CASE REPORT

Fulminant antenatal pulmonary oedema in a woman with hypertension and superimposed preeclampsia

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SUMMARY

An asymptomatic 40-year-old para 1 black African woman with pre-existing hypertension and a booking blood pressure of 120/80 mm Hg, was admitted with superimposed preeclampsia diagnosed because of worsening hypertension and significant proteinuria at 27 +5 weeks gestation. Antenatally, her blood pressure was controlled with labetalol, and blood tests including serum creatinine were within normal limits for pregnancy. Three days later, the patient developed severe hypertension despite treatment, and reported sudden onset severe shortness of breath; oxygen saturations on air dropped to 93%. Auscultation revealed widespread crepitations leading to a working diagnosis of pulmonary oedema. Despite appropriate management, respiratory function continued to deteriorate and she required intubation, ventilation and emergency caesarean section under general anaesthesia. A live male infant was delivered floppy and was intubated and resuscitated. He awaits discharge home on oxygen. The mother's pulmonary oedema resolved postpartum. Echocardiogram showed left ventricular hypertrophy but normal left ventricular function and the patient's hypertension is being controlled on medication.

BACKGROUND

Hypertensive disorders are the most common medical complication of pregnancy, and preeclampsia has an incidence of around 2–8% of all pregnancies in the UK. It remains a leading cause of maternal and perinatal morbidity and mortality globally, with about half of women with severe preeclampsia giving birth preterm.¹ Pulmonary oedema is a well-recognised complication of preeclampsia and is reported to occur in about 2–7%^{2–4} of patients with severe preeclampsia or eclampsia. However, the majority of cases reported occurred postpartum,^{2 4 5} and were often related to fluid overload. This case was unusual because the pulmonary oedema developed acutely with neither hypoalbuminaemia nor intravenous fluids in the antenatal period in a patient who was previously asymptomatic. National Institute for Health and Care Excellence recommends admission to hospital when preeclampsia is diagnosed and this is often justified on the basis of the risk of eclampsia or placental abruption. This case highlights the additional risk of fulminant pulmonary oedema particularly in those women with pre-existing hypertension.

CASE PRESENTATION

The patient booked at 10+1 weeks of gestation, with a history of raised blood pressure (BP) in her previous pregnancy in 2004. She had not received any treatment for this at the time, but was started on antihypertensive medications at delivery, which she continued for a month postpartum. She did not recall being diagnosed with preeclampsia or having been prescribed any medications since. Her previous baby, a boy, was delivered at term by emergency caesarean section due to failure to progress after an induction of labour for raised BP. He weighed 4180 g. The mother suffered from multiple fibroids and had a family history of type 2 diabetes mellitus. She was taking no medications and had no known drug allergies. She had six visits to the antenatal clinic after her booking visit, and was started on 200 mg two times a day of labetalol and 75 mg of aspirin as preeclampsia prophylaxis at her second visit following a BP reading of 144/92 mm Hg and 2+ protein on urine dipstick. In addition, 200 mg once daily of ferrous sulfate was started in view of her anaemia (102 g/L), as well as vitamin D and calcium supplements. She was also having weekly BP measurements at her general practitioner (GP). At 19+5 weeks, she presented to the day assessment unit reporting right iliac fossa pain and, after relevant examination, was diagnosed as having fibroid pain and was discharged. Until her admission 3 days prior to delivery, her BP ranged 120–144/80–92 mm Hg during pregnancy and she remained symptom free with normal assessments except for intermittent proteinuria (table 1 and figure 1). Protein creatinine ratio (PCR) was measured on each occasion where the urine dipstick was positive for protein.

The patient presented to the day assessment unit 3 days prior to delivery after referral from her GP, who was concerned about her raised BP. On arrival, her BP was 172/101 mm Hg, so she was admitted and received 20 mg STAT of oral nifedipine and the labetalol was increased to 200 mg three times a day. She was also given two doses of 12 mg intramuscular dexamethasone 24 h apart for fetal lung maturation. Her urinary PCR was 149 mg/mmol. During the next few days, her BP was labile and ranged 138–177/86–111 mm Hg. Except for a headache, reported once on the day of admission when her BP rose to 177/109 mm Hg, she remained symptom free throughout the admission. During this admission to the antenatal ward, the patient was also admitted to the obstetric high dependency unit twice for poor control of her BP. On day 3, after a shower, she reported acute shortness of

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Table 1 Booking bloods and sequential blood results

Item	Units	30/1/15 Booking	4/3/15	15/5/15	3/6/15 Admit to ward	6/6/15 Predelivery	6/6/15 Postdelivery	7/6/15 24 h post
WCC	$\times 10^9/L$	4.3	4.6	5.6	7.1	13.6	12.1	7.8
RBC	$\times 10^9/L$	3.93	4.04	3.94	3.83	4.08	3.15	2.71
Hb	g/L	102	110	116	115	123	95	83
MCV	fL	81	85	93	92	93	93	95
Platelets	$\times 10^9/L$	238	204	188	169	163	144	118
CRP	mg/L						16	50
Protein creatinine ratio	mg/mmol	N/A	81	12	149			
Bilirubin	$\mu\text{mol/L}$		3	<2	<2	4	4	<2
ALT	iu/L		13	19	18	27	23	37
ALP	iu/L		55	74	82	92	65	58
Albumin	g/L		40	37	34	35	26	26
eGFR	mL/min/m ²		95	82	67	74	59	57
Creatinine	$\mu\text{mol/L}$		61	69	82	75	92	95

ALP, alkaline phosphatase; ALT, alanine transaminase; CRP, C reactive protein; eGFR, epidermal growth factor receptor; Hb, haemoglobin; MCV, mean corpuscular volume; RBC, red blood cell; WCC, white cell count.

breath, saturations on air dropped to 93% and her BP measured 180/111 mm Hg. On examination, she was found to have widespread crepitations and a STAT dose of 10 mg of oral nifedipine was administered. This was followed by 20 mg of intravenous furosemide. She was then given glyceryl trinitrate spray, catheterised and started on 15 L/min of high flow oxygen. Between this time and going to theatre, she was given a total 100 mg intravenous of furosemide, 200 mg intravenous of labetalol and 4 g of magnesium sulfate.

INVESTIGATIONS

The patient's booking bloods, sequential blood results and BP measurements preadmission are given in [table 1](#) and [figure 1](#). A chest radiograph ([figure 2](#)) taken at the bedside showed widespread interstitial shadowing consistent with pulmonary

oedema. An echocardiogram was performed, which showed left ventricular hypertrophy, indicating that the patient had long-standing hypertension. However, it also showed a normal ejection fraction (>55%) excluding peripartum cardiomyopathy as a cause of the pulmonary oedema. Additionally, it excluded mitral stenosis or other valvular heart disease as a cause.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for the sudden onset of shortness of breath included pulmonary oedema, pulmonary embolism, acute respiratory distress syndrome due to sepsis and pneumonia.

Once the clinical diagnosis of pulmonary oedema had been reached, differential diagnosis of the cause included peripartum cardiomyopathy and mitral stenosis, or a condition related to preeclampsia.

TREATMENT

The patient's hypertension was treated with labetalol antenatally. Following admission with a diagnosis of preeclampsia, the labetalol dose was increased. Episodes of severe hypertension were treated with nifedipine. Low-dose aspirin (75 mg once daily), and calcium and vitamin D were prescribed from early pregnancy as preeclampsia prophylaxis, and 200 mg once daily of

Figure 1 Antenatal blood pressure (BP) trend.

Figure 2 Mobile anteroposterior (AP) chest radiograph showing widespread interstitial shadowing consistent with pulmonary oedema.

ferrous sulfate was given to treat anaemia. Following the patient's admission, dalta-parin was given as a thromboprophylaxis. The treatment of pulmonary oedema is not altered by pregnancy, so she was treated with oxygen, diuretics and antihypertensives.

OUTCOME AND FOLLOW-UP

The pulmonary oedema resolved postpartum and the patient's hypertension was treated with 10 mg once daily of amlodipine and 10 mg two times a day of enalapril. She continued to take 200 mg two times a day of ferrous sulfate for persistent anaemia. The BP remained well controlled and the patient was discharged following a long postnatal stay because of pain and fever resulting from fibroid degeneration. She was followed up in the obstetric medicine clinic where it was confirmed that the proteinuria persisted 8 weeks after delivery.

The patient has been informed about the likelihood of pre-existing hypertension ($\pm$ chronic kidney disease) and the need for lifelong medication. If the proteinuria persists at 12 weeks she will be referred to a nephrologist to investigate the underlying cause of this. There was also a discussion about the significant risks of any future pregnancies and she has been advised to seek preconception counselling to optimise her condition and arrange close monitoring throughout the pregnancy. Her son remains in the special baby care unit, awaiting discharge on oxygen via nasal cannulae for his chronic lung disease diagnosed at 28 days of age. He received three packed red cell transfusions for anaemia of prematurity, had jaundice requiring 6 days of phototherapy and his unconjugated hyperbilirubinaemia is resolving.

DISCUSSION

Hypertensive disorders (preeclampsia and eclampsia, gestational hypertension and chronic hypertension) are often cited as some of the most common medical complications of pregnancy.^{6–10} It is also known that women with chronic hypertension are at risk of developing superimposed preeclampsia and such women have higher rates of adverse maternal and fetal outcomes.¹⁰ Furthermore, superimposed preeclampsia develops in 13–40% of pregnant women with chronic hypertension.⁸ The additional risk factors for developing acute pulmonary oedema that our patient fulfilled were obesity and increased maternal age, factors that were highlighted in the review article by Dennis and Solnordal.¹¹ The development of preeclampsia predisposes to subsequent cardiovascular disease and some authors view preeclampsia itself as a primarily cardiovascular disorder.¹²

The incidence of pulmonary oedema in severe preeclampsia or eclampsia is reported as 2–7%^{2–4} with reviews and case series^{2–4–5} reporting a higher incidence postpartum versus antepartum. Our case was unusual in light of these studies in that it occurred in an asymptomatic pregnant woman in the absence of tocolytic use or over-zealous fluid administration. The risk of postpartum pulmonary oedema in women with preeclampsia is well known and drives the use of fluid restriction in their management, and has reduced maternal deaths.¹ However, we feel that the risk antenatally (although more unusual) is an underappreciated complication.

Indeed, a recent case-control study showed that almost half ($n=13$) of 28 women with preeclampsia and pulmonary oedema developed the condition antenatally,¹³ perhaps suggesting that the incidence of antenatal pulmonary oedema may be increasing. While the reasons still remain unclear, this may be related to the rising prevalence of chronic hypertension in the general pregnant population.¹⁰ Our case underscores the need

for clinicians to recognise the importance of admitting pregnant women with preeclampsia not only because of the risk of placental abruption or eclampsia but also due to the risk of pulmonary oedema.

Patient's perspective

I consented for this report to be published on the grounds that it would help clinicians learn from my unusual presentation. I feel very fortunate to have survived the event and to be able to take home my son in the near future, albeit on oxygen. The event has made me realise the importance of managing my hypertension and the need to make lifestyle changes for the longer term. Despite the significant risks that have been explained to me, I am still keen to have a future pregnancy and will seek pre-pregnancy counselling with the obstetric medicine team at the appropriate time.

Learning points

- Fulminant pulmonary oedema can occur in asymptomatic patients with preeclampsia.
- Spikes in blood pressure in a pregnant patient should be treated aggressively to prevent acute pulmonary oedema as well as cerebral haemorrhage though aggressive blood pressure treatment may not always prevent complications.
- Proteinuria with hypertension in pregnancy should be followed up carefully.
- Women with pulmonary oedema should have echocardiography performed to determine if the cause is cardiac in origin.
- Patients with preeclampsia should be admitted not only because of the risk of placental abruption or eclampsia but also due to the risk of pulmonary oedema.
- Staff caring for such patients should be trained in the recognition and treatment of pulmonary oedema.

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Competing interests None declared.

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Reminder of important clinical lesson

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