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CASE REPORT

Undiagnosed phaeochromocytoma mimicking severe preeclampsia in a pregnant woman at term

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SUMMARY. We report an unusual case of phaeochromocytoma in pregnancy. The patient presented with severe hypertension, visual disturbances, proteinuria, glycosuria and pulmonary oedema at 38 weeks' gestation. The initial diagnosis was severe preeclampsia, but rapid deterioration of the fetus necessitated an emergency caesarean section under general anaesthesia, following which the maternal condition deteriorated rapidly. Differential diagnoses included pulmonary embolus, cardiomyopathy, amniotic fluid embolus and ischaemic/embolic cerebrovascular accident. Despite aggressive maximal treatment, mother and baby died 36 h later. Post mortem examination of the mother revealed a 5.5-cm tumour of the right adrenal gland confirmed histologically as a phaeochromocytoma. We examine the diagnostic dilemmas of this case and consider the treatment and management options when faced with a critically ill mother and the need to deliver her fetus by emergency caesarean section. We also question the clinical priorities during management of a sudden deterioration in both maternal and fetal health.

Keywords: Phaeochromocytoma; Pregnancy; Maternal death; Diagnosis; Obstetric; Anaesthesia; Complications; Anaesthetic techniques

INTRODUCTION

Phaeochromocytoma is a dangerous condition, particularly in pregnancy, when it is difficult to diagnose, uncommon and has often been confused with preeclampsia. Such diagnostic dilemmas can present obstetricians and anaesthetists with major challenges. The following case report exemplifies some of the difficulties that phaeochromocytoma presents in pregnancy and highlights its severity.

CASE REPORT

A 40-year-old woman in her 38th week of pregnancy was referred as an emergency to the delivery ward by her general practitioner. This was her 5th pregnancy with the same partner, having previously had four normal vaginal deliveries. She was normally fit and well and the current pregnancy had been uneventful. Her weight was 65 kg at booking (12 weeks' gestation). She complained of general malaise, vomiting, palpitations and headache of approximately 6 h duration and had noticed decreased fetal movements during the day. Before admission, she was apyrexial, with a blood pressure of 190/80 mmHg (measured using a dynamic non-invasive blood pressure monitor and checked manually) and a pulse rate of 72 beats/min. Her blood glucose was 12.3 mmol/L. A urine dipstick test showed glucose, protein and blood in her urine. A glucose tolerance test at 28 weeks' and urinalysis at 36 weeks' gestation had been normal.

On admission to the delivery ward her blood pressure was 204/110 mmHg (mean arterial pressure (MAP) 141 mmHg). She was not in established labour. After assessment by the consultant obstetrician, a provisional diagnosis of severe preeclampsia was made and she was started on our unit's treatment protocol, comprising a restricted i.v. fluid intake of 85 mL/h, hourly urine measurements, continuous cardiotocograph (CTG) monitoring and 4-hourly preeclampsia blood tests (full blood count, urea and electrolytes, liver function tests, urate

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and coagulation screen) and hydralazine to be given if the MAP exceeded 125 mmHg. The obstetric registrar was called to review the patient as the MAP was 123 mmHg. On physical examination there was no evidence of abdominal tenderness, hyperreflexia, peripheral oedema or clonus. Fundoscopy was normal. The cervix was noted to be thick, 1.5 cm long and 2 cm dilated. The head was engaged and noted to be at station −2. The membranes were ruptured artificially. Ten minutes later the patient was reviewed and the CTG was stable. The plan was to contact the anaesthetist to request epidural analgesia once the blood results were available. Twenty-five minutes later a severe, persistent fetal bradycardia (60 beats/min) prompted the call for an emergency caesarean section. At this stage the anaesthetist was notified about the patient for the first time. No hydralazine had yet been administered.

The patient was very agitated but insisted on walking to the operating theatre, where she complained of visual disturbances. Anaesthetic assessment in the theatre revealed no significant past medical history. She was unable to lie in the wedged supine position due to breathlessness and was noted to have mottled skin. Her initial arterial oxygen saturation (SpO_2) on air was 88%, which improved to 99% following pre-oxygenation. Her blood pressure was 160/100 mmHg (MAP 120 mmHg). In view of the profound and sustained fetal bradycardia, a decision was made to proceed to general anaesthesia using pre-oxygenation and a rapid sequence induction. Thiopentone (375 mg) and suxamethonium (100 mg) were given. Pink, frothy fluid was seen in the vocal cords at intubation, diagnosed as frank pulmonary oedema. She was initially tachycardic (160 beats/min) and hypertensive (160/90 mmHg). A male infant in very poor condition was delivered by lower segment caesarean section (induction to delivery interval 3 min). Umbilical cord blood gases showed an umbilical venous pH of 6.735 and an umbilical arterial pH of 6.595. Oxytocin 5 units was administered as a slow intravenous bolus on delivery of the baby, following which the blood pressure dropped to 60/30 mmHg. Resuscitation with vasopressors (ephedrine total 30 mg and epinephrine total 2 mg) and fluids (Hartmann's solution 1 L and Hetastarch 6% 1.5 L) restored the blood pressure to 140/90 mmHg. Central and arterial cannulae were inserted. The central venous pressure (CVP) was +8 mmHg and two boluses of furosemide 20 mg were given. The total blood loss was approximately 700 mL. She remained intubated and ventilated and was transferred to the intensive care unit (ICU) on an FiO_2 of 1.0 and PEEP of 15 cm H_2O.

Examination in the ICU revealed the following: blood pressure 125/85 mmHg (MAP 98 mmHg), pulse rate 153 beats/min (sinus tachycardia), CVP +12 mmHg. She was cold and mottled and anuric. She was ventilated via synchronized intermittent mandatory ventilation (respiratory rate 15 breaths/min, tidal volume 700 mL, using an Evita® ventilator). A chest X-ray showed pulmonary oedema and initial arterial blood gas analysis revealed a severe metabolic acidosis: (FiO_2 0.9), pH 6.9, P_aO_2 13.7 kPa, P_aCO_2 4.1 kPa, base excess −22.6 mmol/L, lactate 11.5 mmol/L. The results reported by the laboratory are given in Table 1.

Persistent vaginal bleeding resulted in a fall of Hb concentration to 7.1 g/dL. An oxytocin infusion was started and carboprost was given. Four units of packed red cells and fresh frozen plasma (10 mL/kg) were infused and an aprotinin infusion started. A sudden decrease in blood pressure was followed by cardiac arrest. Cardiac massage for 6 min, epinephrine 7 mg (total), 10% $CaCl_2$ (20 mL) and 8.4% $NaHCO_3$ (100 mmol) resulted in a degree of cardiovascular stability (blood pressure 130/90 mmHg and pulse rate 148 beats/min). An epinephrine infusion (4 mg in 5% dextrose 50 mL) was started at 19 mL/h and titrated according to the arterial pressure. Supportive treatment was continued. Urine and blood toxicology screens (including cocaine and amphetamines) were subsequently negative. A comprehensive septic screen was performed on admission to ICU, although nothing was cultured.

At this stage there was still no formal diagnosis. Differential diagnosis included preeclampsia, pulmonary embolus, cardiomyopathy, amniotic fluid embolus and ischaemic/embolic cerebrovascular accident. All preoperative blood results had been normal, except the

Table 1. Blood results

Hb	7.9 g/dL
Platelets	205×10^9/L
WBC	20.4×10^9/L
PT	14.6 s
APTT	92.7 s
APTT ratio	3.2
INR	1.25
Na^+	139 mmol/L
K^+	5.7 mmol/L
Urea	4.1 mmol/L
Creatinine	100 μmol/L
Urate	210 μmol/L
Bilirubin	7 μmol/L
Albumin	16 g/L
ALP	99 units/L
ALT	27 units/L
AST	60 units/L
LDH	686 units/L
Ca^{2+}	1.96 μmol/L
Corrected Ca^{2+}	2.41 μmol/L
PO_4^{2-}	3.96 mmol/L
Mg^{2+}	0.94 mmol/L
Total CK	546 units/L
CK-MB	111 units/L (fraction 20%)
Troponin-I*	64.6 μg/L

*The optimum discrimination value for myocardial infarction at 12–14 h is 1 μg/L.