Successful Maternal and Perinatal Outcome in Peripartum Cardiomyopathy – Our Experience

Dr. Mala Srivastava, Dr. I. Ganguli, Dr. Mamta Dagar, Dr. Ashwani Mehta, Dr. J.P.S. Sawhney

ABSTRACT

Background: Peripartum Cardiomyopathy is a rare but grave complication of pregnancy. It is one of the less studied and investigated conditions, which is difficult to diagnose and treat, with quite high morbidity and mortality.

The incidence of peripartum cardiomyopathy varies from 1 in 1300 to 4000 pregnancies. The mortality rates ranges from 25-50%. In the last four years, six cases of Peripartum Cardiomyopathy has been diagnosed and managed in our unit. Presentation of these cases were different. Clinical suspicion of the condition helped in early diagnosis and prompt management of the patients. The etiology of peripartum cardiomyopathy is unknown, but inflammatory cytokines, myocarditis, viral, auto-immune and idiopathic causes may contribute.

Cases: In the first case patient had elective LSCS and she collapsed during LSCS and was later diagnosed as a case of peripartum cardiomyopathy.The second case had LSCS for BOH with term pregnancy, peripartum cardiomyoathy in this case also developed after LSCS. The third case was diagnosed peripartum cardiomyopathy antenatally .She had twins with anemia, LSCS was done in view of both fetuses presenting by breech at term. The fourth case had an emergency LSCS under GA but took longer time for reversal- unexplained metabolic acidosis, tachycardia persisted with low O2 saturation. Cardiologist consulted and echo confirmed the diagnosis of PPCM. The fifth case had NVD & PP ligation. On 6th post-op day patient had sudden collapse and diagnosis of PPCM was made. The sixth case had term pregnancy with PIH and dyspnoea. On investigation she was diagnosed as PPCM antenatally and managed accordingly. All the patients were managed conservatively. Five of them are well and alive and their LVEF has improved upto 65%, whereas one patient still has LVEF of 25% even after four years.

Dept of Obs & Gynaec,
Sir Ganga Ram Hospital, New Delhi

✉ Dr Mala Srivastava

Conclusion: Fortunately maternal peripartum cardiomyopathy is infrequent but serious complications of human pregnancy, that threatens both mother and fetus . Clinicians must be aware of this problem in order to provide prompt diagnosis & management. The successful outcome is possible with an intensive treatment. This condition tends to recur in subsequent pregnancy. A minimal interval of 3 yrs after the recovery of function has shown to be safe for subsequent pregnancies , in consultation with a cardiologist.

Keywords: peripartum, cardiomyopathy, LSCS, conservative, maternal echo.

Introduction

Peripartum cardiomyopathy is a rare but grave complication of pregnancy . It is one of the less studied and less investigated conditions, which is difficult to diagnose and treat, with quite high morbidity and mortality even in best centres.

Hibbard et al from Chicago in 1999[1] has emphasized for uniform criteria that defined peripartum cardiomyopathy. The new definition include heart failure within the last month of pregnancy or 5 months postpartum with the absence of preexisting heart diseases or with no determinable etiology with the echocardiographic criteria of left ventricular dysfunction, with ejection fraction less than 45% or M -mode fractional shorting less than 30% or both and end diastolic dimensions more than 2.7%cm / m^2.

Tateda K. etal in 2000 AD[2] reported that the incidence of Peripartum Cardiomyopathy varies from 1 in 1300 to 4000 pregnancies. The mortality rates of this disorder in the acute and subacute phases range from 25% - 50% The prognosis is especially poor in-patients with cardiomegaly persisting more than 6 month and with low left ventricular ejection fraction.

In last four years six cases of Peripartum Cardiomyopathy had been diagnosed and managed in our unit. The successful maternal and perinatal outcome in this series is presented here.

Case 1

31 yrs old, G2 P 1+0. with previous LSCS term pregnancy admitted for elective caesarean section .

Patient was a diagnosed case of bronchial asthma for last 1 month on medication.

She had LSCS under spinal anesthesia. As soon as the baby was delivered, patient developed severe hypotension not responding to IV fluids, blood volume expander, and mephentine. Patient was intubated, put on intermittent positive pressure respiration and steroids. Since, she was not maintaining her BP and oxygen saturation, she was shifted to ICU. She stabilized in 8-10 hrs, but her tachycardia persisted . She was put on ventimask from BIPAP after 12 hours. Patient was put back on BIPAP support as her oxygen saturation fell to 85%, she was dyspnoeic and her CVP was 22cms.

ECG was done and showed nonspecific ST changes. Echo showed generalized hypokinesia . LVEF 30% severe MR, TR with PAH.

Diagnosis of peripartum cardiomyopathy was made. She responded to ACE inhibitors, digoxin, antibiotics, low molecular weight heparin and lasix and was discharged on 10th post op day in stable condition..

Patient still has LVEF of 30-35% even after four years, and continues to be on treatment for cardiomyopathy.

Case 2

28 yrs old G5P0A4 36+1 weeks amenorrhea with BOH admitted with pregnancy induced hypertension breech presentation and decreased fetal movements, so she had caesarean section done under general anaesthesia .

Through out surgery patient had tachycardia. Patient was extubated but was not maintaining Oxygen saturation, so cardiologist was consulted.

Since her antenatal, preoperative echo was normal, so diagnostic dilemma persisted.

She continued to have tachycardia and low oxygen saturation.

Her ECG was done which showed fresh Right Bundle Branch Block, and then echo was done which showed global left ventricular hypokinesia with LVEF

of 35% with mild MR. Diagnosis of Peripartum Cardiomyopathy was made Patient was shifted to ICCU and managed conservatively with digoxin, antibiotics, fragmin and lasix.

She had improved, and after 3 months her LVEF was 65% and complete resolution of symptoms.

She came after ten months with a pregnancy of six weeks and medical termination of pregnancy was done.

Again she was pregnant after a gap of one year, and this time an elective LSCS was performed with an uneventful antenatal and postnatal period.

Case 3

26 yrs old primigravida with twin pregnancy presented at 26 weeks of amenorrhoea admitted with severe anemia, she was investigated and managed conservatively.

Her echo showed mild MR with LVEF of 65%. This patient at term was posted for elective caesarean section in view of both fetuses presenting by breech .

On the preoperative day she again developed dyspnoea. Her echo was done which showed global left ventricular hypokinesia with LVEF of 30% and the diagnosis of Peripartum Cardiomyopathy was made.

With due precaution caesarean was done, and patient shifted to ICCU as a precautionary step and managed conservatively.

Case 4

36 yrs old G2 P1 with 36+5 wks amen. admitted with leaking P/V. She was booked antenatally and had dry irritating cough with bilateral bronchial breath sounds for last three weeks. She was taken up for emergency LSCS under GA in view of cord prolapse. Surgery was uneventful but the patient took long time to recover from anaesthesia. Unexplained metabolic acidosis and tachycardia persisted with low oxygen saturation. X-Ray chest showed bilateral homogenous density in the upper mid zone .Cardiologist consulted, Echo was done, which showed LVEF of 30%, mild MR TR the diagnosis of peripartum cardiomyopathy was confirmed. She was managed conservatively and discharged in fair condition.

Case 5

29 yrs G3P2 admitted at term pregnancy with labour pains. Had NVD and had PP ligation. She had breathlessness in the post partum, and was managed with nebulisations and bronchodilators. On the 6th post-operative day patient had sudden collapse and was shifted to ICU. Her echo was done – LVEF of < 25%.Diagnosis of PPCM done and managed accordingly.

Case 6

25 yrs old G2P1 presented at 37 weeks with PIH and dyspnoea, she was given treatment for bronchitis, but cough persisted. Cardiologist consulted and echo done. Her echo was done which showed global left ventricular hypokinesia with LVEF of 30% and the diagnosis of Peripartum Cardiomyopathy was made. She was stabilized by anti- hypertensives, digoxin, she went into spontaneous labor and delivered normally

Discussion

Presentation of all these cases were different. In the first case we were taken unawares, our patient was being treated as a case of bronchial asthma, and we took time to diagnose her as a case of Peripartum Cardiomyopathy.

Second case had an antenatal echo done, as the patient complained of breathlessness & palpitation in the antenatal period.

In the post op period since the patient was not maintaining her Oxygen saturation so echo was done and she was diagnosed as a case of peripartum cardiomyopathy.

Third case had anemia at 26 weeks of amenorrhoea with dysnoea, echo was done despite the fact that her dyspnoea which could have been explained by her anemia. But before her elective LSCS she developed dyspnea again, an echo was repeated and the diagnosis of PPCM was made.

We could then take adequate precaution for our patient during LSCS and postpartum period.

The fourth case was diagnosed as chronic bronchitis antenatally. But in the post – operative period her low oxygen saturation persisted. On thorough investigation she was diagnosed as PPCM.

The fifth case was also treated as a case of chronic bronchitis unless she collapsed and was shifted to ICU, & her subsequent echo proved the diagnosis.

The sixth case was diagnosed in the antenatal period with peripartum cardiomyopathy and went into spontaneous labor and delivered normally

Clinical suspicion was important in all the cases. It helped us in early diagnosis and prompt management of the patients.

Discussion:

Peripartum cardiomyopathy is a less recognized, infrequent but serious complication during pregnancy, of unknown etiology and is associated with excess morbidity and mortality in women of child bearing age.[3]

The incidence of peripartum cardiomyopathy varies from 1 in 1300 - 4000 pregnancy

The mortality rates of this disorder in the acute phase and subacute case range from 25% to 50%. The prognosis is especially poor in patients with cardiomegaly persisting more than 6 months, and in patient with low left ventricular ejection fraction.

The etiology of peripartum cardiomyopathy is unknown but viral, autoimmune and idiopathic causes may contribute.[4]

The risk factor includes multiple gestation, pregnancy induced hypertension, multiparity, advanced maternal age, caesarean section and African - American women. Other possible etiologic factors include prolonged tocolysis, proinflammatory cytokines (TNF, IL1, IL6). Abnormalities of relaxin, and ovarian hormone produced during pregnancy, can cause positive inotropic and chronotropic properties and cause excessive relaxation of the cardiac muscles. It is unclear, that whether nutritional deficiency may play a role in the pathogenesis of PPCM.

The cardinal symptom are that of easy fatigability, tachypnoea, orthopnoea, varying degree of dyspnoea and features of pulmonary oedema.

The examination reveals raised jugular venous pressure, pulmonary rales, cardiomegaly and third heart sound.

The murmur of mitral or tricuspid regurgitation may be audible, there may be peripheral oedema.

Since these clinical features are common to many other disease as well as to underlying heart disease, it is important to rule out all other possibilities before diagnosing peripartum cardiomyopathy.

Diagnostic criteria:

ECG - only non specific ST-T wave abnormalities, arrhythmias or an infarct pattern.

Echocardiography: characteristically shows an enlarged heart with global hypokinesia. The ejection fractions are typically 15-25% in symptomatic patients.

Chest X-Ray shows an enlarged heart, elevated hemi-diaphragm with a fluffy pulmonary infiltrate, worse at hilum which becomes less obvious towards the periphery.

Arterial blood gases show: Significant hypoxia, normal ph, low to normal Pco2.

The objective deficit is one of failure to oxygenate, rather than failure to ventilate.

CVP is raised, and CWP (Pulmonary capillary wedge pressure) is raised.

If diagnosed in the antenatal period, prompt delivery depending on the maturity of the foetus should be considered.

Immediate and vigorous diuretic therapy is important, digoxin, anticoagulants, inotropic agents and many hypertensive drugs for after load reduction are somewhat more controversial interventions .

Management of such patients includes multidisciplinary approach.

Bolis et al from Italy 1999[5] reported the case of a women presenting with severe cardiac failure immediately after caesarean section just as in our first case.

The diagnosis of Peripartum Cardiomyopathy was difficult due to the presence of pre-eclampsia and acute pulmonary oedema which occured four hours after delivery.

About 50% - 60% of patients have spontaneous recovery of cardiac function within six months of onset. The remainder either have persistent ventricular dysfunction or deteriorate, to die early or to receive cardiac transplantation.

There is a tendency towards recurrence with subsequent pregnancy.

Hence, a previous history of peripartum cardiomyopathy is a relative contraindication to repeat pregnancy in mothers who have recovered normal cardiac functions, and an absolute contraindication to pregnancy if there is persistent left ventricular dysfunction.

Albanesi et al 1999[6] concluded a study on 34 patients to assess the effect of subsequent pregnancy after Peripartum cardiomyopathy on maternal and fetal outcome. They concluded that subsequent pregnancy are well tolerated after Peripartum cardiomyopathy but not devoid of risk. A minimum of 3 years after recovery of function seem to be safe for subsequent pregnancies.

But our second patient despite all advise was pregnant in quick successions, but we managed to take her safely through term and deliver her.

CONCLUSION

Fortunately maternal peripartum cardiomyopathy is infrequent but serious complications of pregnancy, that threatens both mother and fetus. Clinicians must be aware of this problem in order to provide prompt diagnosis and management. The successful outcome is possible with an intensive treatment which ensures a favorable return of normal left ventricular function. This condition tends to recur in subsequent pregnancy.

A minimal interval of three years after the recovery of function has shown to be safe for subsequent pregnancies, in consultation with a cardiologist.

References:

1. Hibbard JU, Lind heime M., Lang RM. A modified definition for Peripartum cardiomyopathy & pragnosis based on echocardiography .Obst & Gynae .94 (2): 1999 August; 311-6.
2. Tateda K Ishii Y, Hirasawa K Peripartum Cardiomyopathy (Review) Nippon Rimsho – Japanese Journal of clinical medicine 58(1):2000 Jan; 166-70.
3. Mehta NJ, Mehta RN, Khan IA et al in 1999 .Peripartum cardiomyopathy : Clinical & therapeutic aspects. Angiology 2001, Nov ; 52(11) : 739 – 62.
4. Heider AL, Kullar JA Strauss RA .WeIIs SR .Peripartum cardiomyopathy a review of the literature . Obst & Gynae survey 54(8): 1999 Aug ; 526 –31.
5. Bolis C, Prothi S., Piantanide S, Rana R , Cantoin A ,Cuccia E, Rivath G : Case report with literature review. Minerva anestesiologica . 65(9): 1999 Sept. 665-73.
6. Albanesi Filho FM,. Da Silva TT. Natural course of subsequent pregnancy after peripartum cardiomyopathy. Arquiras brasiloiros de cardiologie, 73(1):1999 July; 47-57.