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CASE REPORT: ANAESTHETIC MANAGEMENT FOR CAESAREAN SECTION IN PREGNANT WOMAN WITH SEVERE PULMONARY ARTERY HYPERTENSION DUE TO MITRAL STENOSIS

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ABSTRACT

Introduction: Pulmonary artery hypertension (PAH) in pregnancy is known to cause high maternal mortality. we report the peri-operative management of a case of Pulmonary artery hypertension due to mitral stenosis. **Case Report**: A 28 year old primi gravida presented with progressive dyspnea on exertion, multiple episodes of hemoptysis at 27 weeks of gestation. she was diagnosed to have severe pulmonary artery hypertension and caesarean section was planned at 34 weeks of gestation. A combined spinal and epidural anaesthesia was used along with invasive cardiac monitoring. **Conclusion**: We preferred to continue spinal anaesthesia because gradually increasing the local anaesthetic dose during the procedure may minimize probable undesirable hemodynamic changes, such as hypotension and tachycardia.

KEYWORDS: Mitral Stenosis, Pulmonary hypertension, Pulmonary vascular resistance, Regional Anaesthesia.

INTRODUCTION

Pulmonary Hypertension (PH) is defined as a persistent elevation of mean pulmonary artery pressure (MPAP) ≥ 25mmHg at rest. Pregnancy in patients with PAH causes a very high risk

with a mortality rate of 30- 50%.[2] The perioperative management of pregnant women with PAH posted for cesarean section is challenging. Anesthesiologists have a leading role in the perioperative care of these patients as they may develop respiratory or right ventricular failure anytime in the perioperative period. With the advent of PAH specific therapy such as prostacyclin analogues, phosphodiesterase (PDE) 5 inhibitors, endothelin receptor antagonists, inhaled nitric oxide and iloprost, mortality has come down to around 25%.

CASE REPORT

A 28 year old primi gravida presented with progressive dyspnea on exertion, fatigability and multiple episodes of hemoptysis at 27 weeks of gestation without any similar history. On examination, pale and had bilateral pitting pedal edema. Cardiovascular system examination revealed a left parasternal heave and loud P2. Chest X- ray with an abdominal shield showed prominent main pulmonary artery and pruning of pulmonary vasculature. A 12 lead ECG revealed right axis deviation and right ventricular hypertrophy. Echocardiography evaluation revealed right atrial/ right ventricular/ main pulmonary artery dilatation, moderate tricuspid regurgitation and severe PAH with a tricuspid regurgitation pressure gradient of 107mmHg. Ultrasonographic examination of abdomen and pelvis and color doppler evaluation of both lower limbs was within normal limits. Her symptoms improved with rest, medical management with oxygen inhalation by simple mask, oral sildenafil 50mg thrice daily and other supportive measures but remained dyspneic on performing her routine activities (NYHA class III). Elective caesarean section was planned on completion of 34 weeks of gestation. After preoperative counseling, regional anesthesia was planned. Oxygen inhalation and oral sildenafil were continued in the preoperative period. In the operating room, a wedge was kept under her right buttock to prevent aortocaval compression and left radial arterial line was placed under local anesthesia to assess beat to beat variation in systemic blood pressure. A Swan Ganz pulmonary artery catheter was floated into the pulmonary artery to monitor MPAP, PCWP, pulmonary vascular resistance (PVR) and continuous cardiac output (CCO). Her baseline vital parameters read as: heart rate (HR) -104/minute, arterial blood pressure (ABP) -101/54mmHg, oxygen saturation (SpO2) – 96% on oxygen by simple mask at 6 liter /minute, arterial oxygen saturation (PO2) -106mmHg, central venous pressure (CVP) -10mmHg, PAP -114/59mmHg, MPAP -79mmHg, PCWP -18mmHg, systemic vascular resistance (SVR) -960 dyn-s/cm5, PVR 976 dyn-s/cm5. CCO was monitored by Edwards Vigilance II monitor. A combined spinal epidural (CSE) was administered with 0.5% heavy bupivacaine 0.5ml intrathecally and 0.25% bupivacaine epidurally 5 ml bolus in divided

doses followed by 0.125% infusion at 5ml/hour. Episodes of hypotension were managed with boluses of arginine vasopressin (AVP) 0.1U/ml which did not cause any increase in MPAP and PVR. Epidural infusion was withheld to avoid profound hypotension. Caesarean section proceeded after attaining adequate level of block. Following delivery of baby, MPAP, PVR and SVR were recorded and continuous nebulisation with milrinone (1mg/ml) 1ml over 10 minutes alternated with alprostadil (10µg/ml) 2ml over 10 minutes was started via a jet nebulizer attached to a non rebreathing mask with an oxygen flow of 6 to 8 liters /minute. Oxytocin was administered slowly as an infusion (5U in 50 ml) to attain adequate uterine contraction. Twenty minutes after nebulisation, MPAP and PVR came down to 67mmHg and 767dyn-s/cm5 from previous values of 77mmHg and 894dyn-s/cm5 respectively. Her SVR remained stable at 956 dyn-s/cm5 with a previous value of 965 dyn-s/cm5. Her SpO2 improved to 99% and PO2 to 146mmHg. At 30 minutes, her MPAP, PVR and SVR read as 66mmHg, 783dyn-s/cm5 and 968dyn-s/cm5 respectively. She developed hypotension on restarting epidural infusion which was tackled by vasopressin infusion (1U/ml) starting at 1ml per hour titrated to maintain SVR within normal range. Although SVR improved with vasopressin infusion, no associated rise in PVR was noted. After shifting the patient to intensive care unit (ICU), levosimendan (0.25mg/ml) was started as an infusion at 2ml/hour without a loading dose, titrated to desired hemodynamic effect, for improving the right ventricular function. AVP and levosimendan infusions were continued for 48 hours. Inhaled milrinone and inhaled alprostadil were continued half hourly for 48 hours with close monitoring of MPAP, PVR and SVR. Post operative analgesia was provided by epidural infusion of 0.0625% bupivacaine with fentanyl (2µg/ml). At 24 hours MPAP, PCWP, PVR and SVR values read as 64mmHg, 14mmHg, 769dyn-s/cm5 and 1119dyn-s/cm5 respectively. Her cardiac index (CI) improved gradually from 2.1 to 3.3 liter/min/m2 over 48 hours. Oral sildenafil was restarted on first postoperative day and low molecular weight heparin was added. She was closely monitored in the ICU for two weeks and shifted to the ward. One month later she was stable with NYHA class II symptoms.

DISCUSSION

In the present case, the patient had dyspnea at rest – NYHA class IV, RV hypertrophy and suprasystemic systolic PAP which are all predictors of morbidity and mortality.[6] Any stress including pain, anxiety, hypoxemia, hypercarbia or acidosis can cause a rise in PVR leading to hypertensive crisis and right ventricular failure.[7] Regional anesthesia with invasive cardiac monitoring was chosen in this case as we wanted to avoid the stress of laryngoscopy

and intubation, effect of positive pressure ventilation on venous return and myocardial suppression by anesthetic agents.[8] Moreover, regional anesthesia block sympathetic tone, provides adequate pain relief and thus prevents precipitous rise in PVR. A combined spinal epidural (CSE) allows pain control in the postoperative period avoiding use of opioids which may cause respiratory depression resulting in hypercarbia and hypoxia that further worsens PAH.[9] We made all efforts to avoid hypoxia and hypercarbia that helped in improving PVR. The use of pulmonary artery catheter (PAC) in PAH is controversial as there is a high risk of pulmonary artery rupture and thrombosis.[10] We took the advantage of PAC to closely monitor the PAP and PVR and the response to interventions. Hypotension following CSE was initially managed with vasopressin to maintain SVR. Right ventricular coronary perfusion pressure depends on the difference in the pressure gradients between aorta and RV. Systemic hypotension in patients with RV hypertrophy causes ischemia to RV myocardium. AVP was chosen as data suggests that it causes pulmonary vasodilatation in preconstricted pulmonary arteries whereas it mediates vasoconstriction in systemic circulation. The systemic vasoconstriction is mediated through G protein coupled V1 receptor on vascular smooth muscle cells. The suggested mechanism for pulmonary vasodilatation is NO production via the V1 receptor.[11] There are reports of effective use of arginine vasopressin in management of low systemic vascular resistant hypotension concomitant with PH without causing significant change in PVR.[12] There are no published data about the dose range of AVP for pulmonary vasodilatation. We used AVP 0.1U boluses initially and changed over to infusion 1-4U/hour. We did not notice any increase in PVR following administration of the drug. Inhaled vasodilators are selective for pulmonary circulation avoiding potentially deleterious systemic side effects. Inhaled milrinone is found to be effective in reducing PAP without causing reduction in MAP or SVR.[13] It causes vasodilatation of pulmonary vasculature adjacent to well ventilated alveoli reducing the ventilation perfusion mismatch and improving the oxygenation.[14] A dose of 1mg/ml was chosen as some studies report adequate reduction in PVR with no systemic side effects at this dose even after continuous nebulisation for four hours.[13] One study has reported that MPAP and PVR values return to the baseline 20 minutes after a 15 minute inhalation of milrinone.[15] Inhalation of aerozolised alprostadil (PGE1) has also been found to be effective in the management of PAH.[16] There are reports showing lower MPAP and higher MAP and PO2 with PGE1 inhalation compared to PGE1 infusion in PAH following corrective surgery for congenital heart disease.[17] Inhaled PGE1 directly acts on pulmonary vasculature and undergoes extensive pulmonary metabolism with little systemic absorption.[18] Inhaled milrinone is found to have an additive

pulmonary vasodilatory effect to inhaled PGI2 and the combination may prolong the duration of pulmonary vasodilation.[15] To our knowledge, there is no published data regarding combination of inhaled milrinone and inhaled alprostadil in perioperative management of PAH for caesarean section. With the combination therapy there was reduction in the MPAP and PVR without causing a fall in MAP or SVR in the present case. Levosimendan is a calcium sensitizer that improves the myocardial contractility without increasing oxygen consumption. It also causes vascular smooth muscle relaxation by opening of ATP sensitive K+ channels. There are reports showing improvement in RV systolic and diastolic function with levosimendan in patients with advanced cardiac failure.[19] Levosimendan restores RV-PA coupling better than dobutamine as there is an additional pulmonary vasodilatory effect. We used levosimendan as an infusion to reduce right ventricular afterload and to improve RV performance.[20]

CONCLUSION

We emphasize on a multidisciplinary approach to the perioperative management of suprasystemic PAH for caesarean section. A combination of inhaled milrinone and inhaled alprostadil is a useful, safe and cost effective alternative to inhaled nitric oxide or iloprost which are often unavailable in the developing countries for selective pulmonary vasodilator therapy. Arginine vasopressin may be used to support coronary perfusion during hypotension following regional technique for caesarean section without increasing PVR and levosimendan infusion may be helpful in preventing RV failure in perioperative period. We advocate critical care support and close monitoring extended to the postpartum period.

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