Suprasystemic pulmonary artery hypertension for caesarean section: a case report

Deepu Antony1, Shibu C Kallivalappil1, Mariam Koshi Thomas1, Sugatha M Prakash1

Abstract

Introduction: Pulmonary artery hypertension (PAH) in pregnancy is known to cause high maternal mortality. We report the perioperative management of a case of suprasystemic pulmonary artery hypertension for caesarean section.

Case Report: A 26 year old primi gravida presented with progressive dyspnea on exertion, fatigability and multiple episodes of hemoptysis at 26 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 epidural anaesthesia was used along with invasive cardiac monitoring. Arginine vasopressin (AVP) was used to treat hypotension to improve systemic vascular resistance (SVR) without increase in PVR. A combination of inhaled milrinone and inhaled alprostadil was used in the perioperative period for selective reduction in pulmonary vascular resistance (PVR). Levosimendan infusion was used as an inotropic support to right heart.

Conclusion: A multidisciplinary approach in this regard with critical care extended into the postpartum period may be beneficial for better maternal survival in cases of suprasystemic pulmonary artery hypertension.

Keywords: Pulmonary hypertension, Alprostadil, Milrinone, Pulmonary vascular resistance, Arginine vasopressin, Systemic vascular resistance, Levosimendan.
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) -14mmHg, 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. 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 1119 dyn-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 administered with 0.5U (ICU) 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 1119 dyn-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 vasodilation 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 vasodilation 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 vasodilation. 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 nebulsation 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 aerosolized 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]. Inhalation of milrinone 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].

Discussion

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.

References


Conflict of Interest: Nil
Source of Support: None

How to Cite this Article