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PERIPARTUM CARDIOMYOPATHY WITH ACUTE RENAL FAILURE - a case report

SENTHIL KUMAR
Department of Anaesthesiology,STANLEY MEDICAL COLLEGE AND HOSPITAL

Abstract : Peripartum cardiomyopathy is a rare and potentially fatal disease which presents with symptoms of heart failure , primarily due to left ventricular systolic dysfunction in the last month of pregnancy upto 6 months after delivery.30-50 of the patients recover without complications , with normal left ventricular systolic function. Parturients with peripartum cardiomyopathy require special anaesthetic care during labour , delivery and postpartum period. Medical management is similar to dilated cardiomyopathy with management tailored to choose safe drugs in pregnancy and lactation.

Keyword : Peripartum cardiomyopathy, left ventricular systolic dysfunction , pregnancy and lactation 28years old primi with 9 months amenorrhoea , booked and immunised , LMP= 06/02/11, EDD=13/11/11 referred from a primary health centre with complaints of pain abdomen with difficulty in breathing for the past 5 hours. No significant previous history of heart disease. On examination the patient was conscious oriented afebrile , dyspnoeic at rest, pallor and bilateral pedal edema present. Height 145 cm, Weight 56kgs, her vitals Heart rate 113/ min , Blood Pressure 104/70 mm hg ,Respiratory rate 22/min, CVS=S1S2 + , RS=NVBS+, Per abdomen uterus term acting , Per vaginal examination cervix 3cm dilated, meconium stained amniotic fluid , Foetal Heart Rate was153/ min. Investigations showed Haemoglobin 10.2 grams, Total count 8400 cells/cumm , RBS=115mgs% , Urea=32mgs% , Creatinine=0.8mgs% , Urine-Albumin/sugar negative . In view of meconium stained amniotic fluid and foetal distress, Emergency Lower Segment Caesarean Section was done under subarachnoid block. Intraoperative period was uneventful , blood loss was 1300ml , 1500 ml of crystalloid was infused intraoperatively , urine output was 150ml. In the second postoperative day patient complained of fever , loose stools and breathlessness. On examination patient was conscious, febrile, dyspnoeic , pallor, bilateral pedal oedema was present. Her vitals showed HR=108/min , RR=25/min , BP=92/60mmhg , temperature=99.60f,CVS=S1S2+, RS= bilateral basal crepitations , P/A=soft. Urine out-put=200ml/24hrs.

Investigations :

Haemoglobin = 8.4gms , Total count=14000 cell/cumm, Blood sugar random=142mgs , Urea = 64mgs%, creatinine = 2.1mgs% , Na+ = 136meq/l , K+=4.2meq/l.Patient was treated with back rest , oxygen via face mask , intravenous fluids, antibiotics and antifailure measures. Urine output was 60ml in 5hours. As the urine output did not improve , nephrologist opinion was sought and he suggested hemodialysis. Patient was shifted to our ICU for further management.

On examination in ICU :

Patient was found to be progressively dyspnoeic , pallor , febrile , with bilateral pedal oedema

HR=102/minute , RR= 24/minute , BP=86/50 mmhg , SPO2= 98% 5 l/min of O2 , Temperature = 99.7

0f. Right Internal jugular vein was cannulated with 7F triple lumen catheter and central venous

pressure monitoring showed 21 cmH2O.Haemodialysis was started . 2 units of packed RBC was

transfused.Continuous monitoring of HR , NIBP , ECG , SPO2 , CVP , TEMPERATURE and URINE

OUTPUT was done.

Treatment :

Patient was started on Inj Furosemide infusion at the rate of 0.5 mg/hr

Inj Dopamine = 5mcg/kg/minute

Inj Meropenam 500mg IV bd

Inj Linezolid 600mg IV bd

Inj Dobutamine = 3-5 mcg/kg/min

On the 4th postoperative day patient developed progressive dyspnoea, tachypnoea, restless HR=122/min, BP=90/56mmhg, RR=36/min,SPO2=88% with 5 l/min O2, CVP=18 cmH2O CVS= S1S2,S3,gallop rhythm , RS= bilateral basal crepitations

ABG was done and it showed

P^H	PO_2	PCO_2	HCO_3	Na^+	K^+	Hct
7.29	72	27	12.5	136	3.8	26

ECG showed sinus tachycardia , poor progression of R waves

Chest X ray PA view revealed Cardiomegaly

As the patient became progressively dyspnoeic ,patient was intubated and mechanically ventilated

Ventilator settings= Assist control pressure control mode

P_{insp}	V_{te}	FiO_2	RR	PEEP
12	423	60	14	8

ECHOCARDIOGRAPHY was done and it showed
- Left ventricular end diastolic diameter = 54mm
(Normal ; men=42-59mm ; women=39-53mm)
- Left ventricular end systolic diameter = 42mm
(Normal = < 40mm)
- Ejection fraction = 34% (Normal = 55%)
- Global left ventricular dilatation
- No Regional wall motion abnormalities
- Mild Mitral regurgitation
- Mild pericardial effusion present

Cardiologist diagnosed the condition as **PERIPARTUM CARDIOMYOPATHY** and advised **Tablet DIGOXIN= 0.25mg ½- 0-0 , Tablet HYDRALAZINE=25mg ½-0- ½** Patient was electively ventilated for 4 days and weaned from ventilator on 9th POD.Patient improved haemodynamically and dyspnoea settled .After 16 sittings of HEMODIALYSIS , urine output improved to = 450 ml / day. Renal parameters were Urea = 42mgs% , creatinine = 1.5 mgs%.

Patient was discharged and advised to follow up monthly at Nephrology unit.

CONCLUSION:

It is a rare presentation of PERIPARTUM CARDIOMYOPATHY with ACUTE RENAL FAILURE recovered.

PERIPARTUM CARDIOMYOPATHY

INTRODUCTION

Peripartum cardiomyopathy is a rare and potentially fatal disease which presents with symptoms of heart failure . A relationship between pregnancy and dilated cardiomyopathy was first noted in 1870 when Virchow and Porak first reported autopsy evidence of myocardial degeneration in patients who died in puerperium . Primarily due to left ventricular systolic dysfunction presenting in last part of pregnancy (32-38 weeks) and upto 5-6 months after delivery. It usual ly occurs in the postpartum period 45% in first week and 75% within first month. Clinically very similar to dilated cardiomyopathy , except for its unique relationship with pregnancy and full recovery with normalisation of left ventricular ejection fraction in almost 50% of patients. But it can still result in chronic disability and ultimately death in relatively young women in reproductive years. PPCM needs multidisciplinary approach and management.

INCIDENCE 4 of 1 in 3000 – 15,000 pregnancies

ETIOLOGY 5

It is still not clear exactly how it occurs. Many theories have been documented

1) Inflammatory pathology

a) Autoimmune (antibodies against myocardium)

b) Viral = Cardiotropic virus (coxsackie virus,echovirus)

2) Proinflamatory cytokines = TNF , IL-1 , IL-6

Recent evidence

-**16 kDa prolactin** derivative produced by proteolytic cleavage of prolactin secondary to unbalanced oxidative stress present during late pregnancy and early puerperium. This derivative has been cardiotoxic , antiangiogenic , proapoptotic and proinflammatory , which can potentially impair metabolism and contractility of cardiomyocytes.

Others causes are
- Impaired cardiac microcirculation
- Programmed cell death (Apoptosis)
- Deficiency of micronutrients –Selenium

Microchimerism 5 = Foetal cells present in the maternal system that elicit an inflammatory response.

EPIDEMIOLOGY
- 50% > 30 years , 30% occur in primi

RISK FACTORS 5
- Multiparity
- Advanced maternal age
- Gestational hypertention
- Preeclampsia

DIAGNOSTIC CRITERIA 1
- Onset of heart failure in last month of pregnancy to 5-6 months post partum
- Without any other demonstrable cause of heart failure
- Absence of any heart disease before pregnancy
- Echocardiography criteria include

Ejection Fraction <45%
Fractional shortening < 30% or both
End diastolic dimension >2.7 cm/m2 body surface area.

SYMPTOMS 2
- Dyspnoea on exertion
- Orthopnoea
- Paroxysmal Nocturnal dyspnoea
- Cough
- Chest pain
- Palpitation
- Pedal oedema
- Fatigue

SIGNS 2
- Raised JVP
- HR > 100
- S 3 gallop
- Basal crepitations
- Mitral regurgitation murmur

The severity of symptoms in patients with PPCM can be classified by the New York Heart Association system as follows:

Class I - Disease with no symptoms
Class II - Mild symptoms only with extreme exertion
Class III - Symptoms with minimal exertion
Class IV - Symptoms at rest

INVESTIGATIONS

1) Complete haemogram to rule out anaemia
2) Renal function test
3) Serum electrolytes
4) Thyroid function test
5) ECG may show sinus tachycardia/ST/T wave changes
6) Chest X ray may reveal cardiomegaly/pulmonary oedema/pleural effusion
7) ECHO = dilated LV / EF / EDV / valvular abnormalities
8) M R I heart
9) Cardiac markers = TROPONIN T
CREATINE KINASE-MB

MANAGEMENT
- Needs multidisciplinary team approach

MANAGEMENT GOALS

2Cardioloist, obstetrician,intensivist,anaesthesiologist,paediatrician

- Increase myocardial contractility
- Reduce cardiac preload and afterload
- Prevent complications and mortality
- Fluid restriction upto 2 litres/day
- Salt restriction upto 2-4 gm/day

During pregnancy 5

1) DIGOXIN = 0.25 mg
2) DIURETICS : Furosemide = 20-40 mg
3) NITRATES
4) HYDRALAZINE = 25-100 mg

Postpartum

1) DIGOXIN = 0.25 mg
2) DIURETICS : Furosemide = 20-40 mg , Spiranolactone = 25mg/ day
3) HYDRALAZINE = 25-100 mg
4) ACE INHIBITORS = Carvedilol 25mg bd , Metoprolol 100mg od
5) Early ambulation

VASOPRESSORS

- Inj DOPAMINE = 3-5 mcg/kg/min
- Inj DOBUTAMINE = 5 mcg/kg/min
- Inj MILRINONE = 0.3-0.75 mcg/kg/min

ANTICOAGULATION 1

- inj Heparin 5000U SC
- inj LMWH 0.3ml SC

RECENT ADVANCE 5

- BROMOCRIPTINE = on randomised control trail in US
- PENTOXIFYLLINE = inhibits TNF
- INTRAVENOUS IMMUNOGLOBULIN

SPECIFIC MANAGEMENT DURING LABOUR

- Labour should be induced. Vaginal delivery is preferred over caesarean section. Labour pain during second stage of labour cause maximum hemodynamic and oxidative cardiac stress which can be minimised by epidural analgesia and forceps delivery. Slow induction of epidural analgesia, Sympathectomy and decrease in after load improves cardiac performance. Intrathecal opioids are also useful.
- If a cesarean delivery is required, a continuous epidural or spinal anaesthesia is usually the best anesthetic option. The patient's hemodynamic status is carefully followed and fluid management is guided by data from the invasive monitors while the anesthesia level is slowly raised. A single-shot spinal technique is not recommended because the rapid hemodynamic changes associated with this technique may not be well tolerated in these fragile patients. General anaesthesia is sometimes required when caesarean section is required because of nonreassuring foetal status or acute maternal decompensation. Anaesthetic drugs with myocardial depressant effects should be avoided. Induction and maintenance with a high-dose opioid technique is often preferred.
- Post operatively patient should be monitored in intensive care unit
- It is imperative to give contraceptive advice, as patients should not become pregnant again for at least a year or until LV function has returned to normal.

MONITORING AND FOLLOW UP 1

Patients are monitored on a regular basis to ascertain the response to treatment , with titration of drugs according to AHA guidelines. Frequent echocardiographic assessment of left ventricular function. Patients refractory to medical treatment and showing progressive deterioration of left ventricular function may need left ventricular assist device and early heart transplantation.

PROGNOSIS 5

Good prognostic factors

- Troponin T < 0.04 ng/dl 2 weeks after onset
- QRS duration < 120 ms
- EF > 30% and LVESD = < 5.5 cm

30-50 % of the patients recover without complication, with their baseline left ventricular systolic function at rest returning to normal. If left ventricular function does not return to normal within 6 months of postpartum, it indicates irreversible cardiomyopathy and has worse prognosis

CONCLUSION

Peripartum cardiomyopathy is a rare lethal disease. Diagnosis is confined to a narrow period and it requires echocardiographic evidence of left ventricular systolic dysfunction. Symptomatic patients should receive standard therapy for heart failure , managed by multidisciplinary team . If subsequent pregnancies occur , they should be managed in a peripheral tertiary care centre.

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