's risk of developing PE. In a typical pregnancy, pulmonary and systemic vascular resistance both considerably decrease. Pregnant women are more vulnerable to PE because the gradient between the colloid osmotic pressure and pulmonary capillary wedge pressure is lowered by roughly 30%. If there is either an increase in cardiac preload (such as from fluid infusion) or an increase in pulmonary capillary permeability (such as in pre-eclampsia), or both, PE will be precipitated[6].

Pre-eclampsia is a multi-system major cardio-vascular disease of pregnancy causing hypoperfusion and ultimately release of inflammatory cytokines and anti-angiogenic proteins, resulting in PE. Multiple mechanisms have been proposed for pathogenesis of PE in severe pre-eclampsia includes hypervolemia, left ventricular failure, inflammatory factors[8]. In our case and 880 cases studied by Thornton et al, main potential causative factor for acute PE was underlying pathology i.e., pre-eclampsia[8] Twin gestation was additional factor in our case.

Multiple pregnancy is a risk factor for PE which needs a intensive care admission. The maternal blood volume in multiple gestation is 400ml more than singleton pregnancy. Volume overload contributes to the development of PE and after delivery, blood volume increases further and worsens PE. Inadvertent use of tocolysis[9] in twin pregnancy also contributes more to PE. The causes are usually multi-factorial i.e, majority of cases have underlying chronic hypertension and they are mostly multiparous[9] and advanced maternal age[8]. Acute PE is associated with increased IV fluid administration[8] during labour, C-section, Magnesium sulphate prophylaxis[9]. It is a clinical diagnosis mainly characterised by worsening dyspnea and orthopnea along with tachypnea, auditory crackles and rales, hypoxemia. [10]

Arterial blood gas and Chest-X ray may contribute to the diagnosis. Electrocardiography, Echocardiography, Spiral CT imaging, Ventilation/perfusion scan, pulmonary arteriography to rule out cardio-pulmonary compromise such as pulmonary embolism, pneumonia and cardiomyopathy[11]. In the cases of acute PE of cardiogenic and mixed (both cardiogenic and hypertensive) etiology, echocardiography detected some degree of ventricular dysfunction.

A protocol for the diagnosis and treatment of acute PE should include routine echocardiography at diagnosis due to its important role in identifying the specific etiology involved, particularly in this setting. Our case and Kousalya Chakravarthy et al[12], we followed similar protocol for management of PE . Complications of acute PE in a hypertensive patient and in a twin Pregnancies may complicate and leads to cardiac arrest. As previously reported in Systemic review of Yan Wang[13], timely investigation and management is a paramount to save life.

The management considerations for patients with PE is pointed out. According to present case scenario and recent systemic review, revised management protocol and early identification of risk factors for PE is pointed out. In addition, interdisciplinary approach with care co-ordination and communication among obstetrician, cardiologist, intensivist plays an crucial role for the management. Treatment include Oxygen therapy, urgent anti-hypertensive medication, fluid restriction, IV furosemide, central hemodynamic monitoring, Urgent anti-hypertensive medication and Prompt delivery is an important components.[14] Furthermore, it prevents socio-economic burden.

CONCLUSION

Acute PE is a medical emergency with high maternal and perinatal morbidity and mortality. It is paramount to identify the at-risk patient, early identification of probable cause, timely risk reduction strategies, helps in maintaining the uniformity in the management, and aids a better maternal outcome. Furthermore, this highlights the importance of the need for close liaison between obstetricians and other specialties in the management of PE during conception. The management relies on risk identification, necessary investigation, fluid management, anti-hypertensive medication and prompt delivery. Caution should also be taken while administrating fluids in pre-eclampsia to prevent PE. Appropriate long-term follow-up is necessary to reduce the chance of further complications in later life.

Declaration of Interests:

The authors have no conflict of interest to declare.

Funding:

The authors declared that this study has received no financial support.

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