Successful pregnancy following a peripartum cardiomyopathy

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Introduction

Peripartum cardiomyopathy is a rare complication of pregnancy, characterised by the development of heart failure secondary to a dilated cardiomyopathy in the peripartum period. Peripartum cardiomyopathy (PPCM) carries a significant morbidity and mortality and there is a risk of recurrence in subsequent pregnancies. Many issues relating to this condition are unresolved, including its exact aetiology, optimal treatment and assessment of the risk of recurrence.

We report the case of a 26 year old woman who developed a PPCM three days after the delivery of her first child. She made a complete recovery; she subsequently had a further successful pregnancy with no evidence of recurrence of PPCM.

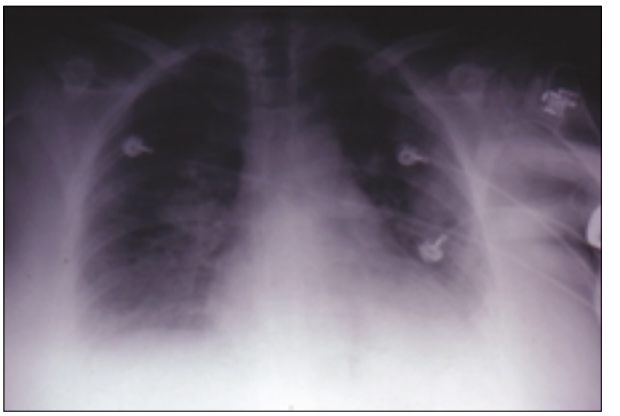
Case report

A 26 year old primigravida woman presented at 24 weeks for her first (booking) antenatal visit. Ten years before she had undergone a complete and successful course of treatment for pulmonary tuberculosis. There was no other past medical history of note. There was no family history of cardiac disease. The patient did not smoke and had abstained from alcohol throughout the pregnancy.

The patient's clinical examination on presentation was normal, with a blood pressure of 120/80 mmHg and normal urinalysis. The pregnancy continued in a normal fashion. At 27 weeks, minimal (1+) leg oedema was noted and at 39 weeks and four days, a trace of protein was found in the urine. Her blood pressure at this point was 140/85 mmHg. Her platelets ($257x10^9$/L) and uric acid (384 µmol/L) were in the normal range. Two days later (39 weeks and six days) her blood pressure was elevated at 140/95 mmHg. On the basis of her mild pre-eclampsia a decision was taken to induce labour the following day.

On admission to hospital for induction of labour, oedema of the patient's legs and fingers was noted. Labour was induced at 8.45 am and artificial rupture of membranes was performed at 2.15 pm. There was failure to progress in the second stage, with the development of fetal tachycardia. A Caesarean section was performed and a healthy male infant (3.3 kg) was delivered. The mother received one litre of Hartmann's solution and one litre of Dextro-Saline (4% dextrose, 0.18% saline) and then intravenous fluids were stopped.

Figure 1. Chest X-ray on admission to the intensive care unit, showing pulmonary oedema



About 70 hours after delivery, the patient became unwell, complaining of breathlessness and dizziness. Her blood pressure and pulse were found to be normal, as was respiratory examination. Three hours later the patient had deteriorated, with increasing dyspnoea and tachypnoea. Respiratory examination now revealed bilateral crepitations on auscultation. The resting electrocardiogram was normal but a chest X-ray revealed perihilar changes consistent with pulmonary oedema (figure 1). Intravenous frusemide and oxygen were administered and the patient's symptoms improved. She was then transferred to the intensive care unit of a neighbouring hospital.

On arrival at the intensive care unit her pulse was 90/min and blood pressure 140/70 mmHg. The heart sounds were normal and mild peripheral oedema was noted. Chest examination revealed bilateral crepitations, as before. Further intravenous frusemide and diamorphine were administered. Blood tests showed a haemoglobin of 8.1 g/dl, white cells $13.8x10^9$/L and a normal platelet count. The following day a chest X-ray showed resolution of the oedema. The patient's cardiothoracic ratio was 0.5.

A transthoracic echocardiogram was performed the next day

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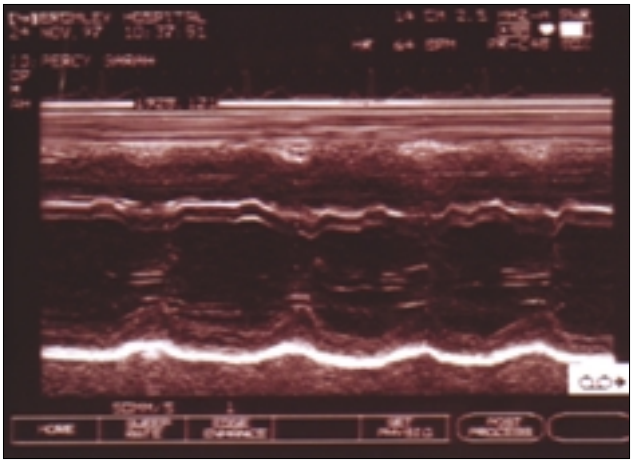
Figure 2. Parasternal, long axis, M-mode echocardiogram through the mitral valve leaflets, showing normal left ventricular function shortly after delivery of the patient's second child



(48 hours after the onset of breathlessness). This demonstrated a slightly enlarged left ventricle, with decreased septal contractility and an overall decrease in ventricular function. The left atrium was also enlarged.

The patient improved clinically and a repeat echocardiogram two days later showed a left ventricle of normal size (end-diastolic diameter = 5.3 cm, end-systolic diameter = 3.5 cm), with the fractional shortening normal at 35%. The left atrium was now of normal size. A chest X-ray taken two days later was entirely normal. The patient made a full recovery.

She became pregnant again two years later. At presentation cardiac assessment was entirely normal. She was kept under regular cardiological review: echocardiography was performed every four weeks up to the 32nd week of pregnancy and then weekly until delivery. Left ventricular dimensions and function remained normal throughout. In an attempt to prevent recurrence of her PPCM an elective Caesarean section was performed at 36 weeks, which proceeded without complication. She remained well post-partum: ongoing echocardiographic monitoring showed normal left ventricular dimensions and function (figure 2).

Discussion

Peripartum cardiomyopathy has been recognised as a distinct entity since the mid-nineteenth century. The incidence is reported to be between 1:1,300 and 1:30 000 live births.[1,2] The infrequency of the condition makes randomised trials difficult and therefore most of our knowledge of the condition comes from case reports and small patient series.

Diagnostic criteria for PPCM were established by Demakis *et al*[3] in 1971. These include: the development of cardiac failure in the last month of pregnancy or within five months of delivery; the absence of a determinable cause for the cardiac failure; and the absence of demonstrable heart disease before the last month of pregnancy. More recently, Lampert and Lang[1] have suggested

Key messages

- Peripartum cardiomyopathy carries a significant risk of mortality and morbidity
- The incidence is between 1:1,300 and 1:30 000 live births
- In those patients who survive the first episode but do not fully recover left ventricular function, subsequent pregnancies carry a significant risk of recurrence

a fourth criterion – an echocardiographically demonstrable impairment in systolic left ventricular function.

Our patient therefore fulfilled the case definition for PPCM. She developed cardiac failure three days post-partum without pre-existing heart disease, and echocardiography demonstrated impaired systolic function. It is also of note that her chest X-ray showed a cardiothoracic ratio of 0.5, which is probably abnormal for a young woman.

The treatment for PPCM is similar to that for other forms of cardiomyopathy. It includes diuretics, vasodilators and inotropic support if necessary. Anticoagulation may need consideration due to the risk of thromboembolic phenomena. Drug therapy is limited to preparations that are safe during pregnancy and the post-partum period. In those patients who fail to respond to medical therapy, cardiac transplantation is an option. Rickenbacher *et al*[4] have shown transplantation to be as effective in treating PPCM as it is in idiopathic dilated cardiomyopathy.

Overall, maternal mortality is estimated to be between 25 and 50%.[5] Nearly half of these deaths occur in the first three post-partum months.[6,7] Patients who recover left ventricular function have a significantly improved survival.[8]

Approximately 50% of patients with peripartum cardiomyopathy do not recover normal left ventricular function.[9] In estimating the prospects of recurrence, the degree of recovery of resting left ventricular function may be important. In those who do not recover completely, the chances of a poor clinical outcome in subsequent pregnancy are increased.[3,10]

The data on the prognosis for future pregnancy in those that do recover normal left ventricular function are conflicting. Some observational studies[10-12] support a poor outcome even in this group, prompting Witlin *et al* in 1997 to consider sterilisation for patients surviving a first episode of PPCM.[10] Lampert and Lang[1] assessed left ventricular contractile reserve in patients with recovered ventricular function after peripartum cardiomyopathy. They showed, by using a dobutamine challenge (dobutamine stress echocardiography), persistently impaired contractile reserve in patients after PPCM. This prompted the authors to suggest caution in advising further pregnancy.

However, Sutton *et al*[13] looked at four patients who had become pregnant after a previous PPCM. Echocardiography was used to assess their left ventricular function throughout preg-

nancy. There was no deterioration in left ventricular size or function; all four cases had a normal delivery. Our case followed this pattern, with complete recovery of resting ventricular function after her PPCM followed by a normal pregnancy.

Conclusion

PPCM is a rare but important cause of morbidity and mortality to mother and child. In those patients who survive the first episode but do not have complete recovery of resting ventricular function, subsequent pregnancies carry a significant risk of recurrence and should be avoided. If recovery is complete, the risk of recurrence from the limited data available appears to be smaller. Nonetheless, careful counselling of patients is recommended, with close monitoring of cardiac function throughout the pregnancy and peripartum period.

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