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POSTPARTUM ANASARCA – RARE CASE REPORT

Cardiology

KEYWORDS:

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INTRODUCTION:

Pregnancy is a critical period in which significant hemodynamic and immunologic changes that in certain cases can become problematic. PPCM is a disease of unknown cause in which left ventricular (LV) dysfunction occurs during the last trimester of pregnancy or the early puerperium, reported in 1:1300 to 1:4000 of live births (1). Demakis et al2, defined peripartum cardiomyopathy as idiopathic heart failure occurring in the absence of any determinable heart disease in the last month of pregnancy or during the first 5 months postpartum.

Current Diagnostic Criteria for Peripartum Cardiomyopathy3

1. Development of heart failure in last month of pregnancy or 5 months postpartum
2. Absence of preexisting heart disease
3. Indeterminant cause
4. Echocardiographic findings (a, together with b or c; or all of these)

a. Left ventricular end-diastolic dimension >2.7 cm/m2
b. M-mode fractional shortening < 30%
c. Left ventricular ejection fraction <0.45

PPCM is a diagnosis of exclusion.

We present to you a case a post-partum cardiomyopathy managed at our center.

CASE REPORT:

21 years old female primipara had asymptomatic 3 months of post-partum, following which she came with complaints of cough, dyspnea and anasarca. There was no history of fever, chest pain, hemoptysis, palpitation, orthopnea, PND. No history of oliguria, hematuria or frothy urine. Patient underwent normal vaginal delivery at term, and her ante partum and intra partum period was uneventful. No history of puerperal complications. She was born out of non-consanguineous marriage. **T**here was no history of similar complaints in the family. There is no history of diabetes, hypertension, asthma, cardiac disease.

On Physical examination, patient was ill looking with facial puffiness, pallor and pitting pedal oedema. Patient was in hypotension (90/70 mmHg), with sinus tachycardia at 120 bpm, tachypnea and raised JVP. Systemic examination revealed a grade III systolic murmur in mitral area and bilateral infra axillary and infra scapular crepitation were heard on lung auscultation. There were no signs of liver disease and neurological examination was normal.

The chest x-ray on admission showed with bilateral pleural effusion and cardiomegaly. ECG revealed diffuse T wave inversion. Total counts were normal, Hb 9.8 g/dl, Urine analysis showed presence of albumin 2+ and urine culture and blood culture were sterile. RFT and LFT were normal. Thyroid profile was normal.Cardiac bio markers, ASLO, ANA, HIV, HBsAg, HCV were negative. Serological tests for the viral agents responsible for myocarditis were negative.

USG Abdomen showed congestive hepatomegaly and ascites. Ascites and pleural fluid analysis was suggestive of transudate effusion. Echocardiography showed global hypokinesia of left ventricle, all chambers dilated, severe MR, severe TR, severe LV systolic dysfunction, large LV apical clot and LV ejection fraction 30%.

In view of all above finding, she was diagnosed to have peripartum cardiomyopathy with systolic heart failure. She was managed symptomatically and patient improved clinically with treatment and was discharged after 10 days.

DISCUSSION

Peripartum cardiomyopathy (PPCM) is a type of dilated cardiomyopathy of unknown origin. It occurs in previously healthy women in the final month of pregnancy and up to 5 months after delivery.1 Although the incidence is low < 0.1% of pregnancies—morbidity and mortality rates are high, ranging from 5% to 32%.[4]

Risk factors for peripartum cardiomyopathy include advanced maternal age, twin pregnancy, smoking, pregnancy-related hypertension and preeclampsia, multiparity, African descent, and long-term tocolysis1,2. Preeclampsia is a risk factor for cardiovascular disorders later in life. Management of PPCM is similar to treatment for other forms of heart failure.[4]

Nitrates are mostly used for preload reduction, most of which are safe during pregnancy and breastfeeding13. Loop diuretics are also used for preload reduction, although caution is warranted in antepartum women because rapid changes in intravascular volume, can alter the blood supply to uterus and fetus also13. Restriction of dietary sodium is also helpful in preload reduction. Bed rest is not recommended, in view of increased chances of thromboembolism13. The current recommendation is light exercise[6.]

Angiotensin-converting enzyme inhibitors are contraindicated during pregnancy, but these medications are the mainstay of treatment of PPCM after delivery for afterload reduction.4 Safe alternatives during pregnancy include hydralazine and nitrates6. - Adrenergic antagonists, have been approved for use in PCCM and can improve survival.7

Warfarin and heparin or low-molecular heparin should be given to women with PPCM whose ejection fraction is 35% or less.11 Anticoagulation therapy should be continued until left ventricular function is normal according to echocardiographic findings.[12]

Prognosis of PPCM depends amount recovery of ventricular activity13. Failure of heart size to return to normal is associated with increased mortality and morbidity.

CONCLUSION

PPCM affects previously healthy women in the final month of pregnancy and up to 5 months after delivery. For few women, the clinical and echocardiographic status improves rapidly and sometimes returns to normal. In other women, the clinical condition rapidly worsens, no improvement occurs with medical therapy, and chronic heart failure develops. No single explanation of the

pathogenesis of PPCM is relevant for all women; the disease has a multifactorial origin.

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