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Acute pulmonary oedema in late pregnancy

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Case history

An 18-year-old Asian primigravida, 34 weeks pregnant, presented to hospital with mild-to-moderate pregnancy-induced hypertension (blood pressure 160/105 mmHg) and 1+ proteinuria on urinalysis. Antenatal assessments had been hitherto normal. She was treated with intravenous hydralazine without success. Methyldopa was added to her therapy, but her blood pressure remained elevated and proteinuria deteriorated to 2+. On the third hospital day, the patient became severely breathless over a period of four hours. On examination, she was moderately distressed and tachypnoeic (34 beats/min) and had a gallop rhythm with diffuse fine inspiratory crackles in both lung bases. A chest radiograph confirmed appearances of pulmonary oedema.

Investigation results

- arterial blood gas analysis on room air: pO₂ 9.3 kPa, pCO₂ 4.1 kPa, pH 7.46, actual HCO₃ 22.1 mmol/l, O₂ saturation 94.9%
- ECG: sinus tachycardia only
- chest radiograph: normal cardiac size and contour, signs of bilateral lower zone parenchymal soft tissue shadowing and Kerley B lines consistent with pulmonary oedema
- plasma creatinine 66 µmol/l, sodium 140 mmol/l, potassium 3.9 mmol/l, serum albumin 29 g/l, total serum protein 53 g/l
- echocardiography (M-mode and 2-D): hyperdynamic left ventricle not hypertrophied or dilated. Left atrium slightly dilated (3.4 cm). No evidence of valvular disease or septal defect. Mild mitral regurgitation noted on colour flow and Doppler flow analysis
- Free T3 6.1 pmol/l (normal 4.3–7.6 pmol/l); TSH 0.14 mIU/l (normal 0.14–3.45 mIU/l)

Questions

- 1 Suggest three cardiovascular causes of acute pulmonary oedema in late pregnancy?
- 2 What is the most likely cause of pulmonary oedema?
- 3 What is the recommended management?
- 4 What is the prognosis?

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Answers

QUESTION 1

The cardiovascular causes of acute pulmonary oedema in late pregnancy are listed in box 1.

QUESTION 2

Pregnancy-induced hypertension.

QUESTION 3

The first line treatment is intravenous frusemide 40 mg or bumetanide 1 mg as a bolus dose. The aim is to achieve a diuresis of 2–3 l in the first 24 hours of diuretic therapy. A Swan–Ganz catheter is desirable in severe pre-eclamptic patients who develop pulmonary oedema antepartum. This allows the clinician to differentiate between fluid overload, left ventricular dysfunction and non-hydrostatic pulmonary oedema, the management of each being different.

Right atrial pressure (16 cmH₂O), pulmonary artery pressure (42/16 mmHg) and pulmonary capillary wedge pressure (PCWP; 24 mmHg) were all raised in this case. The serum albumin concentration dropped to a nadir of 27 g/l (total protein 52 g/l) and the plasma colloid oncotic pressure (COP) was calculated as 18.9 mmHg (normally approximately 25 mmHg). Thus, a lowered plasma oncotic pressure and a raised PCWP both contributed to pulmonary interstitial fluid accumulation and eventually pulmonary oedema. PCWP was reduced by a combination of intravenous frusemide and isosorbide dinitrate infusion. COP may be increased by infusing 200 ml of 20% albumin after achieving a satisfactory diuresis. The intravenous frusemide and dinitrate helped reduced systemic arterial blood pressure and reverse pulmonary oedema sufficient to proceed to emergency caesarian section, which is the most important measure in resolving the pregnancy-induced hypertension.

The patient was delivered of a live, but low birth weight infant (2.2 kg) without signs of neonatal distress. Postoperatively, the patient's blood pressure and PCWP settled to normal levels within 24 hours with the isosorbide dinitrate infusion, which was continued for 48 hours after delivery. The diastolic blood pressure became elevated again (100–110 mmHg) after stopping the dinitrate infusion and the patient was treated with oral slow-release nifedipine 20 mg bid. This therapy was gradually reduced and eventually stopped after three weeks.

QUESTION 4

The prognosis is generally excellent with prompt diuretic therapy and delivery, once the pulmonary oedema is controlled.

Discussion

Unexpected acute pulmonary oedema in pregnant women is uncommon and often generates suspicions of undiagnosed valvular heart disease, acute pulmonary embolism, thyrotoxic heart failure and peripartum cardiomyopathy.

Cardiovascular causes of acute pulmonary oedema in late pregnancy

- rheumatic valvular heart disease
- congenital heart disease
- pregnancy-induced hypertension
- peripartum cardiomyopathy
- hypertensive heart failure
- thyrotoxic heart failure
- beta-agonist tocolytic therapy

Box 1

The latter in particular is worrying for clinicians, since it carries a grave prognosis in approximately 25–50% of patients.¹ However, peripartum cardiomyopathy usually occurs between the last month of pregnancy and the first six months postpartum, making it an unlikely diagnosis in this patient. Pregnancy-induced hypertension is a seldom-mentioned cause of acute pulmonary oedema in previously healthy women, and should be considered in the differential diagnosis in all women with pregnancy-induced hypertension who become suddenly dyspnoeic as this case illustrates.

The patient did not receive intravenous fluid prior to the onset of pulmonary oedema, nor any beta-agonist tocolytics, which occasionally precipitate pulmonary oedema.² Beta-agonists stimulate the renin/angiotensin pathway and antidiuretic hormone release, with resultant sodium and water retention. Pulmonary oedema may occur particularly if beta-agonists (most commonly salbutamol and ritodrine) are administered in balanced salt solutions as opposed to 5% dextrose. The salt and water positive balance with tocolytics is independent of concurrently administered betamethasone or dexamthasone (to prevent foetal respiratory distress syndrome).

Sabai *et al* reported pulmonary oedema in 2.9% of a series of almost 1300 women with severe pre-eclampsia–eclampsia,³ nearly 75% of whom developed pulmonary oedema postpartum. In this series, patients with pulmonary oedema caused by pregnancy-induced hypertension were generally older multigravidas and more likely to have had long-standing hypertension. The pathophysiology of pulmonary oedema resulting from pregnancy-induced hypertension is not completely understood, but studies by Cotton *et al*⁴ and Benedetti *et al*⁵ have noted abnormal COP–PCWP gradients in patients developing pulmonary oedema caused by pregnancy-induced hypertension. Benedetti *et al*, described 10 patients with this condition, five of whom had abnormal COP–PCWP gradients. Among the other five patients, pulmonary oedema was related to increased pulmonary capillary permeability in three, and left ventricular failure in two. We calculated the COP to be 18.9 mmHg in our patient.⁶ Given that the PCWP (24 mmHg) exceeded this level of COP, it is likely that the reduced plasma albumin level, in addition to the raised afterload caused by the increased systemic vascular resistance

(as reflected by the marked diastolic hypertension), both contributed significantly to the development of pulmonary oedema. Cotton *et al*⁷ recommend a nitrate infusion in addition to conventional intravenous doses of frusemide to treat pulmonary oedema caused by pregnancy-induced hypertension. Afterload reduction with hydralazine or sodium nitroprusside may be necessary if intravenous boluses of frusemide and nitrate infusion are ineffective.

It is not clear from the literature whether albumin infusion has a role in the management of pulmonary oedema related to pregnancy-induced hypertension. However, where patients manifest heavy proteinuria as in this case, and where the COP-PCWP gradient can be clearly shown to be negative, it seems

reasonable to correct hypoalbuminaemia, particularly levels less than 30 g/l. The infusion of 200 ml of 20% albumin in this patient coincided with intravenous frusemide boluses and isosorbide dinitrate infusion. Thus, it is not certain to what degree albumin infusion may have helped improve the pulmonary oedema. Further experience of managing this condition will no doubt clarify this point.

Final diagnosis

Pregnancy-induced hypertension resulting in acute pulmonary oedema.

Keywords: pregnancy-induced hypertension, acute pulmonary oedema

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