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Case Report

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PERIPARTUM CARDIOMYOPATHY: A CASE SERIES

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a type of heart failure of unknown etiology occurring late in pregnancy or in the postpartum period. It is a rare disease and because of its low incidence and unspecific symptoms PPCM is often undiagnosed or undetected. Unfortunately, underlying cause and natural history of PPCM is incompletely understood. Nevertheless, it is incumbent on the anaesthetists as well as on the intensivists to be cognizant of PPCM, due to its high maternal mortality and morbidity. A number of recent reviews have described in detail the pathophysiology, clinical picture and management of PPCM. We are presenting illustrative case series of peripartum cardiomyopathy and proceed to give a short comprehensive review.

KEY WORDS: PPCM, heart failure, echocardiography.

INTRODUCTION

Peripartum cardiomyopathy is a type of dilated cardiomyopathy seen from last month of pregnancy or in the first 5 months postpartum in patients with no demonstrable heart disease before the last month of pregnancy and no discernible etiology for heart failure[1], with incidence <0.1 % with high morbidity and mortality rates 5% – 32%.[2] Because of its low incidence and unspecific symptoms PPCM is often undetected or undiagnosed. The diagnosis is confirmed on the basis of diagnostic criteria[3]: a) development of the heart failure during the last month of pregnancy or within 5 months of delivery; b) absence of an identifiable cause for the heart failure; c) absence of recognizable heart disease prior to the last month of pregnancy; d) left ventricular dysfunction determined during echocardiography with ejection fraction <45% .[4] In this case series we review PPCM and present guideline for practice.

CASE 1

A 48 year old primigravida, twin pregnancy following IVF conception with 33weeks 6 days period of gestation was admitted to our hospital with chief complaints of difficulty in breathing, swelling of the lower limbs and pain abdomen on and off. Her Blood Pressure was 160/100mmhg at the time of admission with 86/min pulse rate regular, normovolumic and Spo2 99% in room air with no previous significant medical history of heart disease. Ultrasonography shows normal fetus,but blood examination reveal deranged liver enzymes, with normal renal function and coagulation profile and urine routine shows proteinuria 3+. Her thyroid profile is deranged with TSH-11.97µIU/ml (on thyronorm 25µg OD). She was managed conservatively for 3 days with antihypertensive drugs and betamethasone injection for fetal lung maturity. Patient developed acute respiratory distress at 34weeks 3 days, for which patient was shifted to ICU under mechanical ventilation as her Spo2 dropped to 40% with oxygen. Decision for termination was taken same day in ICU and patient underwent Caesarean Section. Two female babies weighing 1.9 and 2 kg respectively with normal Apgar score were delivered. PPH developed intra operatively secured with B lynch suture. 3 unit packed red blood cells and 1 unit fresh frozen plasma was transfused. Patient developed acute respiratory distress again on 6th postoperative day, managed conservatively. Echocardiography showing signs of dilated cardiomyopathy (ejection fraction 43%), Diastolic dimension 2.7cm/meter square, for which cardiologist consultation was taken and started on treatment with ACE inhibitors, beta blocker and diuretics. Patient was discharged on 18th post opervative day. The condition of the patient and both the babies were stable during the time of discharge.

CASE 2

A 32 year old female P1+0+0+1 post delivery day 6 came to labour room with severe breathlessness and severe diaphoresis with B.P. 170/110 mmhg, tachycardia (140/min) and spo2 36% witht O2.Respiratory rate was 30 breaths/ minute with bilateral basal crepts. There was no history of hypertension during antenatal visits and no

history of any heart disease before. Chest X-ray showed massive pulmonary interstitial edema with normal heart size. Laboratory studies shows hb 9.1 g/dl, TLC 16800 mm^3 and hypokalemia (3.2 mol/l). Patient's coagulation panel, renal and liver function parameters were normal. ECG shows sinus tachycardia otherwise normal ECG. Suspected diagnosis was severe preeclampsia with acute respiratory distress syndrome with septicaemia. Patient was kept under mechanical ventilation, diuretics and started on broad spectrum antibiotics with two packed red blood cell transfusion. Patient was extubated on 5 th day. Echocardiography findings show signs of dilated cardiomyopathy with ejection fraction 37%, and left ventricular diastolic dimension 3.9cm/m^2. The therapy was converted to oral medication with ACE inhibitors, beta blocker and diuretics under cardiology consultation. The patient was discharged on 16th day (21st day postpartum). The condition of the patient and baby was stable at the time of discharge.

Table 1: Showing laboratory values of the patients during the treatment

	Laboratories values at the time of admission and at the time of ICU discharge						
	CASE 1			**CASE 2**			
	Adm	**Day 4**	**discharge**	**Adm**	**Day1**	**discharge**	**Normal reference values**
WBC	8700	9300	8400	16,800	15,300	9800	3500-10000 cells/mcL
Hb	10.9	6.8	10.0	9.1	10	13.3	12.1-15.1 gm/dL
Platelets	1.2	1	2.5	2.82	3.0	5.15	1.5-4.5 L/dL
Sodium	135	144	138	140		135	135-145mmol/L
Potassium	3.5	4.0	3.4	3.2		3.5	3.5-5 mmol/L
Urea	16	24	20	30		14	1.2-3 mmol/L
Creatinine	0.6	0.8	0.6	0.8		0.6	0.8-1.3 mg/dL
AST	260	287	150	267			5-30 U/L
ALT	180	197	120	192			5-30 U/L

CASE 3

A 26 year old female P2+0+0+2 admitted in our hospital for pain abdomen due to ovarian cyst (10*6.5*11.4 cm) along with dyspnea and orthopnea for past 1 month. Her first pregnancy was carried upto term 4 year back and delivered a male baby by normal vaginal delivery which was a institutional delivery and in second pregnancy female baby was delivered vaginally at term 4 months back. No significant history of illness, surgery or heart disease during antenatal and postpartum period and was uneventful. No family history of heart disease. Her complete hemogram, ca ovary markers, renal and liver function, chest X-Ray were within normal limits but ECG was found to have non specificT Wave changes for which cardiology consultation was taken. Cardiac markers were negative for MI. ECHO was advised which shows dilated cardiomyopathy with ejection fraction 36% and left diastolic dimension of 3.2 cm/m^2. Patient was started on medication after cardiology consultation.

DISCUSSION

Despite many hypotheses, the cause and mechanism of pathogenesis of PPCM remain unknown. Early investigations proposed myocarditis as the cause for PPCM where antiinflammatory treatment resulted in clinical improvement.[5] Further studies failed to establish this causal link between myocarditis and development of PPCM.[6;7] Additionally, Felker et al. confirmed that the absence or presence of inflammation on endomyocardial biopsy tissue did not predict outcome in patients with PPCM.[8] Persistent viral antigen has been also postulated as a trigger for the development of PPCM. Presence of viral genomic material, including enterovirus (coxsackie virus), parvovirus B19, adenovirus and hepatitis virus was isolated in biopsy material of patients with idiopathic dilated cardiomyopathy. Further, the authors demonstrated an association between clinical improvement of left ventricular systolic function and viral clearing.[9] Despite this promising findings, further studies are needed to clearly identify virus infection as the cause for development of PPCM. Many animal and clinical investigations support to the hypothesis that immune activation, dysregulation of cellular apoptosis pathways and upregulated humoral immunity contribute to the pathogenesis of PPCM. Whereas the importance of raised high sensitivity c-reactive protein in plasma of patients with new and evolving PPCM merits additional evaluation, concentrations of the inflammatory cytokine TNF-alpha, elevated immunoglobulins against cardiac myosin as well as markers of apoptosis (Fas/Apo-1) are well correlated with left ventricular function and mortality. Additionally, animal data suggest that the cardiac myocyte-specific STAT3 pathway is necessary for protection of the heart from postpartum stress.[10] Taken all available data together, so far no cause has been clearly identified for the development of PPCM.

CONCLUSION

Peripartum cardiomyopathy is an uncommon complication of pregnancy with unknown cause and potentially life-threatening complications. Because of its low incidence and unspecific symptomps PPCM is often undetected or misdiagnosed. To achieve successful pregnancy outcome, a clear understanding of hemodynamic adaptation that occur in pregnancy, meticulous maternal and foetal surveillance for risk factor and complications throughout the antepartum, intrapartum, and postpartum period as well as a multidisciplinary approach is essential.

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