VOLUME - 9, ISSUE - 7, JULY - 2020 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Cardiology

WILL FETUS BITE THE HEART – A RARE CASE OF CARDIOMYOPATHY

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ABSTRACT Septic shock in obstetric patients is a rare clinical event. The estimated incidence is one in 8,338 deliveries. Two thirds of cases occur in the ante partum period and one third in postpartum. 22 year unmarried female with no previous co morbidities was admitted with complaints of Breathlessness, Bilateral pedal edema, Abdominal distension and fever for 1 week duration. Repeat echo done on day – 10 of admission showed improved LV function, Adequate LV systolic function. Sepsis-induced cardiomyopathy is a reversible myocardial dysfunction that typically resolves in 7–10 days. Sepsis-induced cardiomyopathy has three characteristics: left ventricular dilatation, depressed ejection fraction, and recovery in 7–10 days.

KEYWORDS : SEPSIS, SEPTIC CARDIOMYOPATHY

INTRODUCTION

Septic shock in obstetric patients is a rare clinical event. The estimated incidence is one in 8,338 deliveries. Two thirds of cases occur in the ante partum period and one third in postpartum. Sepsis-induced cardiomyopathy (SICM) is an entity of cardiomyopathy which is reversible in 1-2 weeks after recovery from sepsis or septic shock.

CASE REPORT

22 year unmarried female with no previous co morbidities was admitted with complaints of Breathlessness, Bilateral pedal edema, Abdominal distension and fever for 1 week duration. Breathlessness was present for past 1 week. Initially NYHA grade II dyspnea, progressed to NYHA grade 4 dyspnea for 2 days before hospitalization. Orthopnea and paroxysmal nocturnal dyspnea was present. Fever was present for past 2 days, high grade, continous fever, associated with chills and rigor. No history of cough/ expectoration/ hemoptysis. No history of lower abdominal pain, vomiting, loose stools, bleeding p/v, draining p/v or reduced urine output. She was a non-smoker, non-alcoholic, unmarried with LMP not known.

On Examination, she was febrile, dyspnoeic, tachypnoeic, pallor, Icteric, B/L pedal edema grade 4, CVS - S1S2+,tachycardia, RS - NVBS+, B/L basal crepitations, P/A - uterus 26 weeks size, not acting, not tense and tender.

Investigations revealed WBC-36,300 (P74,L 20),Hb- 9 gms/dl, Platelet-3,40,000. Renal parameters were elevated with B. urea-78,S.creatinine-1.8 mg/dl. Bilirubin- total-2. 8, SGOT-1253, SGPT-1256, SAP-189. Serum electrolytes Na+-137, k+-4.5, Serum LDH -1645 IU. Serum CRP – Positive. Preliminary Urine culture , blood culture, high vaginal swab – no growth. Repeat Blood culture – E.coli .D-dimer - > 50000, FDP – Positive, FIBRINOGEN – 238 ng/ml (N 200-400), PT - >60 sec, aPTT- 110 sec PS - Normocytic normochromic anaemia, Neutrophilia and thrombocytopenia. (platelet count was decreasing in trend). TROPONIN T(card test) – POSITIVE. USG Abdomen and pelvis shows -Intrauterine fetal demise of 27 weeks gestation, GB wall edema, B/L Mild HUN, no free fluid in abdomen and pelvis, and Right mild pleural effusion noted. PULMONARY CT ANGIOGRAM showed Bilateral lung parenchyma appears normal. 11*13 mm thrombus in right ventricle with no evidence of pulmonary embolism.

Echo done showed Severe global hypokinesia of left ventricle, Severe biventricular dysfunction, LVEF-24 %. All four chambers were dilated. Large RV thrombus 14*12mm, Mild TR, RVSP 41 mmHg, Moderate Pulmonary artery hypertension. The differential diagnosis considered were severe anaemia with ccf , dilated cardiomyopathy ? Idiopathic , pulmonary/ amniotic fluid embolism, peripartum cardiomyopathy , takotsubo cardiomyopathy , sepsis induced cardiomyopathy.

Meanwhile patient spontaneously expelled dead male fetus of weight 850 grams. Patient was treated with fluid resuscitation, inotropic support – Inj.Noradrenaline and Inj.Dobutamine, Inj.Meropenem , Inj.Metronidazole. Periodic monitoring of coagulation profile and PRBC and FFP substitution. Serum Albumin was persistently low and Inj.Albumin 25% was given for 10 days. T.Carvidilol, Inj.Frusemide, T.Spironolactone. The inotropic support was stopped after 5 days and Beta blockers, cardiac failure drugs continued along with Inj.Albumin transfusion. Repeat echo done on day – 10 of admission showed improved LV function, Adequate LV systolic function, grade 1 LV diastolic dysfunction, no RV dysfunction, thormbus not visualised, LVEF-51 %.

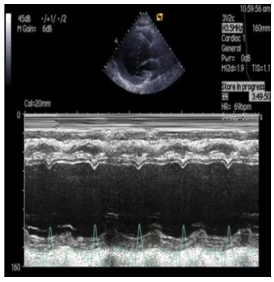

FIGURE -1 : DAY 1 ADMISSION, LVIDd- 6.3cm, LVIDs – 5.3 cm, EF – 24%

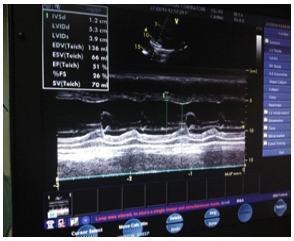

FIGURE – 2 : DAY 10 ADMISSION, LVIDd- 5.3cm, LVIDs – 3.9 cm, EF – 51%

DISCUSSION

Sepsis-induced cardiomyopathy is a reversible myocardial dysfunction that typically resolves in 7–10 days. Sepsis-induced cardiomyopathy has three characteristics: left ventricular dilatation, depressed ejection fraction, and recovery in 7–10 days. In 1984, Parker et al. reported decreased ejection fraction and increased end-diastolic volume in septic shock survivors. In this case, myocardial dysfunction from sepsis leads to systolic heart failure. Endotoxins cause depressed cardiac contractility, which is mediated by enhanced NO production. TNF and IL-1 also contribute to NO overproduction.

The differential diagnosis of heart failure in a parturient includes peripartum cardiomyopathy, myocarditis, and other cardiomyopathies such as viral, familial, dilated, hypertrophic, or drug related. In this case, our patient did not meet the consensus definition for peripartum cardiomyopathy which requires that there be no identifiable cause for cardiac failure other than pregnancy. Myocardial dysfunction was profound with a decrease in ejection fraction to 28%. The patient had symptoms of shortness of breath and lower extremity edema. It also seemed unlikely that our patient had pre existing cardiac disease which was exacerbated by severe sepsis because she reported previous good health before and during her pregnancy. We did not believe myocarditis was likely because of the normal troponin level and electrocardiogram. An endomyocardial biopsy could have been performed, but the rate of a positive biopsy is low even in those with high suspicion for myocarditis and the procedure is invasive. The treatment of septic myocardial dysfunction involves aggressive intravenous pre load resuscitation with either crystalloid or colloid, the use of ionotropic and vasopressor drugs for treating arterial hypotension. Septic myocardial dysfunction in our patient manifested as symptoms of congestive heart failure and dyspnea on exertion. For this reason, she was treated with both a beta blocker and a diuretic because these are first line drug therapies for systolic heart failure.

CONCLUSION

Septic shock represents one of the most challenging and potentially devastating clinical problems for physicians who care for women with a complicated pregnancy. This condition has been associated with a mortality rate as high as 80% in non-pregnant patients. Unfortunately, the clinical presentation and therapeutic management of obstetric septic shock has not been well characterized. Because of its infrequency, it has been difficult to establish specific guidelines for the aggressive hemodynamic management of this disorder.