www.jmscr.igmpublication.org Impact Factor 3.79
ISSN (e)-2347-176x

Journal Of Medical Science And Clinical Research
IGM Publication An Official Publication Of IGM Publication

Management of Peripartum Cardiomyopathy in ICU: A Case Report

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ABSTRACT

A patient presenting with acute onset breathlessness in late pregnancy or immediately after delivery brings in mind a number of conditions such as pulmonary edema, pulmonary aspiration or amniotic fluid embolism.1 One of the condition presenting similarly is peripartum cardiomyopathy. A disease that can endanger the life of the mother and the baby and can cause prolonged and persistent cardiac insufficiency in mother prematurely due to decreased cardiac ejection fraction either in late pregnancy or early puerperium. A 23yr old female who had delivered vaginally the previous day came with complaints of shortness of breath for five days and cough with expectoration for five days. On examination general condition was poor, features of heart failure were present. She was shifted to ICU where she required intubation. Later on ECHO revealed dilated left ventricle with global hypokinesia, LVEF-25% and diagnosis of peripartum cardiomyopathy was made. Treatment was started with digoxin, fursemide, enalapril.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopthy that is defined as deterioration in cardiac function between last month of pregnancy upto 5 months postpartum.[2] It involves systolic dysfunction of heart and is associated with decrease in left ventricular ejection fraction and congestive heart failure associated with increased risk of arrhythmias, thromboembolism, pulmonary edema and even sudden cardiac death.

The etiology of PPCM remains elusive, however, viral, autoimmune, hemodynamic stress of pregnancy, cytokine-mediated inflammation, Gq-related myocyte apoptosis, oxidative stress-induced Cathepsin D production and selenium deficiency causes are being hypothesized. Other possible causes include nutritional deficiencies, small vessel coronary artery disease, excessive salt intake and peripartum fluid shifts.[3]

Predisposing factors include maternal age more than 30 years, multiparity, eclampsia, obesity, racial factors, hypertension, malnutrition and prolonged tocolysis.[4] Our patient had clinical features of acute congestive cardiac failure and echocardiographic changes consistent with PPCM. Management is mainly symptomatic and aims to reduce afterload and preload and enhance myocardial contractility and reduce arrythmogenicity. Therefore, our patient was also managed with bed rest, digoxin, loop diuretics, vasodilators, beta blockers and ionotropic support.

CASE REPORT

A 23year old, G1P0A0 housewife presented with complaints of shortness of breath and cough with sputum since five days at term to labour room casualty. Patient had difficulty in breathing since five days which was present at rest and increased on lying down. Patient also had cough since five days which was productive in nature and was associated with chest pain. Sputum was white in colour and not blood stained. She did not give any history of syncopal attacks, giddiness or change in voice. She denied any history of arthritis, fever or joint pain. She had no history of previous hospitalization or any chronic drug intake. Her antenatal period was uneventful except for last five days.

On the day of presentation in emergency department, patient was conscious, oriented but restless. Her heart rate was 98 min^{-1}, BP was 126/96mm Hg, respiratory rate was 28min^{-1}. No jugular venous distension, pallor, lymphadenopathy was appreciated. Her cardiovascular examination revealed normal heart sounds with no added murmurs but patient had tachycardia. On auscultation bilateral rhonchi were heard. Nervous system examination was normal.

On second day, patient delivered a baby boy vaginally after induction of labour under cover of steroids. After delivery patient started complaining of increased difficulty in breathing and was immediately shifted to RICU. In RICU all routine monitors comprising of ECG, NIBP, SpO_2 were attached. Oxygen through ventimask was given. Sample for all routine biochemistry investigations and blood gases were sent and revealed Hb-9.4 g, TLC-23000mm^{-3}, DLC-88/10/1/1, Platelet count-3.5 lakhs mm^{-3} and peripheral smear was normal. Her BGA was pH-7.377, pO_2-91.2, pCO_2-23.1, HCO_3-13.2, BE- -9.7 and oxygen sat-96.5%. Her serum electrolytes, blood sugar and liver function tests were normal except blood urea which was 60mg/dl and serum uric acid which was 8.9 mg/dl. Prothrombin time was 39.2 seconds and I.N.R was 3.63. Urine examination was positive for albumin. ECG revealed sinus tachycardia and LAD.

After her respiratory distress increased with a respiratory rate of 44/min. She became disoriented. Patient was intubated after giving morphine and midazolam with standard oral endotracheal tube and mechanical ventilation was started.

She developed hypotension for which dopamine infusion was started. Cardiology call was sent and bedside ECHO was done which revealed dilated left ventricle with global hypokinesia and ejection fraction of 25% and mild MR. Her chest X-ray revealed massive cardiomegaly and provisional diagnosis of peripartum cardiomyopathy was made.

Initially patient was started on Inj. Digoxin 0.5 mg iv stat followed by Tab. Digoxin 0.25mg once a day for 5 days per week, Inj Lasix 20mg 8hrly, Tab. Enalapril 2.5mg 12hrly. Nebulisation with duolin was done twice a day. Inj. Kcl 40 mEq per day. Daily electrolyte, BGA, Kidney function test(KFT), ECG monitoring was done. Her dopamine infusion was titrated and stopped gradually when blood pressure came within normal limits. After 1 day patient was weaned off and given T-piece trial and was subsequently extubated. Oxygen through ventimask was given. Her total leucocyte count was 16000/mm^3, blood urea was 60mg/dl, PT was 23.4 seconds, INR 1.95 and her electrolytes were normal. Inj. Lasix dose was increased to 40mg 8 hrly. Oral digoxin and Tab. Enalapril was continued.

Daily BGA, KFT, electrolytes were sent. Her blood urea and serum creatinine gradually increased but electrolytes and BGA were normal. Tab. Enalapril was stopped and Tab. Isonitrate 20mg twice a day was started. Her chest condition improved. After five days patient was shifted to ward with strict advice to continue treatment.

DISCUSSION

Peripartum cardiomyopathy (PPCM) is an unusual form of dilated cardiomyopathy which manifests as acute heart failure in the last trimester of pregnancy or early postpartum period.[5]

One of the recent definitions of PPCM has been provided by the Heart Failure Association of the European Society of Cardiology Working on PPCM which describes it as " an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found. It is the diagnosis of exclusion. The left ventricle may not be dilated but the ejection fraction is nearly always reduced below 45%.[6]

Many risk factors have been implicated in the development of disease condition and include multiparity, eclampsia, maternal age more than 30 years, racial factors, hypertension, malnutrition and prolonged tocolysis.[4]

Many causes are being hypothesized which include viral, autoimmune,cytokine mediated inflammation, oxidative stress-induced Cathepsin D production, and many others. Viral myocarditis has been proposed as the main mechanism for PPCM and was first reported by Goulet et al.[11] This proposal was later supported by Melvin et al,[12] who found myocarditis during

endomyocardial biopsy in 3 women with PPCM. The biopsy specimens had dense lymphocytic infiltration with a variable amount of myocytic edema, necrosis, and fibrosis.

Apoptosis (programmed cell death) of cardiac myocytes occurs in heart failure and may contribute to progressive myocardial dysfunction. Experiments in mice suggest that apoptosis of cardiac myocytes has a role in peripartum cardiomyopathy.[13]

Lower levels of selenium have been found in patients with PPCM. Autoantibodies against myocardial proteins have been identified in patients with PPCM but not in those with idiopathic cardiomyopathy[14].

CRITERIA FOR DIAGNOSIS OF PERIPARTUM CARDIOMYOPATHY[4]

- Development of heart failure in the last month of pregnancy or within 5 months postpartum
- Absence of an identifiable cause of heart failure.
- Absence of recognizable heart disease before the last month of pregnancy.
- Left ventricular systolic dysfunction demonstrated by left ventricular ejection fraction of less than 45%, fractional shortening of less than 30%, or both, with or without a left ventricle end diastolic dimension less than 2.7 cm/m^2 of body surface area.

Clinical features are similar to those seen in any cardiac failure. Women in either third trimester or early peurperium presenting with:-

- Rapid heart beat
- Chest pain
- Difficulty in breathing
- Swelling of feet or whole body
- Excessive fatigue
- Tiredness during physical activity
- Cough
- Paroxysmal nocturnal dyspnea or orthopnea

All these features should raise the doubt of PPCM, especially when the prenatal history has been otherwise uneventful.[15]

In our case the patient presented with complaints of shortness of breath and cough with expectoration at term without any previous positive history. Her shortness of breath increased immediately after delivery of the baby.

Physical examination often reveals tachycardia and tachypnea, blood pressure may be elevated or reduced, and patients are often not able to lie down flat because of shortness of breath, as was seen in this patient. There is usually increased jugular venous pressure, displaced apical impulse, right ventricular heave, murmurs of mitral and tricuspid regurgitation, third heart sound, pulmonary rales, and peripheral edema.

Electrocardiography usually shows sinus tachycardia with nonspecific ST-T wave changes. LV hypertrophy can be found as well as left atrial enlargement and, occasionally, conduction abnormalities including left bundle brunch block [16].

Chest radiography usually shows cardiomegaly and pulmonary venous congestion or pulmonary edema, with or without pleural effusion [16,17].

Echocardiography shows variable degrees of LV dilatation, with moderate to severe depression of systolic function. Right ventricular and biatrial dilatation as well as moderate to severe mitral and tricuspid regurgitation are commonly seen, with increased pulmonary pressures and mild pulmonary regurgitation[16,17,18].

In this patient bedside ECHO was done which revealed dilated left ventricle with global hypokinesia and ejection fraction of 25% and mild MR. Her chest X-ray revealed massive cardiomegaly .

Cardiac magnetic resonance imaging (MRI) has been used in a limited number of patients for the assessment of cardiac function and the detection of mural thrombi or myocardial fibrosis.[19,20] Although MRI is probably safe during pregnancy , intravenous gadolinium crosses the placenta, and the 2007 American College of Radiology document on safe MRI practices recommends that it be avoided during pregnancy and used only if absolutely essential [21].

The goals of medical management in a patient diagnosed with PPCM should include measures to improve oxygenation and maintain cardiac output so as to improve both maternal and fetal outcome. Interventions are required to decrease both preload and afterload as well as to improve cardiac contractility.

Strict bed rest of 6-12 months, as advocated earlier, is associated with lower incidence of cardiomegaly, but the same can be achieved without bed rest. Infact it may predispose to deep vein thrombosis, and subsequently pulmonary embolism. Salt and fluid to be restricted to 2-4 gm/day are important in symptomatic improvement.[4]

Mild to moderate symptoms may be managed with rest, salt restriction and diuretic therapy. Oxygen may be instituted via face mask, or continous positive airway pressure may be applied upto a level which does not jeopardize the cardiac output. Hydralazine and nitrates decrease afterload and are mainstay of treatment in pregnant patients with heart filure. ACE inhibitors ,both direct acting or receptor blockers, are first line of treatment in patients with heart failure, are however contraindicated in pregnancy. Digoxin may be indicated in certain patients for its ionotropic effect. Although it is a safe drug in pregnancy and peurperium, its plasma level should be strictly controlled with close monitoring.

This patient was started on Inj. Digoxin 0.5 mg iv stat followed by Tab. Digoxin 0.25mg once a day for 5 days per week, Inj Lasix 20mg 8hrly, Tab. Enalapril 2.5mg 12hrly. Daily electrolyte, BGA, Kidney function test(KFT), ECG monitoring was done. Patient was subsequently extubated. Oxygen through ventimask was given. Oral digoxin and Tab. Enalapril was continued.

Tab. Enalapril was stopped and Tab. Isonitrate 20mg twice a day was started. Her chest condition improved. After five days patient was shifted to ward with strict advice to continue treatment.

Anticoagulation is recommended in patients with PPCM , especially if the ejection fraction is less than 35% and there are other associated risk factors. Warfarin is teratogenic in early pregnancy and can cause fetal warfarin syndrome while

unfractionated heparin has low bioavailability in pregnant patients and is associated with thrombocytopenia. Thus, low molecular weight heparins are preferred in pregnancy.

Patient presenting in acute failure usually be best managed in an intensive care unit in propped up position with continous hemodynamic and oxygenation monitoring. In case invasive ventilation is required, precautions for aspiration in pregnant women must be taken. Loop diuretics may be more efficient in acute failure. Dobutamine, dopamine or milrinone can be used to provide ionotropic support.

CONCLUSION

PPCM is a rare disease of unknown cause that affects women in their child bearing age. Its diagnosis is challenging and of exclusion which requires high index of suspicion.[5] Principles of therapy remain more or less same as that of heart failure due to any cause but careful selection of drugs is important in case of pregnant and lactating mother.. No matter whatever is the pathology prompt resuscitation is the first priority and includes optimization of myocardial contractility, preload and afterload. Prognosis depends on reversal of ventricular dysfunction.

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