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Peripartum cardiomyopathy: An unusual but significant form of heart failure

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Abstract
Peripartum cardiomyopathy (PPCM) is an uncommon but potentially life-threatening condition that should be promptly diagnosed and correctly treated. We report a case of PPCM diagnosed in postoperative period. The patient presented with pedal edema, difficulty in respiration, palpitation and epigastric discomfort. Electrocardiogram showed sinus tachycardia and poor R wave progression. Echocardiography showed severely impaired left ventricular function with ejection fraction (EF) <15%. Cardiologic drugs were given and the patient was discharged in asymptomatic condition. As shortness of breath, fatigue and pedal edema are common in the peripartum period, a high index of suspicion is required so as not to miss the diagnosis. Measurement of natriuretic peptide and echocardiography are recommended to promptly diagnose or exclude PPCM. Early diagnosis is important and effective treatment reduces mortality rates and increases the chance of recovery of ventricular systolic function. The important role of education and counselling around breast feeding and future pregnancy is emphasized.

Keywords: Peripartum cardiomyopathy, pregnancy, puerperium, echocardiography, heart failure

Introduction
Peripartum cardiomyopathy an unexplained left ventricular heart failure is a rare complication of pregnancy that appears at any time in the last month of pregnancy and upto 5 months after delivery. The incidence of PPCM differs widely depending on the ethnic, racial and regional background of women [1]. Africans are at the highest risk of developing PPCM [2]. In USA, an increasing incidence was described over the past years. The etiology seems to be multifactorial and poorly understood. The diagnosis is often delayed because the symptoms of PPCM closely resemble the spectrum of normal pregnancy and puerperium. Early diagnosis and effective treatment reduces the morbidity and mortality associated with PPCM. Obstetricians should be familiar with this form of heart failure; should keep PPCM in differential diagnosis if a patient is having exaggerated dyspnoea, fatigue and pedal edema in peripartum period. We report a case of PPCM diagnosed after caesarean section.

Case Report
A 27-year-old primigravida with no significant past medical history presented to the emergency department of our institute at 38 weeks of gestation with the chief complaints of pedal edema for 15 days and shortness of breath, palpitation and epigastric discomfort for 2 days. On general physical examination the patient was afebrile, blood pressure was 140/100 mm of Hg, pulse rate was 130 beats per minute, respiratory rate was 24 breaths per minute and grade 3 pitting edema was present on both the legs. There was no evidence of anaemia, cyanosis and jaundice. Her lungs were clear on auscultation with normal vesicular breath sounds, cardiac sounds (S_1 and S_2) were normal and no murmur was appreciable. Oxygen saturation was 85% on room air. Electrocardiogram (ECG) showed sinus tachycardia (heart rate 125 beats/ minute), normal axis with poor R wave progression. The troponin –T test was negative. Physician referred the patient to the labour ward of our department for obstetric evaluation. On per abdominal examination uterus was of term size with breech presentation. Dipstick urine test showed +2 proteinuria. The initial laboratory investigations showed: haemoglobin 10.8 gm%, white blood cell count 16,000/mm^3 with normal differential count, platelets 2,02,000/mm^3, serum sodium 137mEq/L, potassium 4.1 mEq/L, blood urea 42mg/dL, blood sugar 85mg/dL, serum creatinine 1.2mg/dL,

AST 31 U/L, ALT 22 U/L, ALP 335 U/L and prothrombin time was 14 seconds with INR 1.04. Patient was taken up for emergency caesarean section in view of preeclampsia with breech presentation. After few hours patient started complaining of respiratory distress even on high flow oxygen support. Her blood gas analysis showed PH- 7.39, $PaCo_2$ 42.5 mm Hg, bicarbonates 25 mEq/L and oxygen saturation 95%. Her condition further deteriorated that required endotracheal intubation. During evaluation the chest X-ray showed blunting of bilateral costo-phrenic angles with mild cardiomegaly. Ultrasonography revealed mild to moderate pleural effusion and air bronchogram suggestive of pulmonary edema. Analysis of pleural fluid aspirate showed: total leucocyte count 137x 10^3/ml with 49% neutrophils and 51% lymphocytes, protein 1gm/dL and ADA (Adenosine Deaminase) 6.56 U/mL. It was negative for acid- fast bacilli on microscopy. The findings were suggestive of transudative nature of fluid. N Terminal pro B type Natriuretic Peptide (NT-Pro BNP) valued 1342 pg/mL. Echocardiography showed severely impaired left ventricular function with EF <15% with moderate mitral regurgitation and trace tricuspid regurgitation. Both ventricles appeared severely enlarged and diffusely hypokinetic. The patient developed cardiogenic shock. Central line was inserted through internal jugular vein for better fluid management. Intravenous infusion of noradrenaline and subsequently dobutamine were started to support failing heart. Diuresis was started with inj. Lasix (furosemide) 20 mg 8 hourly and tablet spironolactone 50 BD. The inotropes were gradually tapered over next 3 days. Tablet isosorbide dinitrate 5mg TDS, tablet ramipril 1.25 mg OD and tablet ivabradine were added to reduce preload, afterload and heart rate respectively. The patient was also commenced on enoxaparin (low molecular weight heparin) 60 mg BD for thromboprophylaxis. Patient improved clinically and was weaned off from mechanical ventilation. She was ambulatory by day 11 of start of cardiologic treatment and was discharged in asymptomatic condition on tablet vymada 50mg (sacubitril 24 mg + valsartan 26 mg) once daily, tablet lasilactone 20/50 BD (20 mg furosemide + 50 mg spironolactone), tablet ivabradine 7.5 mg BD, tablet sorbitrate (isosorbide dinitrate) 5mg TDS, tablet coreg (carvedilol) 3.125. mg OD. At 1st and 2nd month follow-up, the patient was asymptomatic on cardiologic drugs but on echocardiography EF was < 15%.

Discussion

The initial description of the syndrome suggested that it appears after parturition so it was named ‘postpartum cardiomyopathy’ or ‘puerperal heart failure’. Today we know that PPCM may appear anytime in the last month of pregnancy upto 5 months after delivery. Although historically PPCM risk factors include advance maternal age, black women, multiparity, multifetal gestation and obesity; contemporary trends show that there is an increased incidence (24-37%) in young primigravida and white patients.[3] Other risk factor are family history of smoking, diabetes mellitus, hypertension, preeclampsia, malnutrition and prolonged use of tocolytic beta- agonists.[4] A combined ‘two-hit’ model including systemic angiogenic imbalance host susceptibility (predisposition) is thought to be crucial in the pathophysiology of PPCM [5, 6]. Possible factors leading to PPCM include genetic predisposition, low selenium levels, viral infections, stress- activated cytokines, inflammation, autoimmune reaction, pathological response to haemodynamic stress, unbalanced oxidative stress and induction of angiogenic factors [5, 7].

Majority of the patients present with weakness, shortness of breath, cough, orthopnea, paroxysmal nocturnal dyspnoea and palpitations. Physical examination may reveal tachycardia, tachypnoea, cardiac arrhythmias, pulmonary crepitations and peripheral edema. Such signs and symptoms overlap with those of many other conditions ranging from normal pregnancy to preeclampsia leading to pulmonary edema, upper respiratory infection, pregnancy associated myocardial infarction, hypertensive heart disease during pregnancy and pre-existing heart disease [1, 3]. The diagnosis of PPCM includes following 4 criteria: 1) development of heart failure in the last month of pregnancy or upto 5 months postpartum, 2) absence of an identifiable cause for the cardiac failure, 3) absence of recognizable heart disease before the last month of pregnancy, 4) left ventricular dysfunction (EF of less than 45% or reduced fractional shortening) [8]. Figure 1 summarizes the diagnostic pathway in patients with suspected PPCM including determination natriuretic peptides, X-ray, ECG and echocardiography (according to local availability, not all have to be performed in all the patient) [1, 9]. Transthoracic echocardiography enables differentiation of heart failure with preserved ejection fraction, commonly observed in women with preeclampsia, from that with PPCM in which a reduced ejection fraction is more common [2]. Depending upon the clinical presentation and echocardiographic findings PPCM can be divided into mild, moderate and severe forms. In mild PPCM, patient is hemodynamically stable and has subacute heart failure. The left ventricular ejection fraction (LVEF) is 30-45% and patient can be managed on oral heart failure drugs. Moderate PPCM includes respiratory insufficiency and acute heart failure. The LVEF is between 20-35%. Patient requires oxygen supplementation/non- invasive ventilation, intravenous diuretics, vasorelaxants apart from other heart failure drugs. Patients with severe PPCM presents in cardiogenic shock. The LVEF is <25%. Patient usually requires invasive ventilation, inotrope support and mechanical circulatory support [1, 10]. Heart failure drugs are added after stabilization. The duration of heart failure treatment is determined by the patient’s heart performance at rest and with dobutamine infusion. Patients with normal EF at rest and during dobutamine could taper off the medical therapy in 6-12 months; patients with normal EF at rest and abnormal EF during dobutamine should be treated for longer periods.[11] Patients who continue to have depressed ventricular function at rest should receive medical therapy indefinitely [12]. If medical treatments are not successful, heart transplantation is often last resort. Fortunately in recent years, the rate required transplantation has decreased to about 4 to 7% [13]. Transplantation success rates are good with favourable long term survival rates [14].

The obstetrician who cares for patient with PPCM should councel her about breastfeeding and contraception. Breastfeeding in patients with heart failure is controversial. It may confer important physical and psychological benefits to infants and mother, especially in developing countries. According to the 2018 ESC guidelines for the management of cardiovascular diseases during pregnancy, in patients with severe heart failure preventing lactation may be considered due to high metabolic demands of lactation and breastfeeding. These guidelines state that stopping lactation enable safe treatment with all established heart failure drugs.[15] Decisions on whether to inhibit lactation, terminate breastfeeding or continue breastfeeding with caution should be taken jointly with the patient on a case-by-case basis, taking into consideration both the health of mother and the risk: benefit ratio of breast feeding to the infant. Good counselling and shared decision making are

key [1]. The patients of PPCM have 30% risk of relapse and significant decrease of left ventricular function in subsequent pregnancy; mortality rate is described to be approximately 55% even though it seems associated more with patients who entered the subsequent pregnancy with abnormal systolic function i.e. without making complete recovery. Since complete recovery from a relapse is very rare and due to increased chance of relapse and mortality in the next pregnancy, a second pregnancy is usually not recommended for patients with history of PPCM, which puts both mother and baby at a great risk [2, 3, 16, 17, 18].

In conclusion, PPCM is an uncommon but potentially fatal disease. Thus it is important that obstetricians be familiar with PPCM and therefore consider it in patients presenting with shortness of breath, fatigue and pedal edema. Early diagnosis and effective treatment reduces morbidity and mortality rates associated with PPCM.

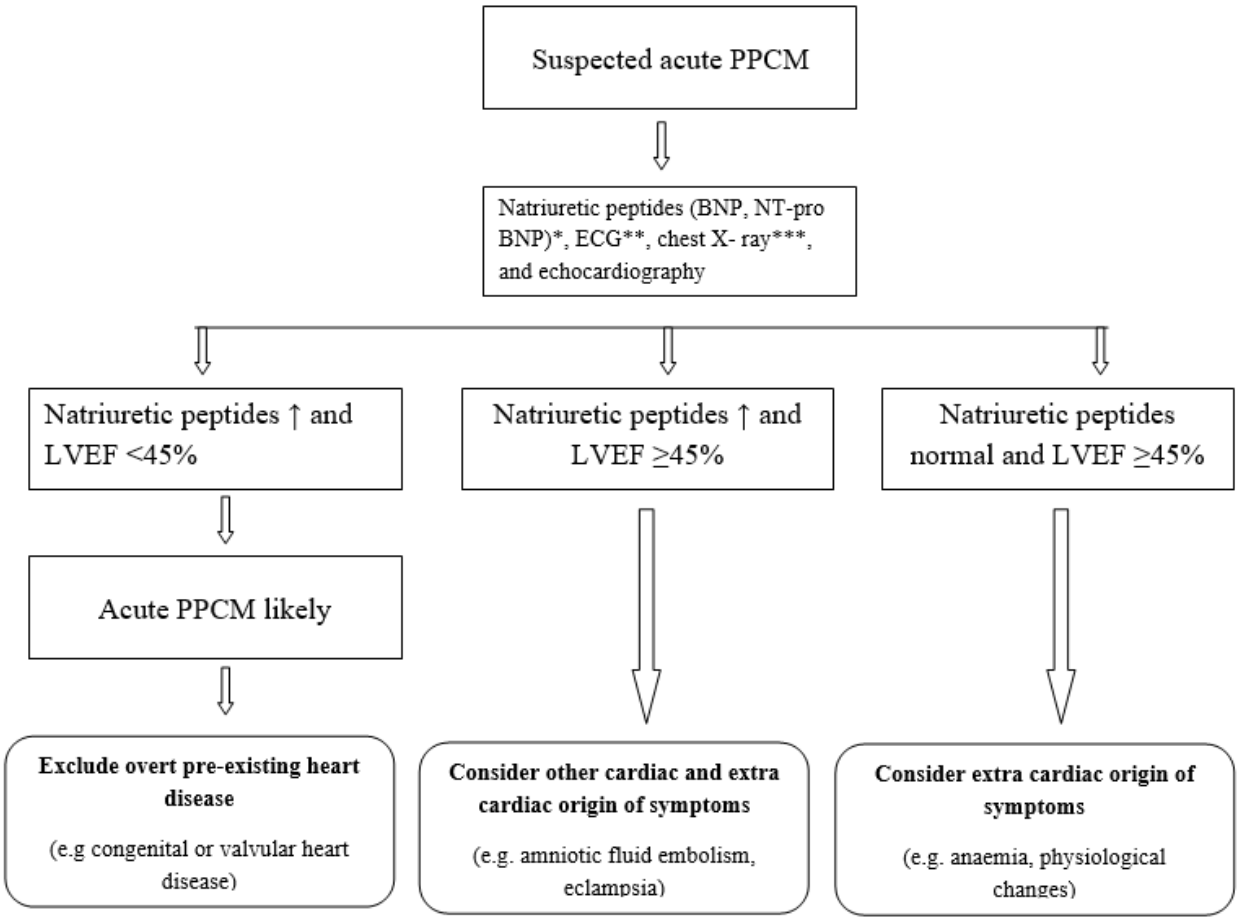

Fig 1: [1, 9] Diagnostic pathway in patients with suspected peripartum cardiomyopathy (PPCM). BNP, B – type natriuretic peptide; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; NT – proBNP, N – terminal pro – B – type natriuretic peptide.
*Cut off for acute heart failure: NT-proBNP >300 pg/ml, BNP >100 pg/ml
**An electrocardiogram may show no abnormalities, sinus tachycardia, nonspecific ST and T- wave abnormalities and voltage abnormalities.
***Chest films may reveal typical signs of congestive heart failure.

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