CASE REPORT

Acute respiratory failure in a 35-year-old woman following preterm vaginal delivery

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Accepted 9 February 2014

SUMMARY

A 35-year-old woman, a non-smoker with a normal body mass index, 'felt wheezy' and developed profound hypoxia 30 min after preterm vaginal delivery at 24+ weeks of gestation. She denied other symptoms, had no fever but was tachycardic and tachypnoeic with normal blood pressure. Pulmonary embolism, amniotic fluid embolism, cardiomyopathy, arrhythmias, sepsis and non-cardiogenic pulmonary oedema were considered as differential diagnoses. Chest X-ray showed an increased pulmonary vasculature, but the blood tests, ECG, echocardiogram and CT pulmonary angiogram were essentially normal. She was managed on a high dependency area with high-flow oxygen and intravenous antibiotics. She improved dramatically and the oxygen requirements dropped to 2 L over the next 4 h. It is plausible that this woman had acute non-cardiogenic pulmonary oedema secondary to a combination of risk factors. This case highlights the importance of a methodical and multidisciplinary approach for a prompt diagnosis and successful treatment of an acutely ill parturient.

BACKGROUND

The physiological changes during pregnancy and in the puerperium cause an increase in oxygen demand. Acute hypoxia during this period is uncommon due to maternal compensation, but undiagnosed pathological causes can be life-threatening.

Owing to its multifactorial aetiology, diagnosis of pathological hypoxia can be a challenge, but with a methodical history, focused examination, appropriate investigations and multidisciplinary involvement, most causes of obstetric hypoxia can be diagnosed and successfully treated.

We report an unusual case of acute respiratory failure following immediate preterm vaginal delivery, which spontaneously resolved a few hours later with no conclusive identifiable cause.

CASE PRESENTATION

A 35-year-old woman with a history of mid-trimester miscarriage at 15 weeks was seen in the antenatal clinic at 16 weeks' gestation in her second pregnancy. She had a booking body mass index of 24 and was a non-smoker. Her medical history included treatment for symptomatic anaemia and an open abdominal myomectomy within the past year. She was not on any medications and had no allergies.

During this pregnancy, she was admitted at 24 weeks' gestation with a history of abdominal

pain with backache and incidentally found to be in threatened preterm labour. She was given antenatal corticosteroids for fetal lung maturity and magnesium sulfate for fetal neuroprotection, but was not given any other tocolytics. The woman was allowed to drink and encouraged to keep well hydrated and intravenous fluids were not necessary. The contractions settled with magnesium sulfate and 28 h after magnesium therapy, her membranes ruptured spontaneously draining clear liquor and 18 h later she delivered a live infant vaginally.

Within 30 min of delivery, she 'felt wheezy' and was found to be profoundly hypoxic. On further questioning she denied any other symptoms, and on examination she was alert and orientated, without fever, tachycardic with a pulse rate of 120 bpm, urine dipstick negative, blood pressure of 120/70 mm Hg, tachypnoeic at 24 breaths/minute, normal jugular venous pressure, auscultation of her heart showed normal heart sounds and her chest revealed fine bibasal crepitations.

INVESTIGATIONS

The arterial blood gas analysis was suggestive of hypoxia (saturation of 88% on room air with a partial pressure of oxygen of 6.8 kPa). A chest X-ray (figure 1) showed increased pulmonary vasculature and an ECG showed a sinus tachycardia. A CT pulmonary angiogram (CTPA; figure 2) showed no evidence of pulmonary embolism (PE) but dependent air space opacities consistent with acute respiratory distress syndrome or non-cardiogenic pulmonary oedema. An echocardiogram showed normal valve morphology and good biventricular function. Blood tests were requested (including full blood count, urea and electrolytes, C reactive

Figure 1 Anteroposterior chest X-ray immediately after the onset of acute respiratory failure.

To cite: Bhandari HM, Gorecha M, Woodman J. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-203676

Figure 2 CT pulmonary angiogram showing no evidence of pulmonary embolism but dependent air space opacity consistent with acute respiratory distress syndrome or non-cardiogenic pulmonary oedema.

protein, liver function test and clotting profile), which were all normal. The placental histology subsequently showed chorioamnionitis with early funisitis.

DIFFERENTIAL DIAGNOSIS

A 35-year-old woman developing acute hypoxia in the immediate postnatal period following a few days of relative immobilisation and a preterm delivery may have PE, amniotic fluid embolism, cardiomyopathy, arrhythmias, sepsis or a non-cardiogenic pulmonary oedema (table 1). She had no chest pain or shortness of breath and the CTPA ruled out the possibility of a PE. ECG ruled out arrhythmias and echocardiogram ruled out any other cardiac causes. Absence of chest pain and shortness of breath, hypotension, normal coagulation profile and a spontaneous resolution were against amniotic fluid embolism.

In our case, it is plausible that the most likely explanation suggests acute non-cardiogenic pulmonary oedema secondary to a combination of suggested aggravating factors. These factors include spontaneous preterm labour, magnesium sulfate and corticosteroid administration and chorioamnionitis (see the Discussion section).

TREATMENT

She was managed on a high dependency area and was treated with 60% humidified high-flow oxygen. She was started on intravenous antibiotics as sepsis was one of the differential diagnoses. Over the next 4 h, she subsequently improved dramatically and oxygen requirements dropped to 2 L on nasal specs (with saturations of >95%).

Figure 3 Anteroposterior chest X-ray taken 40 h after excludes any other pulmonary pathology.

OUTCOME AND FOLLOW-UP

The patient subsequently made a full recovery and a repeat chest X-ray (figure 3) 40 h following this acute episode was clear. She had a postnatal follow-up at 6 weeks and had no residual symptoms.

DISCUSSION

Pulmonary oedema complicates 0.08–0.5% of pregnancies,^{1 2} but can occur in up to 7% of cases following spontaneous preterm delivery between 24 and 33 weeks.³ According to the Scottish Confidential Audit of Severe Maternal Morbidity Report 2008, pulmonary oedema is the fourth most common cause of severe maternal morbidity in pregnant women.⁴

A retrospective case-control study conducted by Ogunyemi³ identified spontaneous preterm labour (OR 10.9), antenatal corticosteroids (OR 2.3) and magnesium sulfate (OR 5.3) administration as independent risk factors for increasing the risk of developing acute pulmonary oedema. A recent systematic review and meta-analysis, though, did not find any significant increase in the risk of pulmonary oedema and respiratory distress with magnesium sulfate prophylaxis for neuroprotection in preterm labour.⁵ Samol and Lambers⁶ elegantly demonstrated that the mean length of duration from the beginning of magnesium sulfate treatment to identification of pulmonary oedema was found to be 1.96 days, and in this case it was 70 h. They also demonstrated that women who received larger amounts of fluids at the onset of magnesium sulfate therapy and those who

Table 1 Common causes of postpartum hypoxia

Common causes of acute postpartum hypoxia		
With pulmonary oedema		
Cardiogenic	Non-cardiogenic	Without pulmonary oedema
Pre-existing cardiovascular disease (congenital, arrhythmias, valvular and hypertension)	Worsening underlying pulmonary condition (asthma)	Pulmonary embolism
Ischaemic heart disease	Iatrogenic—fluid overload	Amniotic fluid embolism
Peripartum cardiomyopathy	Acute respiratory distress syndrome	Lower respiratory tract infection—pneumonia
Sepsis—heart failure	Drugs: tocolysis, magnesium sulfate, corticosteroids and carboprost	Foreign body aspiration
Pre-eclampsia	Thyroid disease	Psychogenic

had a more positive balance on day one were significantly more at risk of pulmonary oedema.

Any disruption of Starling's forces (hydrostatic pressure, oncotic pressure and capillary permeability) surrounding fluid movement across capillary membranes will influence the development of pulmonary oedema (reviewed in ref. ⁷). Wilkens *et al*⁸ discovered that magnesium sulfate and β -sympathomimetic tocolytic agents, when administered simultaneously, increased the risk of pulmonary oedema. It is suggested that these agents increase hydrostatic pressure within the pulmonary vasculature, subsequently resulting in the passage of fluid within the interstitium.⁶

Yeast *et al*⁹ demonstrated that intravenous magnesium sulfate for preterm labour or pre-eclampsia caused a decreased colloid osmotic pressure and increased the risk of pulmonary oedema.

Also, women with clinical chorioamnionitis are found to be at a three times higher risk of pulmonary oedema. This finding underpins the possible mechanism of increased capillary permeability secondary to underlying infection.³

Contributors HMB conceived the idea and drafted the manuscript. MG contributed extensively to the idea and the drafting of the manuscript. JW was involved in overall supervision and advised on the drafting of the manuscript and critical appraisal.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- ▶ Although pulmonary embolism, cardiac failure and lower respiratory tract infection are more common causes of acute respiratory failure in the postnatal period, other possible causes should be sought and ruled out.
- ▶ Pulmonary oedema is common after spontaneous preterm delivery, particularly in women who have received antenatal corticosteroids and magnesium sulfate.
- ▶ This case highlights the importance of a methodical and multidisciplinary approach for a prompt diagnosis and successful treatment of a medical condition in the acutely ill parturient.

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