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CASE REPORT OF STAGE –IV PERIPARTUM CARDIOMYOPATHY WITH SEVERE PREECLAMPSIA WITH GOOD FETOMATERNAL OUTCOME

Gynecology

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery without any identifiable cause.The precise incidence in India is not known , except for which quotes as 1 per 1374 live births from a tertiary care hospital from South India .(2) Though PPCM resembles dilated cardiomyopathy clinically left ventricle may not be always dilated. (3,4,5).

Here we report a caseof 34 wks ANC with severe preclampsia with predelivery LVEF 25-30 % with systolic heart failure who had a good fetomaternal outcome .Patient presented with acute breathlessness with orthopnoea .Patient recieved intensive care management with ventilatory support and delivered by emergency LSCS. After postoperative hospital stay of 25 days patient went home with baby .

KEYWORDS

Peripartum cardiomyopathy , heart failure , echocardiography

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare disease , which can have devasting consequences and should be promptly identified and correctly treated.Though PPCM resembles dilated cardiomyopathy clinically but left ventricle is not always dilated . Ejection fraction is nearly always reduced below 45%. PPC M is considered an independent disease ,whose diagnosis relies on the exclusion of other cardiomyopathies and its relation to pregnancy[5]. For differentiating all these conditions monitoring postpartum course is vital. Oxidative stress and subsequent formation of kDa PRL ,endothelial damage, raised prolactin levels play important role in PPCM pathogenesis. PPCM patients deteriorates rapidly but with vigilant treatment recovery rate is good. However cardiac dysfunction reemerges frequently (30 %) in peri and postpartum phase[6] .Hence , PPCM patients should be advised against pregnancy .

CASE REPORT

19 years old primigravida 34 weeks, presented with complaints of sudden onset breathlessness and orthopnoea since last 1 day .Patient had bilateral pedal oedema , increased weight gain since last 1 month . On admission the patient was afebrile and had a blood pressure of 160/90 mm Hg, a pulse rate of 120 beats per minute, a respiratory rate of 30 breaths per minute, and an oxygen saturation of 90% while receiving oxygen through a 2-L nasal cannula. Crepitations were present all over the lungs on auscultation and her heart rate was regular, with an S3 gallop. Her extremities were oedematous with no calf tenderness. Urinalysis result was suggestive of +3 urine proteins. An electrocardiogram showed a normal sinus rhythm.echocardiogram was done suggestive of global left ventricular hypokinesia with LVEF 25- 30 %. Patient was immediately admitted and shifted to intensive care unit and was put on CPAP mode of ventilation as patient was unable to maintain saturation .Patient was started on inj furosemide 40-40-0 , injection digoxin , tablet lobetalol 200 mg TDS , injectable antibiotics. Patient was given 2 doses of intramuscular steroid injection for lung maturity .Gradually patient was shifted on tab furosemide 40 mg OD , tab Carvediol 3.125 grams , tab digoxin 0.25 mg (5 /7)OD , tab lobet 200 mg TDS .Ultrasonography done which was suggestive of bilateral moderate pleural effusion, Single live intrauterine gestation of 34 wks of EFW 1.9 Kg. Strict vitals monitoring was done. BP was not controlled on antihypertensives .Patients BP was 150 / 90 mm of hg , U / A - +4 .Repeat USG was showing severe oligohydramnios (AFI 3-4) with fetoplacental insufficiency , Hence decision was taken for emergencyLSCS .Patient was prophylactically intubated in ICU and shifted to operation theatre for emergency LSCS . Patient delivered male baby of 1.65 kg. Baby was immediately intubated and shifted to NICU . Intraoperatively one pint PCV was transfused.Postoperatively patient was shifted to CCU for monitoring , gradually weaned off ventilatory support . Patients symptoms improved dramatically in postpartum period .Patient was able to maintain saturation hence gradually weaned off CPAP .Patient and baby were discharged on day 25 of caeserian section .Patient followed up at 6 wks PNC where 2D ECHO showed rise in LVEF 45 - 50 %.

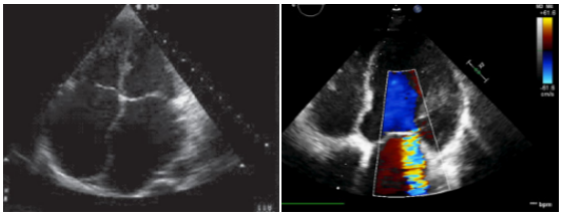


Figure1,2:Peripartum Cardiomyopathy (2D ECHO findings)

DISCUSSION

The definition of PPCM includes following criteria 1) Development of cardiac failure in the last month of pregnancy or within five months of delivery .2) Absence of an identifiable cause for the cardiac failure. 3) Absence of recognizable heart disease before the last month of pregnancy.4) Left ventricular (LV) dysfunction .Ejection fraction of less than 45% or reduced shortening fraction less than 30 % or left ventricular end diastolic dimention>2.7 cm / m square of body surface area [5]

Though PPCM resembles dilated cardiomyopathy clinically but left ventricle is not always dilated. Ejection fraction is nearly always reduced below 45 % .PPC M is considered an independent disease ,whose diagnosis relies on the exclusion of other cardiomyopathies ,its relation to pregnancy [10].Peripartum heart failure due to preeclampsia and peripartum cardiomyopathy resembles in clinical features but Echocardiography can differenciate two conditions ,in PPCM LVEF is always reduced but in peripartum heart failure due to preeclampsia LVEF is preserved .Like this PPCM should be differentiated from genetic forms and pathogen triggered pregnancy associated cardiomyopathies. For differenciating all these conditions monitoring postpartum course is vital .

Oxidative stress and subsequent formation of kDa PRL ,endothelial damage plays important role in PPCM pathogenesis. Peripartum cardiomyopathy often develops in young patients during pregnancy and is a disease which progresses quickly to cardiac dysfunction and failure as is evident in high mortality and transplantation rates .Patient presents with clinical features of acute heart failure such as pulmonary oedema , acute breathlessness , oedema or may present with dysnoea , fatique similar to normal pregnancy hence diagnosis often missed at earlier stages . If diagnosed earlier and treated with b – blockers , ACE inhibitors , bromocriptine[1] there is favourable outcome .This is in contrary to what is often observed in DCM which is characterised by late onset and slow disease progression that might be stabilised but unlikely to reversed to normal or near normal upon tratment.Various investigations and tests aid in the diagnosis of PPCM. ECG abnormalities are frequently but not always present in PPCM patients.

There are no specific ECG patterns for PPCM patients but intra ventricular conductance abnormalities , such as left bundle branch block are common .Chest X –ray will show the signs of decompensated heart failure with pulmonary oedema and pleural effusion . But transthorasic echocardiography remaims mainstay for diagnosis which demonstrates decreased LVEF .NT-proBNP , 16- kDa Prolactin , Interferon –Y , ADMA ,sFlt-1 are increased in blood but are not specific for PPCM .Currently PPCM is treated according to the ESC guidelines for heart failure in pregnancy [6,7,8].

If PPCM patients get pregnant again it should be noted that pregnancy state can be tolerated well , especially if the subsequent pregnancy is entered with fully recovered cardiac function.However cardiac dysfunction reemerges frequently (30 %) in peri and postpartum phase. So , PPCM should be advised not to get pregnant again . IUCDs are contraceptives of choice for PPCM patients [10]

CONCLUSION

PPCM ia a rare but potential life threatening cardiac failure of unknown etiology , encountered late in pregnancy or in the postpartum period. Echocardiography is mainstay for the diagnosis of PPCM. Standard protocols are for symptomatic treatment of PPCM. Bromocroptine and cabergoline hold promise for the future, severe cases might require transplantation. Subsequent pregnancies are associated with a very high mortality more so in those whose LV functions do not improve even after 6 months pueperium .In PPCM patients with regain of normal cardiac function on ECHO evaluation subsequent pregnancy may be considered under close multi disciplinary supervision . Large clinical registries on peripartum heart failure together with experimental research are needed for further understanding of PPCM . [9]

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