

Peripartum Cardiomyopathy: Outcome Analysis in Tertiary Health Care Centre

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1. Introduction

During Pregnancy, heart failure has been recognized since longtime¹. However, peripartum cardiomyopathy was first described by Gouley et al by a case series. PPCM is heart failure of unknown etiology occurring in the last month or the first five months of postpartum period in a previously healthy pregnant women^{2,3,4,5,6}. Even though the incidence of peripartum cardiomyopathy is low, mortality and morbidity related to this disease are very high.

Table 1: Definition of Peripartum Cardiomyopathy^{7,8,9,10,11}

DEFINITION OF PPCM

- Development of heart failure in the last month of pregnancy or within the 5 months following delivery.
- Absence of determinable etiology of heart failure
- Absence of demonstrable heart disease prior to the diagnosis of heart failure
- Left ventricular ejection fraction < 45% ,or fractional shortening <30% or both

Advanced maternal age, multiparity, multiple gestation, obesity, preeclampsia, malnutrition, black race, gestational diabetes and hypertension are risk factors for developing peripartum cardiomyopathy^{12,13,14}. The etiopathogenesis of PPCM is poorly understood¹⁵. The postulated hypothesis are abnormal immune response to pregnancy, viral myocarditis, stress activated cytokines, genetics, hemodynamic stress of pregnancy, prolactin, cardiotropic viruses and prolonged tocolysis^{16,17,18}. Clinical presentation of PPCM is similar to signs and symptoms of cardiac failure and very rarely thromboembolic manifestations¹⁹. NT-proBNP is the only marker available for screening of PPCM²⁰. Here we report five cases of PPCM with various clinical presentations.

Case 1:

A 22 year old woman, who delivered a female baby by labour natural. During the first postnatal period she developed sudden onset of breathlessness and fatigability and cough. On examination, she was found to be dyspnoeic and tachypnoeic. While reviewing her medical history, she was normotensive and euglycemic and had no history of any other chronic ailments.

The patient was shifted to intensive care unit and put on continuous monitoring. The patient was noted afebrile and had a pulse rate of 110 beats per minute. Her blood pressure was 90/60 mmHg and a respiratory rate of 20 beats per minute. Her Oxygen saturation was 90% with 6 liters per minute of oxygen through nasal cannula. Her heart rate was

regular with systolic murmur at apex and S3 gallop. There was bilateral fine basal crepitations. There was no calf muscle tenderness and pedal edema. Urine analysis was negative for proteins. Her plasma D dimer level was 590 pg/ml. Blood gas analysis, renal function, liver function and clotting parameters were within normal limits.

Electrocardiogram showed sinus tachycardia without any ST, T changes. There was cardiomegaly in chest x-ray with increased lung vascularity bilaterally. Echocardiographic evaluation showed global hypokinesia of left ventricle with reduced ejection fraction of 35% with trivial mitral regurgitation. There was relaxation abnormality. Pulmonary artery systolic pressure was normal. A diagnosis of peripartum cardiomyopathy was made and the patient was treated with intravenous diuretics, ACE inhibitor and beta-blocker. Her symptoms improved with treatment. Follow up echocardiogram after three months showed improved ejection fraction of 55% with no symptoms.

Case 2:

A 20 year old primigravida with 37 weeks of gestation referred from peripheral hospital with suspected peripartum cardiomyopathy. There was history of acute onset of shortness of breath, cough and palpitation. We received the patient in a state of hypotension with tachycardia and generalized edema. There was no past history of any medical illness. Echocardiogram showed global hypokinesia of left ventricle with severe systolic dysfunction. There was 15x10 mm clot present in the left ventricle apex with ejection fraction of 20%. The patient was treated with inotropes and others. Suddenly she developed desaturation and put on mechanical ventilation. We lost the patient due to cardiogenic shock before evaluating further.

Case 3:

30 year old third gravida developed shortness of breath one week after delivery. After ruling out other causes of acute onset of breathlessness, provisional diagnosis of peripartum cardiomyopathy was made. While provoking the previous history, there was history suggestive of peripartum cardiomyopathy during second pregnancy at the time of delivery. First pregnancy was uneventful. The patient was hemodynamically stable. Echocardiogram showed hypokinesia of left ventricle with ejection fraction 40%. The patient was treated with PPCM specific therapies. Follow up examination at 3 months showed no improvement in left ventricle function with minimal reduction in symptoms.

Case 4:

A 27 year second gravida delivered by caesarean section for obstetric indication. There was history of pregnancy induced hypertension in the previous pregnancy. In this pregnancy she was normotensive. About 6 hours after LSCS the patient developed shortness of breath and chest discomfort. On examination she was restless, dyspnoeic and tachypnoeic. She had tachycardia with the heart rate of about 120 per minute and she was afebrile. Her blood pressure was 96/70 mmHg, respiratory rate of 32 per minute and oxygen saturation of 88% with 10 liters per minute of nasal oxygen. Cardiovascular examination was normal except for tachycardia. There was bilateral basal crepitations. Chest x-ray showed bilateral opacities suggestive of pulmonary edema.

There was no identifiable cause during initial evaluation. Electrocardiogram showed nonspecific T wave inversions. Echocardiogram showed hypokinesia of left ventricle with severe systolic dysfunction of left ventricle. Ejection fraction was 20% with non dilated left ventricle. The patient was treated with oxygen, intravenous nitrates, diuretics, low molecular weight heparin, ACE inhibitors and betablockers. During hospitalization her left ventricular systolic function was improved significantly and discharged in a stable state. Follow up echo after 1 month showed good left ventricular systolic function.

Case 5:

26 year old primigravida with twin gestation referred from peripheral hospital as severe preeclampsia. On admission the patient was dyspnoeic and tachypnoeic. She had tachycardia and gallop rhythm. After ruling out the common causes of acute onset of breathlessness, echocardiogram was done which showed global hypokinesia of Left Ventricle with ejection fraction of 20%. The patient was treated with inotropes, vasodilators, betablockers and anticoagulation. She responded to the treatment and follow-up after 1 month showed improved ejection fraction.

2. Discussion

Peripartum cardiomyopathy is less common but definitely life threatening heart failure of unknown etiology^{21, 22}. In acute onset of breathlessness, Obstetrician should suspect the diagnosis of PPCM especially those with risk factors in the late pregnancy and early postpartum. Management of this disease includes-principally diuretics, beta blockers, angiotensin-converting enzyme inhibitors, vasodilators, digoxin and anticoagulation in certain cases. Non drug measures like salt and water restriction in women with signs and symptoms of heart failure. The main aim of drug therapy is to reduce the symptoms of heart failure. Safety of drugs to breast feeding mothers should be ensured²³. Prognosis is related to improvement in left ventricle functions. There is very high mortality in subsequent pregnancies in those whose systolic function do not improve even after six months. Avoidance of subsequent pregnancy is necessary.

Age	22	20	30	27	26
Parity	Gravida 1	Gravida 2	Gravida 3	Gravida 2	Gravida 1 ,twin gestation
Comorbid illness	-	-	Previous H/O PPCM	Gestational hypertension	-
Trimester of pregnancy	1 st Post natal period	Antenatal, III trimester	1 st week postnatal period	1 st Post natal period	Antenatal, III trimester
Clinical presentation	Acute pulmonary edema	Cardiogenic shock	class II dyspnoea	Acute pulmonary edema	class II dyspnoea
LV function	Moderate LV systolic dysfunction, EF-35%	Severe LV systolic dysfunction, EF-20%	Moderate LV systolic dysfunction, EF-40%	Severe LV systolic dysfunction, EF-20%	Severe LV systolic dysfunction, EF-20%
Mode of delivery	Vaginal	-	Vaginal	caesarian	Vaginal
Maternal outcome		Maternal death			
Foetal outcome				Foetal death	

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