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Peripartum Cardiomyopathy- A Case Report

M. KAAVYA[1] and K. SARASWATHI[2]

Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Bharath University, Chrompet, Chennai, India.
*Corresponding author E-mail: dr.kaavya87@gmail.com

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ABSTRACT

Peripartumcardiomyopathy is a unusual form of dilated cardiomyopathy with unknown etiology , that can be fatal in young women but with prompt diagnosis and intensive supportive measures can successfully be treated with goodlongterm prognosis in 90% cases.We report a previously asymptomatic women with no risk factor presented with pulmonary odemaon day three of cesarean section who is now under periodic ECHO follow up to assess the recovery in cardiac function.

Key words: Peripartum, Women, Asymptomatic.

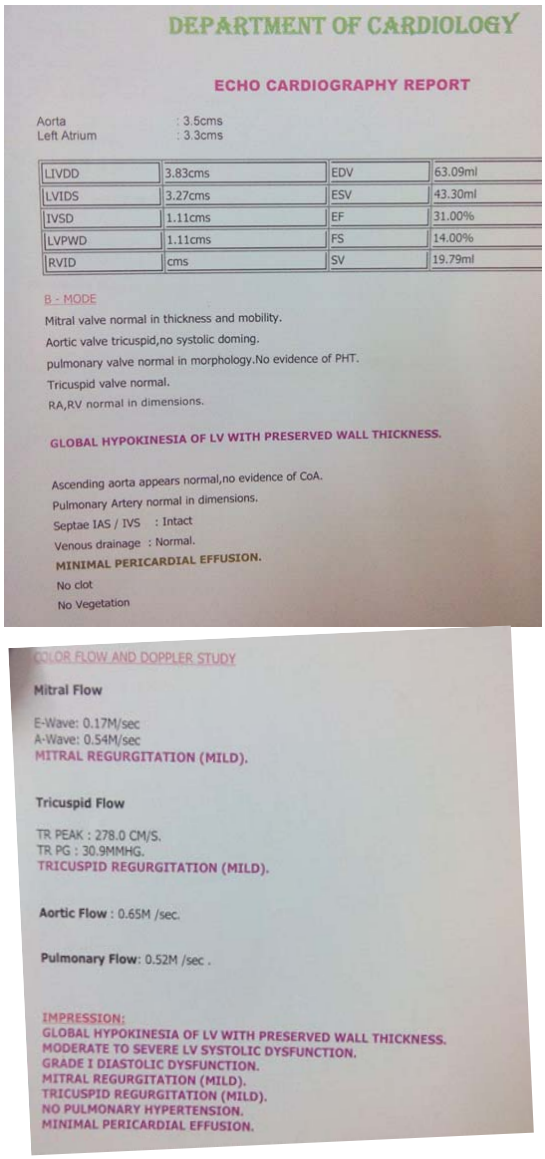
INTRODUCTION

Peripartum cardiomyopathy is a unusual form of dilated cardiomyopathy with unknown etiology[(1)] , that can be fatal in young women but with prompt diagnosis and intensive supportive measures can successfully be treated with good longtermprognosis in 90% cases[(2)] . The diagnostic criteria of peripartum cardiomyopathy : 1) development of cardiac failure in the last month of pregnancy or within 5 months after delivery , 2) absence of an identifiable cause for the cardiac failure , 3) absence of recognizable heart disease prior to the last month of pregnancy 4) left ventricular systolic dysfunction by classic echocardiographic criteria such as depressed ejection fraction or fractional shortening along with a dilated left ventricle . [(3)]We report a previously asymptomatic women with no risk factor presented with pulmonaryodemaon day three of cesarean section who is now under periodic ECHO follow up to assess the recovery in cardiac function.

Case report

Mrs.x,28 yrs old,married for two years ,Primi , GDM on meal plan,came to us for safe confinement. Booked andimmunisedoutside.First visit toSreeBalajiMedical College &Hospital was at 40 weeks. Menstrual H/O- Age at menarche-14yrs,regular cycles,3/30days,not associated with clots & pains.Marital H/O: Married for two years ,Nonconsanguious marriageObstetric H/O:1st Trimester:patient was started on Tab Susten which was taken till 34 weeks, Rest of the trimester uneventful.2nd Trimester:OGCT=155mg/dl,Therefore patient was started on meal plan,Rest of the trimester Uneventful.3rd Trimester: h/o Tab Susten was taken till 34 weeks, Rest of the trimester uneventful.Past H/O:Nil significant.Personal H/O:Normal bladder& bowel habits.Family H/O: Nil significant.On examination:Gc Fair, afebrile,not pale, B/L pitting pedal odema+,no cyanosis,not icteric,no clubbingCVS: S1S2 + RS:NVBS +,P/A-Uterus Term, Not Acting, head unengaged,FHS-Good,P/V-Cx mid position, Ext OS patulous, IntOS admits two finger, Membranes present,vertex at brim can be pushed down,pelvis adequate. Investigations: Haemoglobin-10.8gms,Urine albumin& sugars –Nil OGCT =155mg/dl, FBS-75mg/dl,PPBS-119mg/dl,HbA1c-5.5 %,Serology-negative,TSH 2.87uIU/ml Blood Group-B positive ,USG on 26/06/2015- SLIUG GA= 38-39 wks,AFI=7-

8cm,placenta posterior grade III,FL-7.6cm,EFW-3.59 kg.CerviprimeInduction was done on 26/06/2015 at 5.00pm as patient was on her due date with oligohydramnios.After 6hrsof induction,patient spontaneously ruptured her membranes. P/V-Cx 50% effaced,Os 2 cm dilated, membranes absent,vertex at -3 station, moderate meconium stained liquor drainingpv .Patient was taken up for emergency LSCS in view of Meconium stained liquor&fetal distress.Patient delivered an alive male baby on 26/06/2015 at 11.50pm with B.wt 2.8kg with goodapgar 8/10,9/10.On 3rd POD at 9.35am Patient c/o acute breathlessness.O/E- patient dyspneic,Tachypneic, mild pallor+ , B/L pedal odema+CVS:S1S2+ RS: B/L coarse extensive crepitations+,R.R-40/min,P.R-140/min,B.P-170/130mmHg,Spo2= 60-70 % in room air,PATIENT WAS SHIFTED TO ICU FOR FURTHER MANAGEMENT,Patient was started onInj.Lasix60mg I.V stat,Inj.Morphine 5mg I.V given,ECHO shows features suggestive of peripartumcardiomyopathy with moderate to severe LV dysfunction,ECG shows Sinus Tachycardia, Chest X-ray: B/L homogenous opacity more on right sidePatient was on NIPPV with Fio2 0.5 &Cpap8/15mmHg,Patient was treated with the following drugs: Inj.Lasix 3mg/hr infusion, Tab.Lanoxin0.25mg ½ OD,Tab.Flavedon MR 35mg BD,Tab.Neurokind LC BD,Tab.Ivabrad5mg TDS, Tab.Envas 2.5mg ½ OD,Along with Inj.Taxim1gm I.V BD aspost operativeantibiotics .Patient was symptomatically better&was shifted back to ward from ICU on 5th POD . She was on the following medications ,and she was covered withInj.Heparin5000 units S/C BD for 5 days.Fluids wererestricted to 800ml/day.She was given Duolin Neb 8thhrly.She wason O2withnasal prongs 2to4litres(sos).Tab.Lasix 40mg 1 OD,On (7th POD),Tab.Envas was stopped &Tab. Metoprolol 25mg ½ BD was given.Tab.Lasix40mg ½- ½ - 0, Tab.Ivabrad5mg reduced to BD dose. On (10TH POD)-fluids restricted to 1.5L/day, Tab.Lasix40mg ½-0-0, alternate suture removal was done . On (11th POD)-complete suture removal was done .On (14th POD)-patient was discharged at request.Patient wasadvicedto do repeat ECHO after one week. Patient wasadvicedto continue the following drugs on discharge, Tab.Metoprolol 25mg 1/2 BD, Tab.Lanoxine 0.25mg ½ OD,Tab.Lasix 40mg ½ OD,Tab.Enalapril 2.5mg ½ BD.



DEPARTMENT OF CARDIOLOGY

ECHO CARDIOGRAPHY REPORT

Aorta : 3.5cms
Left Atrium : 3.3cms

LIVDD	3.83cms	EDV	63.09ml
LVIDS	3.27cms	ESV	43.30ml
IVSD	1.11cms	EF	31.00%
LVPWD	1.11cms	FS	14.00%
RVID	cms	SV	19.79ml

B - MODE

Mitral valve normal in thickness and mobility.
Aortic valve tricuspid,no systolic doming.
pulmonary valve normal in morphology.No evidence of PHT.
Tricuspid valve normal.
RA,RV normal in dimensions.

GLOBAL HYPOKINESIA OF LV WITH PRESERVED WALL THICKNESS.

Ascending aorta appears normal,no evidence of CoA.
Pulmonary Artery normal in dimensions.
Septae IAS / IVS : Intact
Venous drainage : Normal.
MINIMAL PERICARDIAL EFFUSION.
No clot
No Vegetation

COLOR FLOW AND DOPPLER STUDY

Mitral Flow

E-Wave: 0.17M/sec
A-Wave: 0.54M/sec
MITRAL REGURGITATION (MILD).

Tricuspid Flow

TR PEAK : 278.0 CM/S.
TR PG : 30.9MMHG.
TRICUSPID REGURGITATION (MILD).

Aortic Flow : 0.65M /sec.

Pulmonary Flow: 0.52M /sec .

IMPRESSION:
GLOBAL HYPOKINESIA OF LV WITH PRESERVED WALL THICKNESS.
MODERATE TO SEVERE LV SYSTOLIC DYSFUNCTION.
GRADE I DIASTOLIC DYSFUNCTION.
MITRAL REGURGITATION (MILD).
TRICUSPID REGURGITATION (MILD).
NO PULMONARY HYPERTENSION.
MINIMAL PERICARDIAL EFFUSION.

ECHO Report of the patient

DISCUSSION

Though peripartumcardiomyopathy is relatively a rare disease (0.1% of pregnancies) it can lead to devastingconsequences with overall morbidity mortality rates as high as 5 to 32%.It is estimated that the incidence of peripartum cardiomyopathy is between 1 in 2500 to 1 in 15,000 live births[3]. Etiology remains unknown other potential causes 1) viral myocarditis 2) cardiovascular stress of pregnancy 3) inflammatory response in pregnancy – elevation of TNF Alpha

& IL-6 , 4) pathologic autoimmune response to fetal cells that lodge in the maternal circulation & cardiac tissue. 5) nutritional deficiencies - selenium [3] . Risk of Peripartum cardiomyopathy can occur in woman (age of parity) either young or elderly gravida , number of pregnancies , multiple pregnancy , pre-eclampsia , gestational hypertension , oral tocolytic therapy like beta adrenergic agonists[1]. Symptoms usually include one or more of the following: orthopnea (difficulty breathing while lying flat), dyspnea(shortness of breath on exertion), pitting edema in lower extremities (swelling), cough, frequent night-time urination, excessive weight gain during the last month of pregnancy (1-2+ kg/week; two to four or more pounds per week), palpitations(sensation of racing heart-rate, skipping beats, long pauses between beats, or fluttering), and chest pain.[5]The shortness of breath is often described by patients as the inability to take a deep or full breath or to get enough air into the lungs. Also, patients often describe the need to prop themselves up overnight by using two or more pillows in order to breathe better. These symptoms, swelling, and/or cough may be indications of pulmonary edema(fluid in the lungs) resulting from acute heart failure . Unfortunately, patients and clinicians sometimes dismiss early symptoms because they appear to be typical of normal pregnancy. Yet early detection and treatment are critically important to the patient with Peripartum cardiomyopathy . Delayin diagnosis and treatment are associated with increased morbidity and mortality.For these reasons, it is paramount that clinicians hold a high suspicion of Peripartum cardiomyopathy in anyperi- or postpartum patient where unusual or unexplained symptoms or presentations occur . Treatment for Peripartum cardiomyopathy is similar to treatment for congestive heart failure. Conventional heart failure treatment includes the use of diuretics, beta blockers (B-B), and angiotensin-converting enzyme inhibitors (ACE-I) only after delivery. Diuretics, preferably furosemide, help the body to get rid of excess water weight and also lower blood pressure. ACE-I and B-B improve blood circulation and contribute to the reversal of the immune system dysfunction . If ACE-I is not well tolerated by the patient, it can be replaced by angiotensin receptor blockers (ARB). Hydralazine with nitrates may replace ACE-I inbreastfeeding mothers or before delivery ; .If EF is less than 35%,anticoagulation is indicated, as there is a greater risk of developing left ventricular thrombi It is important that the patient receives regular follow-up care including frequent echocardiograms to monitor improvement or the lack.Patients who do not respond to initial treatment, defined as left ventricular EF remaining below 20% at two months or below 40% at three months with conventional treatment may merit further investigation, including cardiac magnetic resonanceimaging (MRI),cardiac catheterization, and endomyocardial biopsy for special staining and for viral polymerase chain reaction (PCR) analysis. Antiviral therapy, immunoabsorption, intravenous gamma globulin, or other immunomodulation therapy may then be considered accordingly , it is still recommended that both ACE-I and B-B be continued for at least one year after diagnosis.The most recent studies indicate that with newer conventional heart failure treatment consisting of diuretics,ACE inhibitorsand beta blockers, the survival rate is very high at 98% or better, and almost all patients improve with treatment. over 50% of patients experience complete recovery of heart function (EF 55% or greater) [6]. Once fully recovered, if there is no subsequent pregnancy, the possibility of relapse or recurrence of heart failure is minimal.Subsequent pregnancy should be avoided when left ventricular function has not recovered and the EF is lower than 55% , the risk for recurrence of heart failure in recovered Peripartum cardiomyopathy patients as a result of subsequent pregnancy is approximately 21% or better[7] The chance of relapse may be even smaller for those with normal contractile reserve as demonstrated by stress echocardiography[8]. Where relapse occurs, conventional treatment should be resumed, includinghydralazine withnitratesplus beta-blockers during pregnancy, or ACE-inhibitors plus beta-blockers following pregnancy.

CONCLUSION

Though peripartum cardiomyopathy is relatively a rare disease (0.1% of pregnancies) it can lead to devasting consequences with overall morbidity mortality rates as high as 5 to 32% . The diagnosis of peripartum cardiomyopathy is challenging since most women in last month of

normal pregnancy or soon after delivery experience dyspnoae , fatigue and pedal odema , [2] (as in our case) . Hence the treating physician should have high index of suspicion and consider it when managing dyspneic patients to expedite medical treatment for this potentially lethal condition . [4]

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