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ANESTHETIC MANAGEMENT OF A PATIENT WITH COMPLETE AVSD UNDERGOING EMERGENCY CAESAREAN SECTION: A CASE REPORT

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ABSTRACT
A 21 years old primigravida at 37^{+4} weeks of gestation with complete AVSD was planned for emergency caesarean section. She had single atrium with inlet VSD (Size 9.4 mm), moderate to severe MR, moderate TR with severe PAH. We report the case of a patient with complete AVSD who underwent emergency caesarean section under epidural anesthesia with careful hemodynamic monitoring. Thorough surveillance and effort could help to make the surgery successful.

KEYWORDS: Emergency caesarean section; complete AVSD; Pulmonary arterial hypertension, Epidural anesthesia.

INTRODUCTION
Atrioventricular septal defect (AVSD) constitute 4% of all congenital cardiac malformations.[1] It is also called endocardial cushion defect and results from failure of endocardial cushions to fuse with lower part of atrial and upper portion of ventricular septum. In addition, atrioventricular valves will also be abnormal. Atrioventricular septal defect (AVSD) can be partial, intermediate, or complete.[1]

Left to right shunting and regurgitation leads to volume loading of both atria and both ventricles.[2] Patient will develops pulmonary congestion and pulmonary hypertension.[1] Life expectancy varies according to the type of disease. Patients with complete AVSD rarely survive for decades without surgical treatment. Surgery usually performed at the age of 2-5 years and in some cases earlier. Pulmonary stenosis reduces pulmonary blood flow and protects the lungs from irreversible vascular changes.[3] Patients with complex congenital cardiac anomalies and pulmonary stenosis might have balanced pulmonary and systemic circulations and thus live life with minimal or no symptoms.

The principle of any anesthetic technique chosen is to maintain systemic vascular resistance (SVR), avoiding its fall or increase in pulmonary vascular resistance (PVR).[1]

CASE REPORT
A 21 years old primigravida at 37^{+4} weeks of gestation weighing 50 kg, a case of complete AVSD with moderate to severe MR, moderate TR and severe pulmonary hypertension was planned for emergency LSCS.

Preoperatively, she had dyspnea , cough with frothy blood mixed sputum. Physical examination revealed pulse rate of 80/min, blood pressure (BP) of 100/70 mmHg and oxygen saturation (SpO2) of 90% with oxygen via facemask at 8L/min with no rise in jugular venous pressure.

Auscultation of bilateral lung field revealed bilateral basal crepitations, and cardiac examination showed regular rate and rhythm with loud P2 and pansystolic murmur over the apex. Electrocardiography (ECG) showed normal rhythm.

Two-dimensional echo color Doppler revealed a single atrium with inlet VSD (Size 9.4mm) with bidirectional shunting, single AV annulus with single AV valve opening into RV and LV, moderate to severe MR, moderate tricuspid regurgitation with severe pulmonary hypertension.

Hemoglobin (Hb) was 15.6 gm/dl, and platelet count of 200,000/mm3.The cardiologist advised to undergo an emergency caesarean section under high risk. Peripheral venous access was secured. Meticulous attention was paid to the avoidance of bubbles in lines and syringes because of the risk of paradoxical embolus. She was premedicated with ranitidine 150 mg and metoclopramide 10 mg intravenous 20 min prior to surgery. In the operation theatre, standard monitors — ECG leads, noninvasive BP, oxygen saturation probe were connected. Oxygen via simple face mask at 8 L/min was supplemented. The baseline BP and heart rate were 110/60 mmHg and 82/min respectively.

Under all aseptic precautions, an epidural catheter was inserted at the L3-L4 intervertebral space. Radial artery cannulation was done on left wrist. Central line was placed in right(internal jugular vein) IJV catheter. Incremental doses of 3-5 mL of 0.5% Ropivacaine was administered every 5 min via epidural catheter, and a sensory block to the level of T6 was achieved with 17 mL Ropivacaine over 30 min. Adequate analgesia was achieved.

One episode of hypotension developed intra-operatively. Infusion of Dopamine was started at 5 microgram/kg/minute and tapered to maintain MAP of 65 mm of Hg. Dopamine infusion was stopped 20 minutes before surgery completion after attending stable hemodynamics. No further top up doses of epidural was required.

A live female baby with Apgar score of 7 at 1 min and 8 at 5 min was extracted. Intravenous infusion of oxytocin 20 units was administered slowly over 30 min. A volume of 500 mL Ringer's solution was administered during the 50 min surgery. Estimated blood loss was 600 mL. Urine volume was 200 mL. Patient was shifted to the maternal intensive care unit for hemodynamic monitoring. She was shifted to ward on 3rd postoperative day and discharged on 5th postoperative day.

DISCUSSION

In longstanding uncorrected AVSD , there is a reversal of a left to right shunt occurring due to high right-sided heart pressure as compared to the left side leading to Eisenmenger's syndrome.[1] Pregnancy-induced systemic vasodilation is detrimental in parturients with Eisenmenger's syndrome. Reduced SVR may increase right-to-left shunting and decrease pulmonary blood flow, leading to further hypoxemia with significant risks for both mother and fetus. Anesthetic management herein requires balancing SVR and PVR.

In this patient, the AVSD likely resulted in Right heart failure and pulmonary venous hypertension resulting from irreversible vascular changes and increasing pulmonary vascular resistance (PVR) developed as the shunt progressed so the patient had already been experiencing exertional dyspnea, pulmonary hypertension, and right heart failure at the time of her presentation.

Pulmonary hypertension can be classified as mild (36‑49 mmHg), moderate (50‑59 mmHg) and severe (>60 mmHg) with right ventricular systolic pressure values measured using echocardiography. In cases in which pulmonary artery pressure increases further during surgery due to increased PVR or other factors, pulmonary hypertensive crisis can result, which may lead to decreased cardiac output and hypoxemia.

Therefore, the goal of anesthetic management in patients with pulmonary hypertension is to lower PVR and maintain systemic vascular resistance (SVR). To prevent an increase in PVR, acidosis should be corrected, hypoventilation should be avoided, sympathetic nervous system stimulation should be avoided, normothermia should be maintained, and intrathoracic pressure should be minimized.

Regional and general anesthesia have been used. Inadvertent hypotension can occur with both techniques. The problems of general anesthesia are decrease in venous return and cardiac output. Many induction and maintenance agents depress myocardial function and reduce SVR. The hazards of general anesthesia are avoided by regional anesthesia, although the level of block required using a regional technique might produce excessive sympathetic block and an uncontrolled decrease in the SVR.

Argus eyed monitoring forms the mainstay of intra-operative management. We used invasive arterial BP and central venous pressure (CVP) monitoring.

Fluid management is a double edged sword. We provided adequate uterine tilt, monitored the urine output and dehydration status to judiciously administer fluids. Oxygen is a pulmonary vasodilator decreasing the blood flow across the right-to-left shunt thereby improving the saturation. So it should be provided throughout the perioperative period. At the end of the procedure with oxygen supplementation, our patient achieved a saturation of 98%. Thromboembolism prophylaxis should be encouraged by early ambulation and if prolonged immobilization is anticipated subcutaneous administration of heparin should be given.

The anesthesia technique which has been commonly used in this surgery is spinal anesthesia. However, this technique carries risk of sudden and uncontrolled surges in hemodynamic with possibility of reversal of intracardiac shunt. Also anticipated problems during general anesthesia in these patients are air embolism during vascular access, heart block, dysrhythmias (5–10%), heart failure and infective endocarditis. We employed regional anesthesia approach with epidural anesthesia.

Supplemental oxygen via simple face mask was administered as it would be beneficial in severe PAH. The use of epidural permitted us to have better hemodynamic stability and avoiding sympathetic stimulus as may occur during intubation and extubation in GA resulting in detrimental effects on SVR and PVR. Intraoperatively, all factors were avoided which could cause reversal of shunt or increase shunt fraction. We maintained adequate preload and cardiac contractility, near normal heart rate and SVR and PVR with the use of adequate monitoring. In addition, we carefully monitored the risk of introduction of air into the patient's IV lines to prevent paradoxical air embolism. Postoperatively, as pain could cause dramatic increase in SVR, adequate analgesia was maintained with epidural and it provides better perioperative hemodynamic stability with good analgesia.

CONCLUSIONS

With good preoperative assessment, proper preparation and providing good intraoperative and postoperative analgesia non cardiac surgeries can be easily performed under regional anesthesia in patients of AVSD with severe pulmonary hypertension.

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