A case of breathlessness during pregnancy: the difficulty in diagnosing heart failure

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Summary: We present the case of a 33-year-old woman in her second pregnancy who was transferred to our unit following a one-month history of worsening fatigue and a three-day history of worsening symptoms of heart failure. Shortly after presentation she developed ventricular fibrillation and arrested. At an emergency caesarean section a placental abruption was noted and the baby was stillborn, unable to be resuscitated. The patient required a prolonged intensive and coronary care stay. Echocardiographic findings were consistent with dilated cardiomyopathy and as all investigations to ascertain a cause were negative she was diagnosed with peripartum cardiomyopathy. Her case highlights a potential fatal cause of breathlessness during pregnancy and the role of B-type natriuretic peptide to assist in the differential diagnosis of these cases.

Keywords: cardiac, cardiology, cardiovascular, high-risk pregnancy, intensive care medicine

INTRODUCTION

Breathlessness during pregnancy is a common complaint and is often normal. There are however sinister causes of which medical professionals need to be aware. This case highlights the catastrophic consequences of breathlessness caused by heart failure during pregnancy and the need for prompt diagnosis and treatment.

CASE REPORT

A 33-year-old Caucasian woman who was 31⁶ weeks pregnant with her second child presented to hospital with a one-month history of increasing lethargy and nausea, followed by three days of worsening shortness of breath, orthopnoea and a non-productive cough. She had been recently treated with antibiotics for a suspected pneumonia by an after-hours doctor. On the day of admission she felt cold and clammy and had been unable to pass urine. Her past medical history included obsessive compulsive disorder, predominantly manifested as hypochondriasis, for which she took clomipramine. Her previous pregnancy was uneventful. She reported no family history of cardiac disease or sudden death.

On arrival at the emergency department she was cyanosed, cold and mottled in colour with severe respiratory distress. Oxygen saturation was unrecordable. She was tachycardic and hypotensive with a mean arterial pressure of 35–40 mmHg. The fetal heart was not heard. The provisional diagnosis was sepsis due to worsening pneumonia.

She was intubated but developed ventricular fibrillation requiring 75 minutes of cardio-pulmonary resuscitation before spontaneous circulation returned. At an emergency caesarean section a placental abruption was noted and the baby was stillborn, unable to be resuscitated. A bedside echocardiogram showed mild to moderate biventricular dilation with severe impairment; left ventricular ejection fraction (LVEF) 25% (normal range >50%). There was no significant valve pathology or pericardial effusion.

The patient was commenced on inotropes, vasopressors and levosimendan (a calcium sensitizer used to increase cardiac contractility), with empirical antibiotics and antiviral therapy. A provisional diagnosis of peripartum cardiomyopathy (PPCM) was made and based on recent guidelines bromocriptine was commenced.¹ She required continuous veno-venous haemofiltration for anuria. Hypothermia was induced for neuroprotection and the patient was transferred to our unit for cardiovascular intensive care.

On transfer her electrocardiogram (ECG) demonstrated anterolateral Q waves (Figure 1) with paroxysmal atrial flutter. Severe biventricular dilation and impairment with a LVEF of 10–15% was confirmed by transoesophageal echocardiography. She was treated with multiple inotropes and inhaled nitric oxide. Diagnostic coronary angiography, cardiac biopsy and viral screen for pathogens associated with myocarditis were all normal.

During her third day on the unit, ischaemic areas developed on her right foot and the toes of both feet. Pulses were absent on both examination and Doppler study of the right foot, but were present on the left. She was commenced on a therapeutic heparin infusion as a thromboembolic cause was suspected.

Over the next 12 days the patient was weaned off inotropes and became euvolaemic following haemofiltration. Repeat echocardiography on day 23 of admission showed an

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Figure 1 Admission electrocardiogram

improvement in cardiac function with LVEF 28%, severe left ventricular (LV) dilation, moderate right ventricular (RV) dysfunction and dilation, moderate mitral regurgitation and normal pulmonary pressures. The Q-waves on her ECG had also resolved and small anterior R-waves were present, consistent with her impaired left ventricle. By three months after admission the LVEF had improved to 33% with moderate LV dilation and trivial mitral regurgitation. The RV dilation and dysfunction had also improved (Figure 2).

She had a full return of cognitive function, mild bilateral upper limb proximal muscle weakness consistent with man-in-barrel syndrome² and a healing caesarean scar. She was discharged from hospital on day 29 with improving renal function; however, she had persistent ischaemia of her toes (Figure 3) requiring amputation and treatment for osteomyelitis. Her LVEF continues to improve but is still moderately impaired six months following her delivery. She remains under long-term psychological, cardiac and vascular care.

DISCUSSION

Here we outline the key issues arising from the case, particularly regarding breathlessness in pregnancy, before outlining how the diagnosis of PPCM was made. Finally we address the treatment and long-term management options.

As pregnancy progresses, patients often complain of an increased respiratory rate due to progesterone stimulation of the central respiratory centre and altered sensitivity to CO₂.³ The gravid uterus reduces diaphragmatic movement and there is an increase in metabolic demand which can create the sensation of breathlessness. While in most cases this symptom is physiological, the clinician needs to maintain vigilance for the development of cardio-respiratory disease. In our patient, for example, her breathlessness was significantly more severe than expected, with orthopnoea, a cough and profound

fatigue, which should alert the discerning physician to a more sinister cause such as cardiac failure.

PPCM has recently been comprehensively reviewed following publication of the European Society of Cardiology (ESC) guidelines.^{1,4,5} It is the most likely diagnosis in our patient because she fulfils three out of four of the ESC diagnostic criteria; severe LV failure, LVEF <45% and no identifiable cause. The fourth criterion is more contentious as PPCM by definition presents from 36 weeks gestation (four weeks prior to delivery). At delivery our patient's gestation was 31 weeks.

The differential diagnosis also included an undiagnosed dilated cardiomyopathy (DCM). The absence of symptoms during or after her first pregnancy, her gestation week at presentation (DCM tends to present at 22–28 weeks gestation when cardiac output and blood pressure increases most rapidly) and lack of family history of the disease make this less likely.

Other differentials include myocarditis or a stress-induced cardiomyopathy (Takotsubo cardiomyopathy) due to placental abruption as recently reported.⁶ However her cardiac biopsy was normal making myocarditis less likely and she did not have the typical echocardiographical findings or brisk improvement in cardiac function seen with stress-induced cardiomyopathy.

While echocardiography is required to diagnose PPCM, it would be impractical to image all pregnant women presenting with breathlessness. B-type natriuretic peptide (BNP) is a biochemical marker used in the diagnosis of heart failure and is becoming more generally available. While pregnancy itself causes a small rise in BNP,⁷ the negative predictive value of a BNP value <100 pg/mL has been validated in pregnancy in a small study.⁸ Had our patient's BNP been checked at her first presentation, her heart failure may have been identified and treated in time to prevent loss of the fetus and her subsequent cardiac arrest.

Figure 2 Echocardiogram: end-diastolic images from Day 14 of admission (left side) compared with Day 23 (right side) showing reduction in diastolic dimensions consistent with improving cardiac function

Figure 3 Ischaemic feet

Treatment of PPCM in the acute setting includes diuretics, inotropes, antiarrhythmics and vasodilators to maintain cardiac output. The use of bromocriptine may aid with recovery of cardiac function and its use should be considered.⁹ Patients with a poor LVEF or placental abruption are at risk of thromboembolism. Bromocriptine may aggravate this risk and thus prophylactic anticoagulation should be strongly considered. Earlier initiation of anticoagulation may have prevented our patient's ischaemic toes which still require ongoing treatment six months later.

Long-term management is with standard heart failure medication (Angiotensin-converting enzyme (ACE) inhibitors, beta-blockers and diuretics) but prior to delivery diuretics, hydralazine, nitrates and β_1 beta selective blockers are preferred to reduce adverse fetal effects, particularly from ACE inhibitors.

Provided the patient is haemodynamically stable, the pregnancy should continue to term with delivery based on obstetric indications. Our patient underwent emergency caesarean section during her cardiac arrest to maximize her chances for survival as spontaneous circulation was not restored quickly, she was more than 20 weeks pregnant and the fetus was presumed dead due to the lack of fetal heart rate on admission.¹⁰

Our patient is very keen to have further pregnancies. Current guidelines suggest that pregnancies can be considered in patients whose LVEF has normalized, with appropriate counselling and close monitoring for a recurrence.¹ In this case, due to the critical nature of her presentation and severity of cardiac dysfunction, we have advised against further pregnancies and have discussed other options including surrogacy with her.

CONCLUSION

This case illustrates the potential catastrophic consequences of heart failure during pregnancy and the diagnostic challenge it presents. Knowledge of the underlying diagnosis has the potential to change management, particularly with regard to commencement of medical therapy in PPCM. Therefore all efforts to make an accurate diagnosis should be made. Obstetricians and physicians should strongly consider the use of BNP in unwell mothers to exclude a diagnosis of heart failure.

DECLARATIONS

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