

A case of cardiac arrest caused by peripartum cardiomyopathy

圍產期心肌病引致心動停止的個案

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Many women experience dyspnoea, orthopnoea, and peripheral oedema during pregnancy, and diagnosing cardiac problem is confounded by these signs and symptoms of normal pregnancy. Peripartum cardiomyopathy (PPCM) is a rare life-threatening cardiomyopathy of unknown aetiology that occurs between the last month of pregnancy and the first 5 months postpartum in previously healthy woman. Multiparity, twins, advanced maternal age, preeclampsia, gestational hypertension, and black race are known risk factors. The course of PPCM can range from readily treatable to acutely fatal. We present a lady with dyspnoea in the peripartum period admitted in cardiac arrest as a result of PPCM. (*Hong Kong j.emerg.med.* 2010;17:272-275)

很多婦女懷孕期間會有呼吸困難、端坐呼吸及末梢水腫；但因這些正常懷孕的徵狀而擾亂了心臟問題的診斷。圍產期心肌病是罕有的威脅生命的心肌病，病因不明，在先前健康婦女懷孕最後的一個月至產後的首5個月間發生。多產、雙胞胎、高齡產婦、先兆子癇、妊娠期高血壓及黑人種族為已知的風險因素。圍產期心肌病的病情範圍可以從容易治理以至急劇死亡。本文描述一名圍產期呼吸困難的女士，因圍產期心肌病引致心動停止而送院。

Keywords: Cardiomyopathies, cardiovascular pregnancy complications, heart arrest, postpartum period

關鍵詞： 心肌病、妊娠心血管併發症、心動停止、產後期

Introduction

Many women experience dyspnoea, orthopnoea, and peripheral oedema during pregnancy. As a result, the diagnosis of cardiac problem is complicated by the fact that signs and symptoms of normal pregnancy may be suggestive of heart disease. Peripartum cardiomyopathy (PPCM) is a rare life-threatening cardiomyopathy of

unknown aetiology, and the diagnosis of PPCM is often delayed even if symptomatic. Delayed diagnosis can be associated with increased morbidity and mortality.

Case

In November 2009, a 36-year-old previously healthy female, gravida 1, para 1 (full term live triplets), presented to the emergency department (ED) in cardiac arrest 15 days postpartum. According to the description of her husband, she complained of severe dyspnoea beginning late in the third trimester, and was thought to be within the normal spectrum. She received Caesarean section. On postpartum day 3, she developed fever, sore throat, cough, and mild dyspnoea and was diagnosed to have H1N1 infection, and put on Tamiflu® for 5 days. However, 2 days before the

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ED presentation, she developed increasing orthopnoea, paroxysmal nocturnal dyspnoea, and worsening dyspnoea on exertion. The prenatal history was notable for normal blood pressure and no proteinuria. Family and social histories were unremarkable.

At the time of ED presentation, the patient was in cardiac arrest, with pulseless electrical activity (junctional rhythm at rate of 48/min) for which she was resuscitated with standard Advanced Cardiac Life Support procedures. Five minutes after resuscitation, the patient had return of spontaneous circulation (ROSC). After ROSC, her vital signs were: blood pressure 180/100 mmHg, pulse rate 140 beat/min, and body temperature 36.0°C. The physical examination revealed rapid heart beats and bilateral diffuse rales. Laboratory tests, including haemoglobin, platelet count, white cell count, blood chemistry, urinalysis and cardiac enzyme were normal. The chest radiograph showed cardiomegaly and bilateral consolidation consistent with pulmonary oedema (Figure 1). Emergency echocardiogram revealed enlarged left ventricle (LV) and reduced LV systolic function with ejection fraction (EF) of 22-26%, and concentric left ventricular hypertrophy with global LV hypokinesia. The electrocardiogram demonstrated sinus tachycardia. In consultation with the cardiology department, the patient was admitted to the cardiology intensive care unit. She was placed on cardiac monitor with ventilator, and given intravenous diuretic. Given her predisposition to clot formation in the setting of a low EF, she was given enoxaparin, digitalis and angiotensin converting enzyme (ACE) inhibitor to improve the cardiac output. On the following day, the echocardiogram revealed an EF of 37%. On the 3rd hospitalisation day, she was extubated and her mentality became alert. On the 4th hospitalisation day, the echocardiogram revealed an EF of 45%. On the 15th day, the EF was 54% and the chest radiograph showed clear lung fields (Figure 2). She was discharged on day 17 without complication or neurological deficit and put on digitalis, ACE inhibitor, diuretic, and beta-blocker. Six months later, the echocardiogram revealed an EF of 56% and the chest radiograph showed clear lung fields.



Figure 1. Chest radiograph showing pulmonary oedema and cardiomegaly.

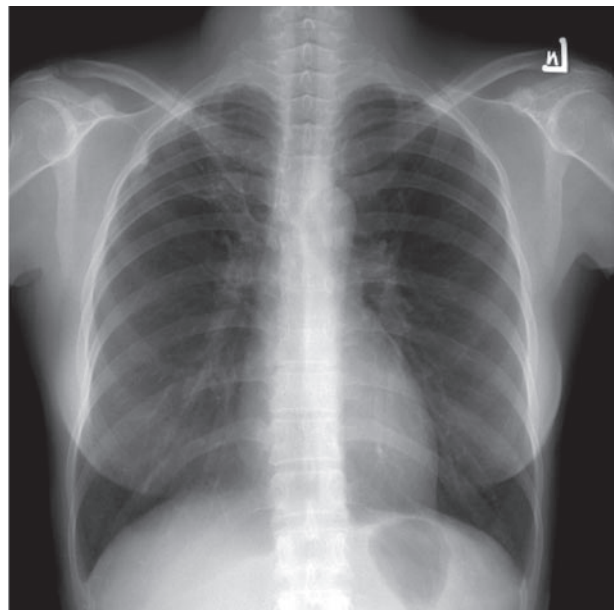


Figure 2. Normal chest radiograph 15 days after admission.

Discussion

PPCM is defined as a syndrome of cardiac failure occurring between the last month of pregnancy and the first 5 months postpartum, without obvious cause and prior evidence of heart disease. PPCM can be classified in the family of dilated cardiomyopathies. As women with dyspnoea may present to the ED, it is important to recognise the association of cardiac failure and pregnancy as a separate syndrome (like PPCM). PPCM should be treated promptly and aggressively. Although the precise aetiology for PPCM is unknown, there is much current interest in genetic factors, viral infection and autoimmune disease that may play a role.¹ Multiparity, twins, advanced maternal age (age >30), preeclampsia, gestational hypertension, and black race have been described as risk factors for PPCM.² Additionally, an association has been reported between prolonged tocolytic therapy and development of PPCM. In particular, twin pregnancy has greater haemodynamic perturbations, greater hormonal change, and greater demand on nutritional reserves, all of which could be involved in the pathophysiology of PPCM.^{3,4}

The diagnosis of PPCM should be considered whenever a patient presents with even mild symptoms of heart failure between the last month of pregnancy and the first 5 months postpartum. However, patients in the last months of pregnancy represent a particular diagnostic difficulty because of the common symptoms produced by pregnancy: dyspnoea, fatigue and pedal oedema. Symptoms and signs that should raise the suspicion of heart failure include paroxysmal nocturnal dyspnoea, chest pain, nocturnal cough, new murmurs, pulmonary crackles, elevated jugular venous pressure and hepatomegaly.^{5,6} A chest radiograph may show cardiomegaly, pulmonary oedema, and pleural effusion.⁵ Typical echocardiography findings are left ventricular ejection fraction less than 45% or M-mode fraction shortening less than 30%, or both and increased end-diastolic diameter greater than 2.7 cm/m². Echocardiography is now the standard non-invasive technique for evaluating cardiac function. Thus, it is an important tool in properly diagnosing PPCM and

predicting the prognosis. The prognosis of women who develop PPCM varies, with an alarmingly high mortality rate of 20-50%.^{3,7} In those who survive, 50% will have improvement of left ventricular function, and of these women, 10% to more than 50% will have complete recovery.⁷⁻¹⁰

The peripartum patient with severe preeclampsia can also present with dyspnoea as a manifestation of PPCM. Hypertension, non-dependent oedema, thrombocytopenia, headache, and proteinuria are the main clinical findings in preeclampsia. Although the common aetiologies of dyspnoea are asthma, anaemia, and pneumonia, there are diseases more unique to the peripartum period. Because pregnancy will result in a hypercoagulable state, immediately life-threatening diseases such as pulmonary embolism must be excluded. Although less common, acute myocardial infarction in pregnancy or the early postpartum period occurs in approximately 1/10,000 pregnancies and is associated with an overall mortality of 21%.¹¹

The treatment of PPCM is similar to that for other forms of heart failure. The classic treatments of congestive heart failure are bed rest, low salt intake, diuretics, digitalis, and anticoagulants. The goals of treatment are reducing preload and after load, and increasing contractility. Angiotensin converting enzyme inhibitors are usually used to reduce after load by vasodilatation, but they should only be considered after delivery because of potential toxic effects on the foetus. Hydralazine should replace ACE inhibitors during pregnancy. Beta-blockers have been shown to improve survival in patients with dilated cardiomyopathy. Digitalis may help to maximise contractility and rate control, and diuretics are used to reduce the preload and relieve symptoms.

Conclusion

In postpartum patients with mild cough and dyspnoea, it is important to recognise the association of cardiac failure and pregnancy and PPCM should be ruled out promptly. The diagnosis of PPCM is often delayed even

when symptomatic. Delayed diagnosis can be associated with increased morbidity and mortality. Therefore, emergency physicians should be alert to the possible development of PPCM when assessing peripartum patients.

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