Peripartum Cardiomyopathy: A Review of Three Case Reports

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy that presents with heart failure secondary to left ventricular systolic dysfunction toward the end of pregnancy or in the months after delivery. Incidence of PPCM ranges from 1:300 to 1:15,000 pregnancies. Causes and pathogenesis are poorly understood. Clinical presentation includes signs and symptoms of heart failure. PPCM remains a major cause of maternal morbidity and mortality. Many cases of PPCM improve or resolve completely but others progress to heart failure; as early diagnosis and medical treatment may affect the patient’s long-term prognosis. The aim of this report is to make health professionals aware of the possibilities in a woman with dyspnoea in the postpartum period.

Keywords: Peripartum cardiomyopathy, Left ventricular dysfunction, Echocardiograph.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an infrequent but critical disorder in which a destabilized heart is diagnosed within the last months of pregnancy or early puerperium and often complicating obstetrics as well as anesthetic management.1 The definition of PPCM includes four criteria: (1) Development of cardiac failure in last month of pregnancy or within 5 months of delivery, (2) absence of identifiable cause for cardiac failure, (3) absence of recognizable heart disease before the last month of pregnancy and (4) left ventricular (LV) dysfunction (ejection fraction of less than 45% or reduced shortening fraction.1,2 It is possible to misdiagnose postpartum cardiomyopathy for pulmonary embolism (PE). An error in diagnosis is life-threatening for the patient with a potential for fatal outcome in about 20%.

Out of the following three case reports; two illustrate typical presentations and an atypical one.

CASE REPORTS

Case 1

A 21-year-old primigravida was admitted to our labor room on 17/4/2007 at 12:00 pm as a case of 36 weeks twin pregnancy with pregnancy-induced hypertension with mild anemia and acute onset breathlessness and anxiety.

She was also admitted 7 days back for similar complaints during which she was investigated and managed conservatively.

After her symptoms subsided she was discharged on persistent request 3 days after admission. At the second time of admission, her pulse rate was 98 per minute, respiratory rate was 36 breaths per minute, blood pressure was 156/90 mm Hg. Her hemoglobin was 9.0 gm/dl, LFT and KFT were within normal limits. Cardiovascular system and respiratory system examination showed no abnormality. She passed into labor 8 hours after admission and had fetal distress during early labor for which she underwent emergency lower segment cesarean section under GA on 16 April 07 and an alive first female baby weighing 2.7 kg and second female baby 2.8 kg was delivered. C-section was uneventful with no intraoperative or postoperative complications. On second postoperative day she developed tachycardia and breathlessness. Her chest was full of bilateral coarse crepitations. She was given intravenous furosemide and high flow oxygen but her general condition did not improve. Urgent cardiology opinion was taken. Chest X-ray PA view was done which showed cardiomegaly and transthoracic echocardiography (Fig. 1) performed which showed hypokinesia of left ventricle with decreased left ventricular systolic function, ejection fraction of 38% and left ventricular end diastolic dimension 52 mm and was diagnosed as a case of peripartum cardiomyopathy (PPCM).4

She was shifted to medicine intensive care unit and given ventilatory support in form of continuous positive airway pressure (CPAP), she was managed conservatively with intravenous furosemide, digoxin and blood transfusion. Ventilatory support was continued for 48 hours and thereafter extubation was done. She was discharged on 24/4/2007 at which her pulse rate had improved to 78 beats per minute, respiratory rate was 18/min and blood pressure 124/80 mm Hg. Chest was clear and cardiovascular system was normal. At the

Fig. 1: Transthoracic echocardiography examination showing dilated left atrium and left ventricle
time of discharge she was advised to continue furosemide tablet 20 mg once daily and tab metoprolol 12.5 mg once daily for 2 weeks. At 6 months follow-up she was asymptomatic, her ejection fraction had improved to 76%.

Case 2

A 30-year-old primigravida came to our labor room on 12/2/2010 at 5:45 pm with amenorrhea for 9 months and swelling over body for 9 days and diagnosed as full-term pregnancy with mild preeclampsia, severe anemia with ascites. At the time of admission her pulse rate was 120 beats per minute, respiratory rate was 28 breaths per minute and blood pressure was 180/98 mm Hg. Her investigations LFT, KFT and complete blood count were within normal limit except her Hb% which was 7 gm%. She underwent emergency lower segment cesarean section under general anesthesia on 15th February 2010 for fetal distress and meconium-stained liquor and an asphyxiated male baby weighing 3.5 kg was delivered. Her ascitic fluid was sent for cytobiochemical analysis. Her intraoperative and immediate postoperative period was uneventful. On 3rd day of examination she developed tachycardia, respiratory distress for which medicine and cardiology opinion was taken. Chest was full of coarse crepitations and fine basal crepitations. Her chest X-ray showed cardiomegaly, ECG showed left axis deviation and USG whole abdomen revealed congestive hepatomegaly with ascites, with mild left pleural effusion, with mild pericardial effusion, with postpartum bulky uterus and with dilated cardiomyopathy.

She was shifted to medicine ICU, there her transthoracic echocardiography was done which showed globally dilated and hypokinetic left ventricle with ejection fraction 40%. She was managed with IV fluids, blood transfusion, intravenous furosemide, metoprolol 50 mg twice daily. Her general condition improved. She was discharged on 5th March 2010 with advised to continue oral furosemide 20 mg, metoprolol 12.5 mg twice daily for 3 weeks. She did not come for follow-up.

Case 3

A 29-year-old multigravida (G3 P2 L2 Ao) came to our labor room on 26/2/2010 at 8:40 pm with chief complaints of amenorrhea for 8 months swelling all over body for 10 days and breathlessness for 12 hours. On admission her pulse rate was 110/minute, respiratory rate was 40 breaths per minute, blood pressure was 138/108 mm Hg. On auscultation her chest had bilateral coarse crepitation and fine basal crepts. Her hemoglobin was 12.7 gm%.

She had an echocardiograph done from elsewhere which reported dilated cardiomyopathy with ejection fraction 22%.

She was managed conservatively with intravenous furosemide, nebulization with salbutamol and oxygenation.

She left against medical advise on the same day.

DISCUSSION

PPCM is a rare lethal disease having incidence of one case per 1,374 live births in an Indian study.3 It occurs in 1 in 3,000 to 1 in 4,000 pregnancies in United States.1,2 Higher rates in developing countries may be due to variations in local cultural as well as puerperal practices, ecological factors, environmental influence, diagnostic criteria and reporting pattern used.5 Though the exact cause of PPCM remains unknown, viral myocarditis, autoimmune phenomena and specific genetic mutations that ultimately affect the formation of prolactin have been proposed as possible causes.1,4 Risk factors include multiparity, black race, older maternal age, preeclampsia and gestational hypertension.1 Symptoms of PPCM include fatigue, edema and dyspnea which are similar to those for normal spectrum of peripartum states and pregnancy such as pulmonary emboli and eclampsia. Therefore, diagnosis is often delayed and the disorder is under recognized with devastating consequences. Mortality is as high as 20 to 50%.

Treatment of PPCM is the same as for other forms of congestive heart failure (fluid and salt restriction, B blocker, diuretic and digoxin), except for angiotensin converting enzyme inhibitors and angiotensin receptor blockers, which are contraindicated in pregnancy.7 In about 50% of patients, the ejection fraction normalize. Regardless of the recovery, however, a second pregnancy is usually not recommended for these patients because PPCM recurs in more than 30% of subsequent pregnancies, which puts both mother and baby at risk.8 The risk factors though may help in screening out the high-risk patients but women without the enumerated risk factors and with atypical presentation as seen in one cases may be the candidates of PPCM.

Thus, it is important that obstetricians should be familiar with PPCM and therefore consider it when diagnosing dyspeptic patients to expedite medical treatment for a potentially lethal condition.

REFERENCES


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