Peripartum Cardiomyopathy a Rare Case

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Abstract

Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery, though it was first described in the 1800s, yet its etiology is still unclear. The definition of PPCM includes four 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, and 4) left ventricular (LV) dysfunction (ejection fraction of less than 45% or reduced shortening fraction). Peripartum cardiomyopathy is a relatively rare disease, which can have devastating consequences and should be promptly identified and correctly treated. We report of a rare case of Peripartum cardiomyopathy in primigravida with diabetes and hypertension.

Keywords: Haemoglobin; Peripartum Cardiomyopathy; Pregnancy

Case Report

A 29-year-old primigravida, known overt diabetic and hypertensive was admitted to hospital for elective LSCS at 38 weeks of gestation. Patient was obese. She was diagnosed with diabetes mellitus type 2, 8 months prior to conception. At the time of diagnosis, her HbA1c was 10.5. Her FBS was 229 and PPBS was 354. She was not taking any medications for diabetes. Her haemoglobin, thyroid was normal and remained normal throughout pregnancy. She was tested negative for HIV, HBsAg and VDRL. Her ophthalmological examinations were also normal. She had one episode of fever and URTI during 1st trimester. At the time of conception, her FBS was 111 and PPBS was 192. HbA1c was 7.6. She was treated with novarapid insulin subcutaneously 4 unit before breakfast and 4 unit before lunch. During 2nd trimester, her insulin requirements rose to 8 unit novarapid insulin before breakfast and before lunch and 10 units with dinner. During 3rd trimester, she needed 10-10-12 insulin with meals till the time of delivery[1-4].

- She had undergone NT scan at 13 weeks GA which showed NT 1.7 mm. (normal).
- Anomaly scan (18 weeks)- no anomaly.
- Quadruple marker test (18 weeks)- revealed low risk for trisomy 21, trisomy 18 and open neural tube defects
• Fetal echo at 27 weeks GA which was normal.
• Obstetric Doppler at 36 weeks’ gestational age which indicated
that patient had polyhydramnios (AFL 25.75cm), EFW 3008
gm. Diastolic noth in both uterine arteries. Fetoplacental
circulation was normal.

Patient was treated with tablet ecosprin 75mg till 36 weeks
GA. Iron and calcium supplements and insulin throughout
pregnancy. She was given injection dexamethasone 4 doses at 28
weeks of gestation. She developed gestational hypertension in 37th
week of pregnancy for which she was treated with tablet labetalol
50 mg BD. Her pre op investigations were normal except for she
had UTI (15-20 pus cells in urine routine and microscopy)

On the day of LSCS, she was kept on injection insulin human
actrapid 10 unit with DNS at 16 drops per minute (neutralising
drip). RBS charting was done every 2 hours. Intraoperative period
was uneventful. Patient delivered female child, 3.38 kg, apgar
score 8 (1 min), 9 (5 min).

As soon as patient was shifted to complete oral diet on
second day post op period, she was kept on insulin 6U-6U-6U
with meals. Insulin dose was titrated up to 10 U- 8U- 14 U with
meals till discharge according to 7-point sugar profile. Tablet lobet
was continued in dose of 50 mg BD on which her BP was well
maintained in range of 130/90 to 150/90. On day 2 post LSCS,
patient developed fever. She had complaints of difficulty in
deglutition, backache, headache, cold. Treatment for URTI was
started. To which patient responded well. On 3rd day post op,
patient started complaining of breathlessness on rest, cough with
expectoration. Her pedal edema was increased. She developed
facial puffiness as well. Her BP was 150/110 mmHg, her respiratory
rate was 38/minute and she had basal crepitations on auscultation.
Her saturation was 88% on room air. Immediately, physician and
chest TB expert opinion was taken. Injection Lasix 20 mg stat
dose was given. Since, peripartum cardiomyopathy was suspected,
patient was shifted to ICU for observation and treatment.

She was treated by oxygen inhalation at 1L/min. patient
maintained saturation 95-96% with oxygen. Injection Lasix was
started in dose of 20 mg TDS. Her ABG showed respiratory
acidosis compensated by metabolic alkalosis. Her antibiotic was
changed from oral tablet Taxim O 200 mg BD to injection piptaz 4.5
gm 8 hourly. Piptaz was continued for 10 doses. Initially,
patient was given injection monofec 1gm 12 hourly for 3 doses,
administration of tablet 8 hourly for 3 doses, injection gentamycin 80
mg iv 12 hourly 2 doses on 1st day post op. Dose of tablet lobet was up
titrated to 100 mg BD. Her BP was still uncontrolled. Hence,
she was shifted on T. carvidilol 3.125 mg BD. Tablet stamlo 5 mg OD
was added to treatment for further control of BP. Tablet lobet
was stopped. Her echo report was s/o global LV hypokinesia, LVEF
50%, mild LV systolic dysfunction, intact septa. Strict input/output
monitoring was done to see that output should exceed input. Patient
improved on day 3rd of ICU treatment. With treatment, patient’s
breathlessness was gone, pedal edema subsided, facial puffiness
was also gone. Eventually she was discharged on injection insulin
10U-8U-14U with meals, tab cardivas 6.25 mg BD, tab stamlo 5
mg OD, sykesol 10 ml BD and other supportive treatment with
fair control of BP, diabetes and CCF. Baby was shifted to NICU for
hyperbilirubinemia. Baby received phototherapy. Baby was also
discharged with mother after vaccination.

Discussion

Peripartum cardiomyopathy (PPCM) is a rare, but life-
threatening disease, which affects women in the last month of
pregnancy or in the first 5 months after delivery. PPCM is a form of
Dilated Cardiomyopathy with left ventricular systolic dysfunction
that results in signs and symptoms of heart failure. Symptoms
usually occur in the last trimester and diagnosis is usually made
in the peripartum period. Peripartum cardiomyopathy usually
presents with symptoms of worsening cardiac failure. These include
dyspnoea on exertion, fatigue, ankle oedema, embolic phenomena,
atypical chest pains and haemoptysis. Examination may reveal
evidence of a raised CVP, tachycardia, cardiomegaly with a
gallop rhythm (S3), mitral regurgitation, pulmonary crackles and
peripheral oedema. Symptoms like decreased exercise capacity,
tiredness, dyspnoea, orthopnoea and palpitations may occur even
in normal pregnancy and can be mistaken for a diseased state.
Kotekar et al reported a case of peripartum cardiomyopathy posted
for caesarean section [5]. Chan and NganKee presented a case of
PPCM at 18 weeks’ gestation. She was managed medically with
systemic anticoagulation, until 31 weeks, when fetal distress and
maternal liver dysfunction forced an emergency caesarian section
[6]. Shrestha B.R. et al reported a case of PPCM with ejection
fraction of 18% brought for emergency caesarean section. It had
a successful outcome using epidural with Lignocaine 2% and
Adrenaline [7].

Conclusion

Peripartum cardiomyopathy (PPCM) is a relatively rare
disease, which can have devastating consequences and should
be promptly identified and correctly treated. Early diagnosis is
important and therefore women who develop symptoms of heart
failure during pregnancy or shortly after should be investigated
for this condition. Effective treatment reduces mortality rates and
increases the chance of complete recovery of ventricular systolic
function.

References

42-45.
2. Demakis JG, Rahimtoola SH, Sutton GC, Meadows WR, Szanto PB,
et al. (1971) Natural course of peripartum cardiomyopathy. Circulation
44: 1053-1061.
Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute
and Office of Rare Diseases (National Institutes of Health) workshop
recommendations and review JAMA 283: 1183-1188.
thy: An intriguing challenge. Case report with literature review. Cur-
rCardiol Rev5: 268-272.
peripartum cardiomyopathy posted for caesarean section. Indian J An-
aesth 51: 60-64.
7. Shrestha BR and Thapa C (2006) Peripartum cardiomyopathy under-
going caesarean section under epidural anaesthesia. Kathmandu Uni-