

CASE REPORT

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PERIPARTUM DILATED CARDIOMYOPATHY; ANAESTHETIC MANAGEMENT

DR. SAFDAR ALI KHAN, FCPS
Classified anaesthetist
Combined Military Hospital
Gujranwala, Cantt.

DR. SHAHAB NAQVI, FCPS
Head of the department of anaesthesia and
intensive care, Armed forces institute of
cardiology, Rawalpindi.

DR. MUHAMMAD BUKHSH , FCPS
Classified anaesthetist Armed forces institute of
cardiology, Rawalpindi.

ABSTRACT... sak1958@hotmail.com. Peripartum cardiomyopathy is a relatively rare but life threatening disease. The etiology and pathogenesis of peripartum cardiomyopathy remains largely theoretical and is generally centered upon viral and autoimmune mechanism. This case report describe the anaesthetic management of a patient suffering from dilated peripartum cardiomyopathy, successfully managed with epidural anaesthesia.

Keywords: Peripartum Cardiomyopathy, Epidural Anaesthesia, Caesarian Section, Peripartum Dilated Cardiomyopathy-Anaesthetic Management.

INTRODUCTION

Peripartum cardiomyopathy is a relatively rare but life threatening disease¹. A wide variation in incidence rate have been reported although the currently accepted incidence is approximately 1 per 3000 to 1 per 4000 per live birth².

A recent analysis of maternal mortality found cardiomyopathy to be the leading cause of maternal death in North Carolina, USA³. Based on a recent report, peripartum cardiomyopathy is defined by the presence of four criteria. These include:-

1. Development of cardiac failure in the last

months of pregnancy or within five months of delivery.

2. Absence of an identifiable cause for cardiac failure
3. Absence of recognizable heart disease prior to the last months of pregnancy.
4. Left ventricular systolic dysfunction demonstrated by echocardiographic criteria such as depressed ejection fraction.

The etiology of peripartum cardiomyopathy remain poorly understood. Despite much investigation

proposed causes include myocarditis, abnormal immune response to pregnancy, and maladaptive response to the hemodynamic stresses of pregnancy. There is more evidence to support myocarditis or an autoimmune process as the cause of the disease than for other proposed etiologies⁴.

CASE HISTORY

A 25 years old woman, G₃ P₂, transferred from a peripheral hospital, where she presented with 34 weeks of gestation, pregnancy induced hypertension and symptoms of cardiac failure. Her previous pregnancies had been uncomplicated and each time she delivered at home. The first half of the present pregnancy was also uncomplicated but in the mid trimester she was diagnosed as having pregnancy induced hypertension. She was advised treatment but non compliant in regular medication. At the beginning of the 32nd week of pregnancy, the patient developed shortness of breath on mild exertion, orthopnoea and cough with mucoid sputum. Her symptom rapidly got worse and within few days she was breathless even at rest.

At the time of admission, her heart rate was 120 per minute and blood pressure was 160/90 mm/Hg. On auscultation of chest, there were bilateral coarse crackles along with a pansystolic murmur at the mitral area. There was loss of R wave in the anterior chest leads in ECG. She was having a blood hemoglobin 10.3 gm/d, with a normal blood chemical profile. Echocardiography showed dilated left atrium with full sized left ventricle, moderate left ventricular contractility, mitral regurgitation grade III and aortic regurgitation grade I, with an over all ejection fraction (EF) of 40%.

A cardiologist opinion was sought to evaluate the progressively worsening symptoms of the patient, led to the diagnosis of peripartum dilated cardiomyopathy. Treatment started with furosemide, digoxin, GDN (glycerol dinitrate) infusion and potassium supplement. Once the

symptoms of congestive cardiac failure (CCF) resolved, delivery of the baby through caesarian section was planned.

A day prior to surgery, on pre-anaesthetic assessment, she was quite comfortable at rest. On examination pulse was 84/min, blood pressure was 150/90 mm Hg. There were fine crackles audible at both bases on auscultation of chest. She was having a mallampatti class II airway with normal neck movement. She was placed in ASA = III category and due to her deteriorating cardiac condition, surgery under epidural block was planned.

On arrival in operation room, she was eupnic, chest was clear on auscultation. Her pulse was 90/min & blood pressure was 140/90 mm Hg.

A double lumen venous catheter and an arterial cannula were placed under the effect of local anaesthetic into right internal jugular vein and right radial artery respectively. Five hundred milliliters of ringer solution was infused over a period of 20 minutes to increase the central venous pressure.

A lumbar epidural puncture was performed at her L3-L4 interface in sitting position and a catheter was placed in the epidural space through a 16 gauge toughy's needle. A mixture of lignocaine 1.0% and bupivacain 0.25% injected into the epidural space through the catheter. The level of block reached at T⁵ level in 10 minutes. A wedge was placed under right hip to minimize aorto caval compression. The surgery proceeded uneventfully resulting in delivery of a healthy baby girl.

Post operatively bupivacain 0.125% was given via epidural catheter on as required basis. She was kept in intensive care unit for 24 hours and then shifted to high dependency unit (HDD). Her post operative course was uneventful and she was discharged from hospital on 5th post operative day with an advice for follow up.

DISCUSSION

Peri-partum cardiomyopathy most often occurs in women over the age of 30 years during the last three months of the pregnancy or within 4-6 months after delivery⁵. It is an important cause of mortality and morbidity in association with pregnancy in the Asian and African countries⁶. It is also responsible for significant number of cardiac failure in pregnant female⁷. Identical risk factors for peripartum cardiomyopathy include advanced maternal age, multiparity, obesity, multiple gestation, pre-eclampsia, chronic hypertension and black race⁸.

Although etiology remain unknown but endomyocardial biopsies in women with peripartum cardiomyopathy have demonstrated myocarditis in many patients. The highest incidence of myocarditis reported was 76 % but one of the most recent series found myocarditis in only 8.8% patient⁹. Patients with peripartum cardiomyopathy presents with the typical signs and symptoms of left ventricular failure. The majority of cases occur after delivery and the immediate postpartum period. However, when the disease develops during the last months of pregnancy the diagnosis of cardiac failure is difficult to make by signs and symptoms alone, since some of these symptoms such as, fatigue, orthopnoea pedal edema are common among normal parturient during late pregnancy. A chest x-ray and echo- cardiography are very helpful for establishing a diagnosis of cardiac failure. Once it is identified, peripartum cardiomyopathy must be differentiated from other disease processes that lead to heart failure, such as valvular heart disease.

Parturient with peripartum cardiomyopathy require special anaesthetic care. The cardiovascular stress of labour and delivery may lead to cardiac decompensation. When that situation occur cesarean delivery may be required and anaesthesiologist may need to inform. Vasodilator agent such as nitroglycerine or nitroprusside for preload and after load reduction and dopamine,

dobutamine and milrinone for inotropic support may be used.

There is scanty information in the literature regarding anaesthetic management of peripartum cardiomyopathy. Some authors have argued the case for general anaesthesia in severe cases, as "cardiac reserve" is considered so limited that any reduction in systemic vascular resistance due to epidural blockade could be catastrophic¹⁰. Others have argued the case for regional anaesthesia on the basis that reduction in after load may be beneficial in a situation of poor ventricular function, where no outflow tract obstruction present¹¹. Roman shanideretal, presented a case report in 2001 of the use of combined spinal and epidural anaesthesia in a patient with peripheral dilated cardiomyopathy undergoing caesarian section. The choice for combined spinal epidural may be opted for several reason. First combined spinal epidural has a lower failure rate¹². Second, intraoperative patient satisfaction, anxiolysis and post operative pain score has been superior with combined spinal epidural¹³. Third, lower incidence of hypotensive episode with combined spinal epidural¹². Fourth, lower maternal and umbilical cord blood concentration of local anaesthetic¹². The advantage of epidural anaesthesia over general anaesthesia in peripartum cardiomyopathy is three folds¹⁴.

1. It avoid the need for either cardiodepressant drugs or high dose opioids technique necessitating post operative ventilation for both mother and infant.
2. Epidural anaesthesia may be induced slowly in a gradual and controlled manner while adjusted to fluid, and inotropes may be titrated to maintain optimum filling pressure and cardiac output.
3. Particular advantage in those patients with high susceptibility to aspiration of gastric

content, which makes slow intravenous induction of anaesthesia more difficult.

Major centro-neuraxial blockade may actually improve myocardial performance by reducing the after load on the left ventricle without improving contractility.

CONCLUSION

In our case, timely cardiologist help, early initiation of medical treatment careful administration of fluid under the guidance of invasive monitoring and a well tailored lumbarepidural technique satisfied the anaesthetic goal. That is why we believe that the well titrated lumbar epidural anaesthesia is the most appropriate option for patients with peripartum dilated cardiomyopathy undergoing caesarian section.

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