The Journal of Medical Research

Case Report

JMR 2021; 7(3):97-99
May- June
ISSN:2395-7565

www.medicinearticle.com
Received:11-05-2021
Accepted:10-06-2021

Undiagnosed Peripartum Cardiomyopathy for emergency Caesarean section: Lessons learnt

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Abstract

Peripartum cardiomyopathy is a form of dilated cardiomyopathy. It is a systolic heart failure of unknown aetiology and poorly understood pathophysiology. The prognosis of patients with PPCM is generally good with 70-80% recovering with ejection fraction >50% at the end of 6 months. However, the intrapartum period, especially in case of operative delivery, is fraught with danger.

Keywords: Caesarean section, Neuraxial anaesthesia, Peripartum cardiomyopathy.

INTRODUCTION

Peripartum Cardiomyopathy (PPCM) is heart failure that develops in the last month of pregnancy or up to 5 months postpartum with left ventricular systolic dysfunction (left ventricular ejection fraction <45% or fractional shortening <30% or both) without a clear cause. Black ethnicity, advanced maternal age, multiple gestations and co existing pre-eclampsia are all risk factors for PPCM [1]. The association of pre-eclampsia and eclampsia with PPCM may reflect a shared pathophysiology [2]. The close resemblance of the clinical presentation of cardiomyopathy with signs of normal pregnancy makes the diagnosis challenging. Elderly mothers, multiple gestations, pregnancy induced hypertension (PIH) are also associated with aggravated symptoms related to pregnancy. We present a case of undiagnosed peripartum cardiomyopathy presenting in the last month of pregnancy, highlighting the challenges we faced in her diagnosis and management.

CASE STUDY

A 40-year-old primigravida with twin pregnancy was admitted to our hospital with uncontrolled hypertension at 34.6 weeks of gestation. She gave history of increasing breathlessness over the past two days and worsening pedal oedema. There were no other symptoms of PIH with end organ involvement. On examination, the patient was sitting upright talking comfortably. Her heart rate was 85/minute and blood pressure (BP) 150/110 mm Hg while on regular treatment with Labetalol 200 mg thrice daily. Saturation on room air was 95%. There was pitting oedema up to the knees. On auscultation, there were fine basal crepts and normal heart sounds.

Treatment was started with a stat dose of oral Labetalol and intravenous $MgSO_4$, supplemental oxygen at 4 litres/min and catheterisation for input/output monitoring. A fresh set of investigations (complete blood count, liver function tests and coagulation profile) was sent. All these were within normal range, with haemoglobin of 10.7, platelet count of 245000/mm^3 and INR 1.14. A 2D echocardiogram was ordered.

The BP continued to remain high with serial diastolic readings above 110 mmHg. So, decision was taken to shift the patient to theatres for an immediate Caesarean section before further investigations could be done. In the absence of 2D echocardiographic findings, the patient was managed as an uncontrolled PIH patient with normal coagulation profile. The anaesthesia plan was a single shot spinal anaesthetic.

In theatre, the patient was given propped-up position with two pillows. Oxygen was continued at 6 litres/minute via mask. Standard non-invasive monitors were attached. 6% hydroxyethyl starch was started at 50ml/hr via a 20G peripheral line. Considering the severity of breathlessness, 20 mg injection Frusemide was given preinduction. In sitting position, spinal anaesthesia was administered using a

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25G pencil point needle. Heavy sensorcaine 0.5% 2ml with 10 µg fentanyl was given and the patient was maintained in a propped-up position. Serial assessment of the level of sensory and motor block was made over the next few minutes and the final position was given with a single pillow under the shoulders and 10 degree head up tilt of the operating table.

BP was monitored every 2 minutes and all equipment for resuscitation was kept ready in the event of maternal collapse. Adequate level was achieved and incision was taken after 8 minutes of induction. BP was maintained around 120 mmHg systolic using a Phenylephrine infusion (100 µg/ml). A small dose of 2-7 ml/hr was needed in the initial part of surgery and could be tapered off towards the end. After delivery of the twins, a bolus of injection oxytocin 3 Units was given over 2 minutes and 10 units was added to the colloid. Haemostasis and closure were completed in 30 minutes. Total blood loss was around 500 ml and urine output during surgery was 100 ml. Total 200 ml of colloid was administered intraoperatively. Blood pressure remained stable with systolic readings between 120-130 mmHg.

However, even after delivery of the neonates, the patient needed supplementary oxygen to maintain normal saturation. On auscultation, clear breath sounds were heard bilaterally with quiet bases. Another 20 mg of injection Frusemide was administered and a decision was taken to transfer the patient to ICU for close observation.

Within an hour of ICU admission, the patient deteriorated. Her BP could not be recorded on the non-invasive monitor and she was showing signs of agitation. Emergency intubation was done and an arterial line was sited. The first invasive arterial reading was 200/110 mmHg. Coarse crackles were heard on auscultation immediately after intubation. A 2D echocardiogram was done even as invasive lines were being secured. It showed a left ventricular ejection fraction of 15-20% and dilated left ventricle with normal valves.

Treatment for cardiomyopathy was started with a stat dose of 40 mg IV Frusemide followed by an infusion, infusion of Milrinone and positive pressure ventilation with PEEP. Bromocriptine was started through a nasogastric tube. Over the next two days the patient stabilised with gradually decreasing requirements of diuretics and Milrinone. She could be extubated on day 2, was discharged from ICU on day 4 and discharged home on day 7. Serial echocardiograms showed some improvement. Ejection fraction on day 2 was 25% and on day 5 was 25-30%. At one month follow up it had increased to 30-35% and at six months it stabilized at 40%.

DISCUSSION

PPCM is one of the causes of heart failure in pregnancy along with PIH, hypertensive cardiomyopathy, pulmonary hypertension and valvular heart disease [3]. Differentiating it from others requires careful history taking, clinical examination and specific investigations. Even the physiological changes of pregnancy, particularly in the cardiovascular and respiratory system can confound the diagnosis of early decompensated heart failure.

In pregnancy, there is profound increase in cardiac output, stroke volume and total blood volume along with a fall in systemic vascular resistance. BP decreases initially, but often there is an increase in the last trimester [4]. The decreased venous return from the lower extremities results in pedal oedema of varying degrees. The haemodynamic changes of pregnancy reach a peak by the 28th week. Further increases are to be expected during labour. The peak blood volume is reached immediately after delivery due to autotransfusion from the contracting uterus [5]. This is a particularly vulnerable period for the pregnant cardiac patient.

The increased oxygen and metabolic requirement in pregnancy is fulfilled by an increase in tidal volume and a less pronounced increase in respiratory rate. Pregnancy may also be accompanied by a subjective feeling of breathlessness without hypoxia. This is physiological and most pronounced in the third trimester [5]. Most of the cardiorespiratory changes are aggravated in multiple gestations.

Pre-eclampsia commonly results in pulmonary oedema due to increased forward pressure. The patient is at risk of heart failure, especially in case of fluid overload. The pathophysiology behind heart failure in PIH is the diastolic dysfunction. However, a small, transitory component of systolic failure may accompany some of these cases [6].

The various overlapping features notwithstanding, the presence of cyanosis, clubbing, resting tachycardia, any arrhythmia, hypertension or hypotension, signs of respiratory distress, elevated JVP, diastolic murmur, pansystolic murmur, or any signs of pulmonary oedema should prompt further evaluation of the pregnant patient [7]. These are warning signs of an underlying pathology that needs antepartum optimisation and treatment.

Echocardiography remains the gold standard for diagnosing PPCM. Without this crucial investigation, we went ahead with what is considered the ideal anaesthetic for a caesarean section in a PIH patient, i.e. spinal anaesthesia.

There is no single best anaesthetic technique for PPCM patients undergoing operative delivery. General and regional anaesthesia are both acceptable choices. More important than the choice of anaesthesia technique, is the appropriate management of haemodynamics. Vigilant monitoring and prior planning for the worst possible clinical scenario are essential for a good outcome.

While we chose a spinal anaesthetic for the surgery, minor modifications were made keeping in mind the possibility of PPCM. A prophylactic dose of diuretic was administered. After induction of anaesthesia, the final position was given slowly over 5-7 minutes allowing the level of spinal block to ascend gradually. We relied more on vasopressors than on fluids to maintain the mother's blood pressure. Throughout the intraoperative period, the team was ready for resuscitation in case of deterioration of the patient's clinical condition. Also, the decision to transfer the patient to ICU allowed emergency treatment without any delays.

ICU treatment for our patient included medication for acute heart failure, such as vasodilators and diuretics. Along with these Bromocriptine was used to supress Prolactin secretion. A 16kDa fragment of prolactin, produced by the cleavage of the hormone is thought to be one of the factors responsible for the imbalance between pro and anti angiogenic factors in the body [8]. This dysregulation is believed to be the cause of PPCM.

Several valuable lessons were learnt from this case of an undiagnosed PPCM. We now have a low threshold for a thorough cardiac evaluation of pregnant patients with severe breathlessness. Our patient had several risk factors for PPCM, with age > 40 years, PIH, twin pregnancy. For such patients, a 2D echocardiogram is now part of the antenatal work up in the third trimester. Of course, we have to bear in mind that less than 10% cases present in the prepartum period and majority are diagnosed within 4 months after delivery [9].

CONCLUSION

So even with the best antenatal work up, we may have an expectant mother presenting with symptoms for the first time at the time of, or immediately after delivery. Making it a safe surgical experience for all these patients is the real-life test of our knowledge and training.

Conflicts of Interest

The authors declare no conflicts of interest.

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