A PARTURIENT WITH GESTATIONAL HYPERTENSION AND ANTERIOR PLACENTA PREVIA COMING FOR LSCS DIAGNOSED TO HAVE PERIPARTUM CARDIOMYOPATHY

Priyadarshini M. Bentur[1], Kavya K. G[2], Priodarshi Roy Choudhury[3]

[1]Associate Professor, Department of Anaesthesia, J.J.M. Medical College, Davanagere, Karnataka.
[2]Consultant Anaesthetist, Department of Anaesthesia, Bangalore, Karnataka.
[3]Consultant Anaesthetist, Department of Anaesthesia, Kolkata, West Bengal.

HOW TO CITE THIS ARTICLE: Bentur PM, Kavya KG, Choudhury PR. A parturient with gestational hypertension and anterior placenta previa coming for LSCS diagnosed to have peripartum cardiomyopathy. J. Evid. Based Med. Healthc. 2018; 5(48), 3351-3353. DOI: 10.18410/jebmh/2018/682

PRESENTATION OF THE CASE

Peripartum cardiomyopathy is an uncommon but life-threatening disease that affects women in the last few months of pregnancy or within the first five months after delivery, without previous evidence of heart disease & with no other definable cause of heart failure.[1]

We present a case report of a 37-year-old patient with gestational hypertension & anterior placenta previa who developed pulmonary oedema intraoperatively and was managed postoperatively in the ICU and was diagnosed as peripartum cardiomyopathy on Echocardiography.

CLINICAL DIAGNOSIS

Parturient with gestational hypertension and anterior placenta previa developing pulmonary oedema intraoperatively.

DIFFERENTIAL DIAGNOSIS

1. Pulmonary oedema secondary to hypertension.
2. Pulmonary oedema secondary to peripartum cardiomyopathy with low ejection fraction.
3. Pulmonary oedema secondary to fluid overload.
4. Prostodin induced bronchospasm resulting in pulmonary oedema.

PATHOLOGICAL DISCUSSION

A 37-year-old (70 Kgs) patient G_5P_1 L_1A_3, 38 Weeks gestation with gestational hypertension and anterior placenta previa was taken up for elective LSCS.

On preoperative examination, she was well built and nourished without comorbidities. Her antenatal checkups were regular, she was diagnosed to have gestational hypertension at 35 weeks of gestation and was started on Tablet labetalol 100 mg OD.

Patient had on and off productive cough with scanty expectoration, since 1 month, for which she was treated with a course of antibiotics. Her cough had subsided 1 week prior to LSCS.

Financial or Other, Competing Interest: None.
Submission 17-10-2018, Peer Review 20-10-2018,
Acceptance 07-11-2018, Published 26-11-2018.
Corresponding Author:
Dr. Kavya K. G,
#11, 8th Main, Girinagar,
Banashankari 3rd Stage,
Bangalore- 560085, Karnataka.
E-mail: kavya.gudi@gmail.com
DOI: 10.18410/jebmh/2018/682

On examination, Heart rate was 90 bpm and BP – 140/90 mmHg, no pallor, icterus, cyanosis or lymphadenopathy but patient had bilateral pitting oedema of lower limbs.

Airway examination revealed Mallampati class III.

Cardiovascular & Respiratory examination was within normal limits.

Preoperative Investigations (CBC, electrolytes, RFT, LFT) were within normal limits.

Her LDH levels were slightly on the upper limit – 307 IU/L.

Since she was a case of anterior placenta previa – general anaesthesia was planned and two pints whole blood arranged, anticipating severe haemorrhage.

The patient was kept nil per orally for 6 hours before surgery. Two 18 G IV cannula were secured, and Ringer lactate was started. ECG, NIBP, pulse oximeter monitors were attached.

Patient was premedicated with injection ondansetron 4 mg iv, injection Ranitidine 50mg IV, injection glycopyrrolate 0.2 mg iv.

She was induced with injection propofol 120mg IV and once induced, cricoid pressure was applied (SELLICK'S MANOEUVRE), injection succinylcholine 120mg was given I.V. Once relaxed, Laryngoscopy was performed, since it was Cormack Lehanac Grade 3, her trachea was intubated with the help of a guide wire {BOUGIE} with a 7-size cuffed ETT, cuff was inflated cricoid pressure was released and b/1 equal air entry checked, and tube secured in position.

Patient was maintained on O2 + N2O + intermittent vecuronium and IPPV intraoperatively.

Baby was extracted 15 minutes following the onset of surgery and had an APGAR score of 9 after 5 min following delivery. Patient was given Inj midazolam 1-5 mg and inj fentanyl 150mg IV.

Oxytocin infusion was started in RL at the rate of 0.04 IU/min.

B.P. was 170/110 mm of Hg and HR 100 bpm after 25 min of start of surgery. In view of high BP – Inj. labetalol 10mg was given slow iv.

Since the uterus was atonic inspite of increase in oxytocin infusion, Inj. Prostodin 250mg was given I.M. Following this uterus was well contracted and closure of uterine incision was started.

During closure, patient developed acute bronchospasm, bilateral extensive rhonchi and basal

crepitations on auscultation. SPO2 was 90% with 6 ltrs. of O_2. Inj. Deriphyllin and inj. Hydrocortisone 100mg I.V stat, were given.

Frothy secretions started coming out of the ETT, following which inj. Lasix 40mg I.V. was given, and ventilation was continued with 100% O2 and 15 degree propped up position in view of acute pulmonary oedema. SpO_2 came up to 95% following this.

After the completion of surgery, she was shifted to ICU for elective ventilation, acute pulmonary oedema been suspected. Mechanical ventilation was continued in the ICU with FiO_2 – 0.6, TV- 500ml, RR -12/Min and PEEP-8.

Propped up position was given, and patient was started on Inj. Lasix 20mg 6th hrly., Inj. Morphine 6mg stat and repeated 6th hourly, broad spectrum antibiotics, Inj. Hydrocortisone 100mg TID.

All the necessary investigations were sent. ABG showed metabolic acidosis, PH=7.18, PaCO2- 48.9, PO2= 73mmHg, PaO2= 83mm of Hg for which correction was given. Bedside chest x-ray was performed which showed bilateral diffuse infiltrates and enlarged cardiac shadows. ECG showed left axis deviation and LV strain pattern.

Cardiac enzymes i.e. CK-MB, came out to be elevated mildly i.e. 77.3 U/L. Bedside echocardiogram could not be done due to lack of facility.

Patients heart rate was 150bpm and BP 130/80 mm of Hg on ventilator. She was started on tab metoprolol 100mg BD through Ryles tube.

On the 2nd day on ventilator, the patient's vitals were well maintained and SPO2 was 98%.

On auscultation of the chest – there was bilateral equal air entry with minimal basal crepitations. After assessing the general condition of the patient and ABG values she was weaned off from the ventilator and extubated.

Patient was kept for observation in the ICU and all the medications were continued, SPO2 was 98% with O2 at 4 ltrs via facemask. Vitals were well maintained.

Echocardiogram was performed on the 3rd postoperative day which showed global hypokinesia (anterior > posterior), dilated LV, with trivial MR and TR, moderately decreased LV systolic function, LVEF-43%, no pulmonary artery hypertension. It was diagnosed as peripartum dilated cardiomyopathy on echocardiogram. Patient was started on Tab. furosemide 20mg OD, Tab. Ecosprin 75mg OD.

Patient was discharged on postop day 7. She was advised salt restricted diet, repeat echocardiogram a week later and consult a cardiologist in this regard.

DISCUSSION OF MANAGEMENT

PPCM occurs in approximately 1 per 10, 000 deliveries with a reported mortality range of 30-60%.[1] The hallmark of the disease is onset of decreased cardiac ejectile force either in late pregnancy or early puerperium.

Many risk factors have been implicated in the development of this disease condition which include advanced maternal age, multiparity, twin pregnancy, pre-eclampsia, gestational hypertension & diabetes mellitus.[2]

We report a case of gestational hypertension with anterior placenta previa who was taken up for elective LSCS.

In view of anterior placenta previa anticipating severe haemorrhage, general anaesthesia was planned. Antepartum haemorrhage complicates 3-5% of the pregnancies and is a leading cause of perinatal and maternal mortality worldwide.[3] The incidence is increased beyond the age of 35 years, with high birth order pregnancies and in multiple pregnancies. The mode of anaesthesia for caesarean section depends upon many factors. A regional technique may be appropriate for posterior placenta previa, with no active bleeding.

Patients with placenta previa remain at risk for increased intraoperative blood loss for atleast 3 reasons– the obstetrician may cut into the placenta during uterine incision, after delivery lower uterine segment implantation site does not contract, placenta previa is at increased risk for placenta accreta and uterus does not contract for the same reason.[4]

Prostodin is 15 – methyl prostaglandin F2 alpha, which is a uterotonic agent. It acts as a smooth muscle stimulant and is a recognized second line agent for use in management of postpartum uterine atony unresponsive to oxytocin / ergometrine. Side effects of PGF 2 alpha include nausea, vomiting, diarrhoea, bronchospasm and systemic HTN.[5] Although pulmonary oedema after administration of prostaglandins is common in patients with pre-existing cardiac disease, it has also been reported in patients with normal heart.[6] Prostodin was used in our case because of mild uterine atony. Since pulmonary oedema developed following injection of Prostodin, it was suspected to be the cause for the same.

Peripartum cardiomyopathy is a rare but devastating form of heart failure which by definition has its onset during the last month of pregnancy or during, first 5 months postpartum.[7,8]

The prevention is often insidious, initially clinical presentation may be limited to symptoms of mild URTI, chest congestion and fatigue. These symptoms progress rapidly to florid cardiac failure with biventricular hypokinesia, low Cardiac Output, elevated filling pressure and ventricular ectopy.

Aetiology of PPCM is unknown-viral, autoimmune and toxic factors have been implicated. PPCM seems to be more common in women with multiple gestation, PE, obesity or advanced maternal age. Our patient had advanced maternal age, gestational HTN and obesity as some of the predisposing factors for PPCM. Treatment goal is to reduce amount of volume returning to the heart (preload reduction), decrease resistance against which heart must pump (afterload reduction) and increase contractile force of heart (ionotropy).[9]

In our case use of Prostodin might have precipitated pulmonary oedema in an already compromised cardia of

the patient because of underlying gestational HTN and peripartum cardiomyopathy.

FINAL DIAGNOSIS

Parturient with Gestational Hypertension
Anterior Placenta Previa
Intraoperative Pulmonary Oedema during LSCS
Peripartum Cardiomyopathy.

Figure 1. Intraoperative Vitals

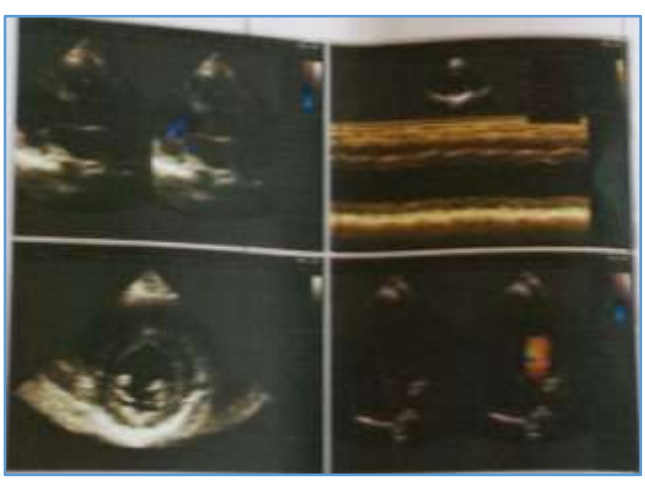

Figure 2. 2D Echo done after Recovery from ICU Stay showing Peripartum Cardiomyopathy with Ejection Fraction of 43%

REFERENCES

[1] Chan F, Ngan Kee WD. Idiopathic dilated cardiomyopathy presenting in pregnancy. Can J Anaesth 1999;46(12):1146-1149.
[2] Popescu WM. Heart failure and cardiomyopathies. Chap- 6. In: Paul AK, ed. Stoelting's anesthesia and co-existing disease (Second South Asia Edition). 2nd edn. Elsevier 2014: p. 139.
[3] Calleja-Agius J, Custo R, Brincat MP, et al. Placental abruption and placenta praevia. Eur Clin Obstet Gynaecol 2006;2:121-127.
[4] Mzyer DC, Smith KA. Antepartum and postpartum hemorrhage. Chap- 37. In: Chestnut D, Polley L, Wong C, et al, eds. Chestnut's obstetric anesthesia: principles and practice. 4th edn. Mosby Elsevier Publishers 2009: p. 812.
[5] Hayashi RH, Castillo MS, Noah ML. Management of severe postpartum hemorrhage with a prostaglandin F2alpha analogue. Obstet Gynecol 1984;63(6):806-808.
[6] Rodriguez de la Torre MR, Gallego Alonso JI, Gil Fernandez M. Pulmonary edema related to administration of 15-methyl-prostaglandin F2alpha during a cesarean section. Rev Esp Anestesiol Reanim 2004;51(2):104-107.
[7] Demakis JG, Rahimtoola SH, Sutton GC, et al. Natural course of peripartum cardiomyopathy. Circulation 1971;44(6):1053-1061.
[8] Lampert MB, Lang RM. Peripartum cardiomyopathy. Am Heart J 1995;130(4):860-870.
[9] Indira K, Sanjeev K, Sunanda G. Sequential combined spinal epidural anaesthesia for caesarean section in peripartum cardiomyopathy. Indian J Anesth 2007;51(2):137-139