

Peripartal Cardiomyopathy: Problems in Diagnosis and Management

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ABSTRACT

Peripartal cardiomyopathy is a rare form of heart disease in pregnancy with an unpredictable outcome. We describe one patient who presented in a decompensated state who was successfully managed with medical antifailure treatment. The etiology, management and future obstetric outcome are discussed.

Keywords: Peripartal cardiomyopathy, pregnancy

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INTRODUCTION

Peripartal cardiomyopathy is an uncommon form of heart disease of unknown aetiology. Alternate terms like toxic postpartum heart disease, postpartum myocarditis and postpartum heart disease have been used implying a type of myocarditis due to an infectious process although an autoimmune or idiopathic process is most likely.

The term peripartal cardiomyopathy is now generally accepted to describe the onset of heart failure in the last month of pregnancy or in the first five months post delivery in the absence of a demonstrable heart disease or a determinate cause for heart failure⁽¹⁾.

We describe here one patient who fulfills this criterion for peripartal cardiomyopathy. A review of the current literature is included to highlight the need for awareness of this condition among those practicing obstetrics in our community.

CASE REPORT

A 38-year-old Malay patient G₈ P₄ A₄ presented to the Accident and Emergency Unit on 26.06.98 at 38 weeks gestation with sudden onset of shortness of breath and inability to maintain a conversation, together with premature rupture of membranes.

She had been booked for antenatal care at a private practitioner's clinic. There had been no previous cardiovascular or pulmonary disease. On admission, she was orthopnoeic with central cyanosis and oxygen saturation of 60%. Blood pressure was 138/75 mmHg and pulse rate was 130/min. Bilateral coarse crackles

were widespread in both lung fields. Abdominal examination confirmed a term pregnancy in longitudinal lie with a cephalic presentation. The cervix was fully dilated and cervical os was 3 cm. Membranes were absent and clear liquor was leaking.

She was in established labour and was diagnosed to have heart failure. She was intubated and ventilated in the intensive care unit. Antifailure treatment with intravenous frusemide was initiated together with thromboprophylaxis with subcutaneous heparin. Syntocinon augmentation (fixed-volume regime) was started and a baby girl was delivered assisted by forceps delivery in the Intensive Care Unit on 26.06.98, six hours after admission.

Electrocardiogram in Intensive Care Unit revealed the heart to be in sinus tachycardia with poor V-wave progression from V1 to V3. Echocardiography revealed a dilated left atrium and left ventricle, AO/LA: 2.3/4.4 cm (AO = Aorta, LA = Left Atrium), LVIDS - 4.4 cm (Left Ventricular Dimension in Systole), IDD 5.7 cm (Dimension in Diastole) with ejection fraction of 35%. Overall left ventricular function was impaired globally. Functional mitral regurgitation was noted on continuous wave (CW) doppler studies. No mural thrombus or valvular lesions were present.

Haematogram was normal. The blood urea and electrolytes were also normal. pH was 7.10 with pCO₂ 48% and Base Excess of -20. After ventilation and oxygen therapy, her clinical condition improved. After three days, she was transferred to the high dependency ward and maintained with oral Perindopril 4-mg daily, oral Frusemide 40-mg daily and oral Potassium Chloride 1200-mg daily.

Echocardiography was repeated on 30.06.98 with minimal change from the original findings. She was discharged well on 07.07.98. Review on 13.07.98 showed left ventricle to be still dilated with an ejection fraction of 40%. No valvular lesion was present. She was reviewed a month later on 28.08.98 and again on 15.10.98. Echocardiography done during both follow-ups revealed no changes, ejection fraction was still around 35% to 40% respectively. She refused any form of contraception and there was no subsequent follow-up.

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DISCUSSION

The incidence of peripartum cardiomyopathy is 1: 3000 to 1:15,000 pregnancies, although a higher incidence is reported in Africa⁽²⁾. There were 7 cases reported in Ipoh Hospital from 1990 - 1997⁽³⁾. Although the aetiology is unclear, reference is made to nutritional deficiencies⁽⁴⁾. The patient discussed was in a good health prior to her ailment. Probably a type of myocarditis related to an infectious agent, or due to an autoimmune or idiopathic process is plausible. Patients with peripartum cardiomyopathy present with symptoms and signs related to systolic left ventricular dysfunction. Dyspnoea, orthopnoea, oedema and fatigue are common and this was evidenced in the patient discussed, reminiscent of left and right heart failure. Although our case had sinus tachycardia, atrial arrhythmias may be present worsening the clinical condition^(4,5).

The clinical presentation is often sudden and catastrophic. With shared care approach in our maternity services, poor access to records on premorbid illness presents severe difficulties in managing such patients in our context. As graphically presented in the case report, the patient was booked at a private practitioner's clinic and presented to the casualty in a moribund state without any record of her antenatal care. Central cyanosis in this patient warranted intubation and ventilation in the Intensive Care Unit for three days though the cornerstone of treatment in the acutely ill and highly symptomatic patient was intravenous preload and afterload reducing agents.

Treatment strategies follows diagnosis of the condition which is made by exclusion of other causes of congestive cardiac failure and the demonstration of ventricular dysfunction on echocardiography in the peripartum period. Whilst consideration of the foetus must be taken into account when instituting pharmacological treatment, in general the aim is to reduce amount of blood returning to the heart and to decrease resistance against which the heart must pump. Although Angiotensin II receptor blockers are ideal, these are contraindicated in pregnancy⁽⁶⁾. Calcium channels blockers, hydralazine and even nitroglycerine are suitable alternatives. Preload reduction was done using Frusemide. With labour being imminent in this patient, we were not reluctant in the use of Frusemide for cardiac indication. Aggressive haemodynamic monitoring often guides the acute phase of therapy. The haemodynamic goals would be to maintain a mean arterial pressure of 75mmHg, heart rate between 60 - 80 beats/min, systemic vascular resistance between 800 - 1200 dynes/sec/cm² and pulmonary capillary wedge pressure between 16 - 20 mmHg⁽⁷⁾.

Thromboprophylaxis with heparin was warranted in view of the high incidence of thromboembolism in peripartum cardiomyopathy. Immunosuppressive therapy has been advocated by some studies, but this approach needed further blended studies. Apart from pharmaco-

logical treatment, dietary advice should be directed to sodium and fluid restriction (less than two litres). Early ambulation, once the clinical situation allows, should be included in the active exercise program for these patients. This patient was performing her normal duties two months after delivery. It is generally agreed that 50% of patients have spontaneous resolution of their symptom⁽⁹⁾, but mortality is high in the remaining especially when treatment for heart failure is not available. Patients who continue to deteriorate despite aggressive intensive care therapy may be considered for cardiac transplantation as a final alternative.

Left ventricular ejection fraction has been shown to be higher in survivors when compared with non-survivors (22.8% versus 10.6%). Our patient had (LVDS of 4.8 cm) and ejection fraction of 35%. When the patient remained symptomatic beyond two weeks, prognosis was generally poor.

Tubal sterilization was advised but the patient declined when she was reviewed in the postnatal clinic. Our concern is the risk of recurrence in future pregnancy⁽¹⁾, although successful pregnancies have been reported⁽¹⁰⁾. Although patients may be asymptomatic after recovery from the acute illness, they have decreased contractile reserve when they are subjected to the haemodynamic stress of pregnancy. It is vital to extensively counsel such patients including the risk to be faced in future pregnancies.

Peripartum cardiomyopathy affects relatively young pregnant women. Although the aetiology is obscure, adherence to treatment protocols in intensive care setup should be advised to produce favorable outcomes. In our context, future pregnancies should be discouraged, as there is a risk of recurrence of cardiomyopathy.

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