Flash pulmonary oedema in obstetrics

Introduction

Flash (sudden onset) pulmonary oedema is rare in obstetrics (Euser et al, 2012). It is a medical emergency and should trigger timely, multidisciplinary intervention to minimize the risk of cardiac arrest and other adverse outcomes. This article presents a case of flash pulmonary oedema that presented as acute shortness of breath and type 1 respiratory failure in the antenatal period. Although rare, the importance of this case rests in the dramatic presentation and potentially fatal complications.

Discussion

Flash pulmonary oedema may present antenatally, intrapartum or postnatally, and occurs in up to 3% of patients with pre-eclampsia (Dennis and Solnordal, 2012). It is a contributory cause of death in hypertensive pregnancies (Moodley, 2004) and is associated with the use of tocolytic agents, increased intravenous fluid administration, cardiac disease, pre-eclampsia, intubation during general anaesthesia procedures, magnesium sulphate infusions and multiple pregnancy (Sciscione et al, 2003; Euser et al, 2012). In this case the pulmonary oedema was attributed to the patient's symptomatic pre-eclampsia, a condition that causes endothelial damage, increased peripheral vascular resistance and decreased colloidal osmotic pressure.

All differentials for chest pain were considered, including pulmonary embolism, which presents similarly with shortness of breath and desaturation in a pregnant woman. The patient had identifiable risk factors for developing a venous thromboembolic event, which were current smoker, pre-eclampsia and pregnancy. However, given her chest radiograph findings a trial of furosemide was given. There was marked improvement in oxygen saturation and symptoms post-furosemide administration, which would not be in keeping with pulmonary embolism.

Pulmonary oedema is a medical emergency and should trigger an emergency response, requiring a multidisciplinary team approach. Appropriate management is time-sensitive as further deterioration may occur, leading to cardiac arrest (Sciscione et al, 2003). Initial assessment using an airway, breathing, circulation, disability, exposure (ABCDE) approach is important to identify patients who are haemodynamically unstable. Supplementary oxygen is used to achieve satisfactory oxygen saturation and correct respiratory failure. Early epidural placement limits the requirement for increased maternal respiratory effort. Urgent and controlled reduction in high blood pressure with antihypertensives is vital to prevent further deterioration and eclampsia. The drug of choice in pre-eclampsia with pulmonary oedema is a glyceryl trinitrate infusion (Regitz-Zagrosek et al, 2011). Diuresis with intravenous furosemide is used to relieve symptoms and aid venodilatation. This is performed in conjunction with strict fluid balance to aid the offload of fluid.

With the increasing prevalence of cardiac disease and hypertensive disorders in pregnancy, it is vital for clinicians to identify at-risk patients to prevent a delay in diagnosis or mismanagement of acute pulmonary oedema. Strategies to reduce the risk of acute pulmonary oedema can also be implemented by re-evaluating fluid administration policies for women with high risk. **BJHM**

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CASE REPORT

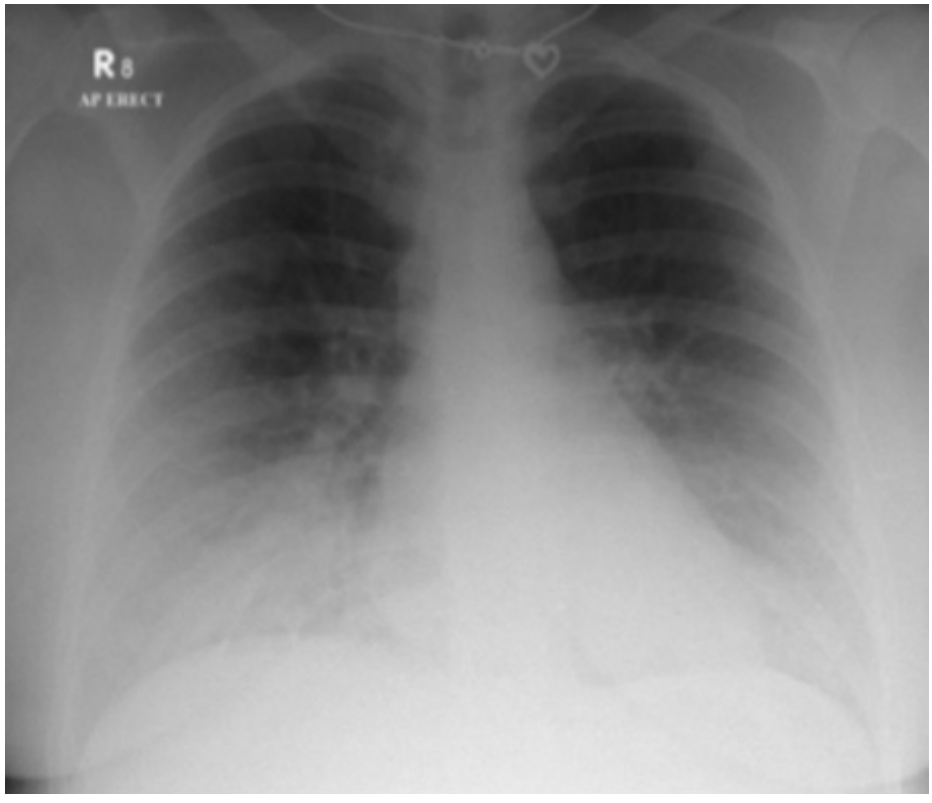
A 20-year-old fit and well nulliparous woman developed pre-eclampsia at 31 weeks. She presented to maternity triage at 32+6 weeks gestation with blurred vision and a blood pressure of 152/86 mmHg. Urinalysis revealed proteinuria and urine protein/creatinine ratio was raised at 1044 mg/mmol. Given her symptoms and raised blood pressure, she was admitted for inpatient management. Her blood pressure was controlled with regular labetalol and a single dose of nifedipine. Two days later, she developed sudden onset shortness of breath and pleuritic chest pain.

On examination, she was tachypnoeic with a respiratory rate of 26 breaths per minute and desaturating to 89% on room air. Auscultation demonstrated a reduced air entry at the bases with crackles. Arterial blood gas showed type 1 respiratory failure. Portable chest radiograph (*Figure 1*) showed bilateral shadowing consistent with pulmonary oedema. Transthoracic echocardiogram showed normal cardiac anatomy and function. Her blood tests were normal (full blood count, liver function test, urea and electrolytes) and there were no signs of fetal compromise on the cardiotocograph.

The woman was treated for pulmonary oedema with oxygen and intravenous furosemide. She was placed on fluid restriction, and given magnesium sulphate for seizure prophylaxis and corticosteroids for fetal lung maturation in preparation for imminent delivery. The patient had a marked improvement of her symptoms and her hypoxia resolved. She underwent an emergency caesarean section a few hours later under regional anaesthesia and a male infant was born in good condition.

Postnatally she made a full recovery and was discharged home on day 7. She was advised outpatient follow up by her GP to review her blood pressure control and antihypertensive requirements.

Figure 1. Portable chest radiograph revealing signs of pulmonary oedema – upper lobe diversion, Kerley B lines and pulmonary infiltrates.



LEARNING POINTS

- Flash pulmonary oedema usually presents with sudden onset breathlessness or chest pain, tachypnoea and tachycardia. A high index of suspicion is required for diagnosis as it can lead to rapid deterioration and cardiac arrest.
- Typical findings of pulmonary oedema are type 1 respiratory failure on arterial blood gas and upper lobe diversion, Kerley B lines and pulmonary infiltrates on chest radiograph.
- Treatment includes oxygen supplementation, diuresis and antihypertensives. Intensive care admission may be required.
- Pulmonary embolism remains high on the differential list as it presents similarly, but bedside tests such as electrocardiogram, chest radiograph and a trial of furosemide would aid differentiation from pulmonary oedema.

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