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Case Report

Peripartum cardiomyopathy complicating major postpartum haemorrhage with pre-eclampsia and HELLP syndrome

Ahmed Ali Kindawi^{1*}, Shamma Al-Inizi²

¹James Cook University Hospital, Middlesbrough, United Kingdom

²Department of Obstetrics and Gynaecology, Sunderland Royal Hospital, South Tyneside and Sunderland NHS Foundation Trust, Sunderland, United Kingdom

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*Correspondence:

Dr. Ahmed Ali Kindawi,

E-mail: ahmed.kindawi1@nhs.net

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ABSTRACT

Peripartum Cardiomyopathy (PPCM) is a rare, potentially life-threatening cause of heart failure, developing in the later stages of pregnancy, or immediately postpartum. Diagnosis can become challenging as obstetric emergencies may provide differing explanations for cardiovascular collapse. We demonstrate a rare case of acute severe biventricular failure occurring very soon after an emergency Caesarean section for a pregnancy complicated by HELLP syndrome in the setting of post-partum pre-eclampsia and severe postpartum haemorrhage. The diagnostic value of an urgent echocardiogram in an obstetric emergency is underlined and the challenges faced by clinicians in managing two serious medical emergencies at the same time are highlighted.

Keywords: Peripartum cardiomyopathy, HELLP syndrome, Pre-eclampsia, Postpartum haemorrhage

INTRODUCTION

Cardiac disease is a leading cause of indirect maternal death in the UK¹. Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy which is characterised by heart failure due to severe impairment of the left ventricle around the time of delivery.² Though the definition of PPCM no longer requires left ventricular dilatation, it does require a decrease in left ventricular function to less than 45% as defined by measurement of left ventricular ejection fraction (LVEF).^{2,3} There are overlapping features of PPCM and normal pregnancy as well as the postpartum period, thus clinical recognition of PPCM can be challenging.

CASE REPORT

A 38-year-old primigravida with well-controlled Type 1 diabetes, who had a consultant-led antenatal care and was regularly seen in the obstetric endocrine antenatal clinic. She had a well-controlled blood sugar, and fetal growth

scans were unremarkable. She had a normal BMI and was a nonsmoker with no other medical disorders. She presented at 38 weeks gestation, with severe frontal headache, normal blood pressure, deranged liver and kidney function, with no proteinuria. (ALT 548 IU/l, GGT 78 IU/l, alkaline phosphatase 197 IU/l. Creatinine was 119 µmol/l with an estimated GFR of 50 ml/min/1.73m² and normal platelets of 180).

A pathological fetal CTG prompted emergency caesarean delivery. She subsequently sustained major post-partum haemorrhage, requiring Bakri balloon tamponade with 4 units of blood transfused. A sudden drop in platelets thereafter with gradually rising blood pressure and developing proteinuria raised concern for post-partum pre-eclampsia with HELLP syndrome. She proceeded to develop reduced consciousness and became hypotensive, progressing to a peri-arrest event requiring adrenaline, with cardiogenic pulmonary oedema. She was rapidly resuscitated and transferred to CCU (coronary care unit). An urgent transthoracic echocardiogram showed severe

biventricular systolic impairment, with a left ventricular ejection fraction of 20%, but no structural or valvular abnormality. Following intensive-care stabilisation, she was extubated the next day and commenced on bisoprolol and ramipril. She continued to gradually recover and was discharged home 4 weeks post-natal. Cardiac magnetic resonance imaging at six weeks demonstrated normal ventricular function, with sustained recovery. The patient remained well on long-term heart failure therapy and was counselled regarding the risk of recurrence of PPCM and the need for cardiac review before any future conceptions.

DISCUSSION

The presence of severe biventricular dysfunction which occurred post-delivery supported a diagnosis of PPCM. However, HELLP syndrome, major postpartum haemorrhage and cardiovascular shock could provide alternative explanations. Hypertensive disorders are recognised associations of PPCM; however, their relationship remains uncertain.² This case illustrates the importance of urgent echocardiography, particularly in cases of acute cardiovascular deterioration coinciding with obstetric emergencies. Differentiation from other causes of heart failure in the postpartum period is also necessary. Differential diagnoses for a cardiorespiratory acute event in the peripartum period include pulmonary embolism, acute coronary syndrome, myocarditis, sepsis, amniotic fluid embolism, fluid overload, hypertensive emergency causing pulmonary oedema, stress-induced cardiomyopathy and shock of hypovolaemic or haemorrhagic origin.²⁻³ A significant proportion of women with hypertensive disorders in pregnancy develop PPCM, but the underlying pathophysiological link remains elusive.^{3,4,5}

In this case, the diagnosis of PPCM was made as the patient met the key phenotypic features used to diagnose PPCM. New heart failure occurring at the end of pregnancy or soon after delivery, severe left ventricular systolic dysfunction and the absence of any pre-existing structural heart disease are the key features used to make a diagnosis of PPCM.^{1,2} Right ventricular dysfunction also occurs in a proportion of women with PPCM and may even define a more severe form of the condition with poorer prognosis but with potential for complete recovery.⁶ While PPCM is considered a distinct clinical entity, PPCM is a diagnosis of exclusion. There are several other medical conditions which can cause acute myocardial dysfunction. The patient in this case had several insulting factors active at the time of her cardiac deterioration including pre-eclampsia/HELLP syndrome and major postpartum haemorrhage. While stress-induced cardiomyopathy was considered, echocardiograms demonstrated a global biventricular impairment pattern rather than a classic regional wall motion abnormality. However, stress-induced cardiomyopathy and PPCM can coexist in some cases.^{7,8} The patient's condition was not solely due to PPCM but also was affected by several other medical conditions such as severe pre-eclampsia, massive post-

partum haemorrhage and HELLP syndrome. Absence of proteinuria at first presentation does not rule out pre-eclampsia.⁹ However, it is crucial to understand the patient's post-partum rise in blood pressure and related organ dysfunction at first presentation. For this patient, it appeared that she had developed a hypertensive disorder immediately post-delivery with pre partum features of HELLP syndrome given the marked rise in transaminases, development of acute kidney injury and a drop in platelet count following delivery. There is evidence that PPCM can resolve completely with time and that return to normal can occur in up to 70% of cases at 12 months post-event.¹⁰ Late gadolinium enhancement cardiac magnetic resonance (CMR) is also increasingly used in PPCM for identification of other causes of heart failure and myocardial damage.¹¹ The management of this patient involved the resuscitation of the shock status by the multidisciplinary team with the management of the cause of her heart failure. Once delivery had occurred and she was stabilised, she was started on bisoprolol and ramipril for her newly diagnosed case of heart failure.¹² The patient's management involved a multidisciplinary team of obstetricians, cardiologists, anaesthetists and intensive care physicians. It is known that women with PPCM can have relapses of heart failure in subsequent pregnancies, particularly if left ventricular function has not returned to normal hence, she was properly counselled regarding the recurrent risk of this condition. It is important to note that not all collapses following postpartum haemorrhage will be cases of PPCM. They should however prompt consideration of cardiac involvement in the acute situation. Bedside echocardiography can be used to quickly establish severe biventricular dysfunction and in turn alter resuscitation and strategy for longer term management in the critically unwell patient. We believe this case is the first reported case of acute PPCM following a major postpartum haemorrhage complicated by pre-eclampsia with HELLP syndrome.

CONCLUSION

PPCM is a rare but serious cause of maternal morbidity and mortality. The major challenge in this patient is the rapid severe biventricular failure coexisting with major postpartum haemorrhage and post-natal preeclampsia with HELLP syndrome. Multidisciplinary input with urgent resuscitation and echocardiography and rapid transfer to CCU resulted in the full recovery of this patient.

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