CASE REPORT

Anaesthesia Management in a Case of Large Ventricular Septal Defect with Eisenmengerisation Undergoing Caesarean Section

Kirti A. Kundalwal[1], Surekha Shinde[1], Prajakta M. Tayade[1]*

[1]Department of Anaesthesia, B. J. Govt. Medical College, Pune- 411001(Maharashtra) India

Abstract:

Incidence of cardiac disease in pregnancy in developed countries is 0.2-3% and that in developing countries is still higher. Ventricular Septal Defect (VSD) is one of the commonest congenital heart diseases. Pregnant patient with small VSD generally tolerate well but patients with unrepaired large VSD develop pulmonary hypertension and left heart failure over a period of time. If pulmonary pressure reaches systemic levels there is reversal or bidirectional flow. As per WHO classification of maternal cardiac risk disease, pulmonary hypertension is categorized under class 4. Here we report a case management of a pregnant patient with large VSD (15 mm) with severe pulmonary hypertension (105 mmHg) with eisenmengerisation posted for emergency caesarean section. It was done under general anaesthesia with successful maternal and foetal outcome.

Keywords: Anaesthesia, Pregnancy, Ventricular Septal Defect, Eisenmenger's syndrome

Introduction:

Ventricular Septal Defect (VSD) is one of the common congenital heart diseases and is present in 1.5-2.5 of 1000 women with a pregnancy resulting in live birth [1]. Isolated VSD is usually well tolerated during pregnancy but patients with large VSD complicated with other cardiac problems poses a significant risk to anaesthesia. Here we report a case management of a primigravida with large VSD with severe pulmonary hypertension with Eisenmenger's syndrome posted for caesarean section.

Case Report:

A 22 year old 50kg primi gravida (36 weeks 5 days) came with chief complaints of breathlessness at rest since 8 days without any significant past history. On examination, she was thin built with 156 cm height. Lying in left lateral position she had PR-110 per minute, BP- 150/90 mmHg, RR- 24 per minute and oxygen saturation-86% on oxygen flow of 4 lit/min by face mask. Bilateral pedal oedema was present. There was no pallor, clubbing, lymphadenopathy or cyanosis. On systemic examination, bilateral basal crepts were present. Cardiac auscultation demonstrated pansystolic murmur in left parasternal area with ejection systolic murmur in pulmonary area and loud P_2. On neurological examination patient was conscious, oriented.

On evaluation her 2-D echocardiography revealed complex congenital cyanotic heart disease with large ventricular septal defect of 15mm size with severe pulmonary artery hypertension with bidirectional shunt, right ventricular systolic pressure-105 mmHg, overriding of aorta >50%, aorta from morphological right ventricle with double outlet right ventricle, mildly dilated right ventricle and right atrium, grossly dilated pulmonary artery with left ventricular ejection fraction of 60%. ECG revealed right ventricular hypertrophy, T wave inversion in avL, avR leads. Arterial blood gas analysis showed pH-7.45, PaO_2-71, $PaCO_2$-30, SO_2-85%, HCO_3-20.3. Lab investigations including hemogram, renal and

liver function tests, coagulation profile were within normal limits.

Considering the patient's complex cyanotic cardiac disease and general condition at presentation, it was decided to deliver the baby by caesarean section under general anaesthesia. After confirming nil by mouth status patient was shifted to operation theatre. Central venous and radial arterial line was secured. ECG and pulse oximeter were attached. Infective endocarditis prophylaxis was given. Her baseline parameters were showing HR-110/min, regular, ABP- 160/100 mmHg, CVP-15cm H_2O, SO_2-86-88% on ventimask. IV line was secured with 18G cannula in left upper limb. Special attention was given to avoid air bubbles in IV lines. Patient was premedicated with Ondansetron 4 mg iv, Ranitidine 50 mg iv, Glycopyrrolate 0.2 mg im. About 15˚ of left lateral tilt was maintained to avoid any supine hypotension. After preoxygenation with 100% oxygen for 3 minutes, Xylocard 60 mg was given iv slowly followed by rapid sequence induction with etomidate 14 mg iv and succinylcholine 50 mg iv . Patient was intubated with ETT no.7 cuffed and ventilated manually to maintain $EtCO_2$ of 32-35 mmHg.

Anaesthesia was maintained on 0.8% sevoflurane with oxygen and Injatracurium. Nitrous oxide was avoided. After delivery of live, healthy baby of 3 kg, 20 units of Oxytocin in 500 ml crystalloid were given slowly over 30 minutes. Midazolam 1 mg and Fentanyl 50 µg iv was given after delivery of baby. Total surgical duration was 1 hour. She received 1 litre of crystalloid intraoperatively. Total blood loss was 500 ml and urine output of 500 ml. She was stable hemodynamically throughout the surgery with oxygen saturation of 94-96%. About 20 mg lasix was given at the end of surgery. For postoperative analgesia 100 ml of iv Paracetamol was given. Also local infiltration with local anaesthetics was done on incision site. Patient was extubated on table with inj. glycopyrrolate and neostigmine for reversal. Postextubation her vital parameters were BP-130/90 mmHg, p-100/min, SpO_2-94% on O_2 by ventimask, CVP-12cm H_2O. Patient was shifted to surgical ICU for intensive management of her cardiac condition in collaboration with cardiologist and obstetrician.

Discussion:

Cardiac disease in pregnancy is still a major problem worldwide, particularly in poor countries although its incidence varies between 0.1- 4 % [2]. It's a significant cause of maternal death worldwide [3]. Pregnancy itself is associated with substantial and progressive hemodynamic changes starting early in pregnancy. Major alterations in pregnancy include 30-50% increase in both cardiac output and blood volume, in addition to decreased blood pressure. In cardiac pregnant patients, these changes may lead to clinical decompensation, exposing these women to potentially life threatening situations [4].

VSD is one of the common congenital heart diseases and is present in 1.5-2.5 of 1000 women with a pregnancy resulting in live birth [1]. Pregnant women with isolated VSD generally tolerate pregnancy well if shunt is small to moderate and if pulmonary artery pressure is normal [5]. But the presence of pulmonary hypertension increases risk of ventricular dysfunction [6]. As per modified WHO classification of maternal cardiac risk disease, pulmonary hypertension is categorized under class 4 [7]. Pulmonary hypertension in pregnancy is associated with high mortality (30-50%) [8].

The presence of large VSD in our patient with severe pulmonary hypertension not maintaining oxygen saturation in spite of oxygen therapy was

the indication of termination of pregnancy and LSCS for better maternal and foetal outcome, but these are high risk patients for anaesthesia. Several authors have suggested epidural anaesthesia as a choice of anaesthesia for these patients of Eisenmenger's syndrome [9-10] but considering the reduction of SVR by inevitable high sympathetic blockade of epidural anaesthesia which can increase right to left shunt and general condition of our patient at presentation, we preferred general anaesthesia. Our main aim was to avoid hypothermia, hypercarbia, acidosis, hypoxia and high ventricular pressures which all can increase pulmonary vascular resistance and we maintained systemic vascular resistance. We preferred etomidate as induction drug which causes less hemodynamic alterations. Nitrous oxide was avoided which is potent pulmonary vasoconstrictor. Xylocard was used to avoid pressor response of laryngoscopy and intubation. Patient was ventilated as per $ETCO_2$ levels to maintain normocarbia. After delivery of baby oxytocin was given slowly over 30 minutes in drip and bolus dose was avoided to avoid hypotension and tachycardia.

Conclusion:

Choice of anaesthesia in obstetric cases with heart disease changes depending on structural pathology of the disease and clinical status of the patient at presentation, though regional anaesthesia is safe anaesthesia in many cases. So, each patient should be individually assessed. Meticulous examination, early risk assessment and intervention with combined care by a team of anaesthesiologist, obstetrician and cardiologist gives rise to better outcome in pregnancy associated with high risk heart disease.

References

1. Jackson GM, Dildy GA, Varner MW, Clark SL. Severe pulmonary hypertension in pregnancy following successful repair of ventricular septal defect in childhood. *Obstet Gynecol* 1993;82(4 Pt 2 Suppl):680-2.
2. Klein LL, Galan HL. Cardiac disease in pregnancy. *Obstet Gynecol Clin North Am* 2004; 31(2):429-59.
3. Pretoria: A report of the National Committee on Confidential Enquiries into maternal deaths in South Africa (1999-2001) DOH: 162-74.
4. Ouyang DW, Khairy P, Fernandes SM, Landzberg MJ, Economy KE. Obstetric outcomes in pregnant women with congenital heart disease. *Int J Cardiol* 2010; 144(2):195-9.
5. Kuczhowski KM. Labor analgesia for parturient with cardiac disease- what does an obstetrician need to know? *Acta Obstet Gynecol Scand* 2004; 83(3): 223-33.
6. Weiss BM, Hess OM. Pulmonary vascular disease and pregnancy: current controversies, management strategies, and perspectives. *Eur Heart J* 2000; 21(2):104-15.
7. European Society of Gynecology (ESG); Association for European Paediatric Cardiology (AEPC); German Society for Gender Medicine (DGesGM), Regitz-Zagrosek V, Blomstrom Lundqvist C, Borghi C, *et al.* ESC Guidelines on the management of cardiovascular diseases during pregnancy. *Eur Heart J* 2011; 32(24):3147-97.
8. Borges VT, Magalhaes CC, Martins AM, Matsubura BB. Eisenmenger's syndrome in pregnancy. *Arq Bras Cardiol* 2008; 90(5):39-40.
9. Ammash NM, Connolly HM, Abel MD, Warnes CA. Noncardiac surgery in Eisenmenger syndrome. *J Am Coll Cardiol* 1999; 33(1):222-7.
10. Pollack KL, Chestnut DH, Wenstrom KD. Anaesthetic management of a parturient with Eisenmenger's syndrome. *Anesth Analg* 1990; 70(2):212-5.

[*] ***Author for Correspondence:*** *Dr. Kirti A. Kundalwal, Department of Anaesthesia, B. J. Govt. Medical College, Pune- 411001(Maharashtra) India Email:drkirtikundalwal@gmail.com Cell:9112196906*