Postpartum cardiomyopathy: a diagnostic dilemma

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Abstract

Postpartum cardiomyopathy (PPCM) is the development of heart failure in the last month of pregnancy or within 5 months after delivery without an identifiable cause in a previously healthy female. It is a rare condition, which carries a high maternal mortality. We describe the case of a 35-year-old South Asian woman who presented with dyspnoea, fatigability, peripheral oedema and abdominal distension. Given the non-specific symptoms, echocardiographic findings indicative of left ventricular systolic dysfunction established the diagnosis of PPCM.

Keywords

Cardiomyopathy; postpartum; echocardiogram; ventricular dysfunction.

Case report

A 35-year-old South Asian woman underwent an elective caesarean section (CS) at 38 weeks gestation at our unit in May 2005. Her obstetric history included one CS 2 years previously following a myomectomy for a fibroid uterus. She gave a history of hypertension in both pregnancies during the ninth month of gestation for which no treatment was necessary. Her past medical and family history was otherwise insignificant. However she did mention that during the last month of her current pregnancy she was mildly dyspnecic felt tired and noted pedal oedema. She also gave a history of palpitations. She underwent an elective CS and was discharged home on the sixth post-operative day in satisfactory condition.

Three months later in August 2005 the patient presented to the hospital with 3 weeks of progressive dyspnoea, orthopnea and paroxysmal nocturnal dyspnoea. She experienced chest pain, sweating and fatigability and noticed severe peripheral oedema and abdominal distension. On examination she appeared pale and was in severe respiratory distress. She had a respiratory rate of 28 breaths/min, pulse of 130 beats/min and blood pressure of 138/90 mmHg. Her jugular venous pressure was elevated at 22 cmH\textsubscript{2}O. Praecordial examination showed an apex beat displaced to the 6th intercostal space, 3 cm lateral to the midclavicular line. The 1st heart sound was soft and the 2nd heart sound normal with left ventricular summation gallop.

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Chest examination revealed bilateral basal crepitations. Abdominal examination revealed pulsatile, tender hepatomegaly with a liver span of 13 cm and moderate ascites. Blood chemistry indicated a low serum albumin (2.6 g/dl), slightly raised serum glutamic transaminases and LDH. Her haemoglobin was 10 g/dl. Chest X-ray showed gross cardiomegaly and pulmonary congestion. ECG demonstrated sinus tachycardia. The initial echocardiogram revealed a dilated left ventricle (left ventricular end diastolic diameter (LVEDd) of 6.1 cm and left ventricle internal diameter (LVID) of 5.3 cm) with global thinning and hypokinesia of the left ventricular walls (Figs. 1 and 2, left panels). The right ventricle and the left atrium were slightly dilated with moderate tricuspid and mild mitral regurgitation. There was small pericardial effusion and the left ventricular systolic function was severely impaired (fractional shortening (FS) 14%). The echocardiographic findings were strongly consistent with dilated cardiomyopathy.

She was treated with frusemide, enalapril, spirinolactone, carvedilol and digoxin. She was anticoagulated initially with unfractionated heparin and maintained on warfarin. She responded well to the treatment and was discharged in on the tenth day. She was recommended to continue her treatment along with a low salt diet and reduced physical activity.

On her follow up visit significant recovery of most of her clinical and echocardiographic abnormalities were noted. A repeat echocardiograph in February 2006 revealed a normal left ventricle size and function, normal left atrium and right ventricle and disappearance of the small pericardial effusion. Mitral regurgitation had also disappeared and the tricuspid regurgitation was mild. The last LVEDd was 5.4 cm (Fig. 1, right panel) and LVID was 3.8 cm (Fig. 2, right panel), FS 30% and ejection fraction was 57%.

Discussion

PPCM is a rare, complex, clinical condition that results in cardiomyopathy. The true prevalence of PPCM is unknown. A recent report has highlighted the clinical relevance of G-3 subclass immunoglobulins in PPCM patients from different global regions[1]. The diagnostic criteria of PPCM have been clearly described and the echocardiographic findings of reduced left ventricular systolic function and depressed ejection fraction substantiate the diagnosis[2]. Advanced maternal age and multiparity have been noted as high risk factors[3]. Our patient complained of fatigability, palpitations, dyspnoea and pedal oedema. Many women do experience these symptoms towards the end of pregnancy due to physiological changes. Ten weeks after birth, she developed cough, chest pain and mild dyspnoea and was thought to have a chest infection. Her condition worsened and she had all the features of congestive cardiac failure. The diagnosis of PPCM was finally established by echocardiography. As the incidence of PPCM is low and symptoms non-specific diagnosis can often be delayed and may even be missed unless echocardiography is performed. The treatment includes diuretics and carvedilol, a beta-blocker with the vasoactive property.
of reducing after-load. ACE inhibitors are contraindicated during pregnancy due to teratogenic effects on the foetus. Rarely patients may require heart transplantation for symptoms refractory to medical treatment\textsuperscript{[4]}. Post-operative survival in this group of patients is equivalent, regardless of parity, to survival in women requiring transplantation for idiopathic dilated cardiomyopathy.

Our patient achieved normalisation of left ventricular size and function within 6 months. It is possible that patients who have recovered from PPCM and regained normal left ventricular size and function will still have depressed contractile ventricular function when subjected to the haemodynamic stress of pregnancy\textsuperscript{[5]}.

Teaching point

This report highlights the importance of recognition of symptoms and treatment of cardiac failure in the post-partum period especially as deaths from PPCM feature regularly in studies of pregnancy-related deaths\textsuperscript{[6]}.

References