Case Report

Anaesthetic management of a patient with Atrial septal defect for total abdominal hysterectomy

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Abstract

Atrial septal defect is a type of congenital acyanotic heart disease. It accounts for 9% of congenital cardiac defects in adults. We discuss a case of large ASD with moderate pulmonary hypertension who underwent elective total abdominal hysterectomy under general with epidural anaesthesia.

Key Word: Atrial septal, abdominal hysterectomy.

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CASE REPORT

A 41 year old female, 60 kg was posted for elective total abdominal hysterectomy. Patient was P3L3, all apparently being uncomplicated hospital deliveries. On preanaesthetic evaluation, patient gave history of dyspnoea on exertion [NYHA Grade II]. No history of cyanosis, recurrent respiratory tract infections and fever. No history of palpitations, syncope and chest pain. No history of pedal oedema, paroxysmal nocturnal dyspnoea.

On examination, baseline pulse rate was 88/min, regular and good volume. Blood pressure 140/90 mmHg. no pallor, icterus and clubbing. Jugular venous pressure was normal. Cardiovascular examination revealed a loud P2 and systolic murmur. Respiratory system examination showed air entry bilaterally equal and no adventitious breath sounds.No palpable spleen. All haematological investigation were normal (Hb: 11.4gm%, TLC:6640per mm^3 and platelets: 2,15,000/mm^3). ECG revealed P-pulmonale and RBBB. Chest X-Ray showed prominent pulmonary vessels with cardiomegaly. 2dECHO revealed dilated RA and RV, moderate pulmonary hypertension and Mild Tricuspid regurgitation. TEE further showed large 11 mm SVC type of Sinus Venosos Ostium Secundum ASD with left to right shunt. Ejection fraction was 55 %. Patient was shifted to ICU 12 hours prior to surgery where right sided internal jugular vein was cannulated using 16 G central venous catheter by Seldinger's technique. Post cannulation X-Ray showed that central line was properly placed and no pneumothorax. CVP was 9 cm of H20. Patient was preloaded with 500 ml Ringer's Lactate. 18 G epidural catheter was placed in L1-L2 intervertebral space with loss of resistance to saline technique. Radial artery was cannulated for invasive BP monitoring. Patient was premeditated with Inj. Midazolam 2 mg, Inj. Glycopyerrolate 0.2mg and Inj fentanyl 100 µg. She was preoxygenated with 100 % oxygen. Induction was performed with Inj. Etomidate 12mg and Inj. vecuronium6mg. The patient was intubated with cuffed endotracheal tube number 7.5. Anaesthesia was maintained using 100% oxygen, sevoflurane and boluses of Inj Vecuronium 1mg every 30 minutes. Epidural anaesthesia was supplemented with Inj. Ropivacaine 0.125% 8ml and Inj. Fentanyl 10 µg through epidural catheter as premtive analgesia. Intraoperative monitoring showed pulse: 79-96/min, IBP: 96/72 to 156/82 mmHg, capnometry:32 to 36 mmHg and spO2 : 100%. Central venous pressure was maintained at 10 cm of water. Arterial blood gas analysis was normal. Intraoperative, patient received 1500 ml Ringer Lactate and inj. Fentanyl 20 µg. The residual neuromuscular blockade was reversed with Inj. Neostigmine 2.5 mg and Inj. Glycopyerrolate 0.5mg. After extubation, patient was conscious responding to commands with pulse rate 100/min, IBP: 154/76 mmHg and 100% spO2. The patient was observed for 24 hours postoperatively where she received face

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mask Oxygen at 4 L/min.No supraventricuar dysarrytmias and AV conduction disturbance seen.Analgesia was maintained with epidural boluses of Inj. Ropivacaine 0.125% 8ml every 8 hourly for 48 hours.

DISCUSSION

Atrial septal defect is a defect in the interatrial septum.it is an intermediate complexity congenital heart disease.[2] It can be classified onto following 3 types depending on the location of defect:-

1. sinus venosus ASD: high in interatrial septum near the entry of the superior vena cava into the right atrium.
2. ostium primum ASD: adjacent to the atrioventricular valves
3. ostium secundum ASD: involves the fossa ovalis, mid-septal in location.

In ASD, blood is shunted from left atrium to the right atrium and then to right ventricle increasing the right ventricular output and pulmonary blood flow. Thus leading to enlargement of right atrium, right ventricle and pulmonary arteries. There is reversal of shunt on development of pulmonary hypertension.

The magnitude of the left to right shunt depends on the ASD size, ventricular diastolic properties and the relative impedance in the pulmonary and systemic circulation.[2] Patients with ASD are mostly asymptomatic in early life. However some patients may have increased tendency of respiratory infections or physical underdevelopment. Cardiorespiratory symptoms occur later in life. Beyond 4th decade, patients may show atrial arrhythmias, pulmonary hypertension or right heart failure. Chest radiography shows prominent pulmonary artery and increased pulmonary vascular markings. Echocardiograph shows Right Bundle Branch Block and axis deviation (right: ostium secundum and left: ostium primum). Echocardiogram and transoesophageal echocardiography gives the details of size and location of the ASD, magnitude and hemodynamicimpact of the left to right shunt, and the presence and the degree of pulmonary hypertension.[3,4] Operative repair is advised for all patients with uncomplicated secundum ASD with pulmonary to systolic flow ratios ≥1.5:1. In ostium primum ASD, cleft mitral valves may require repair in addition to patch closure of ASD.[2] The complications anticipated during general anaesthesia in these patients are air embolism during vascular access, heart block, dysrhythmias (5%-10%), heart failure and infective endocarditis.[5,6] Infective endocarditis prophylaxis is advised if ASD is associated with valvular disease. Pulmonary arterial hypertension is defined as mean PAP greater than 25 mm Hg or greater than 30 mm Hg with exercise. Preload should be maintained in these patients. So hypovolemia whether primary (from blood loss) or secondary (from vasodilation) should be immediately managed. Sympathetic stimulation (eg. Light plane of anaesthesia, pain), acidaemia, hypoxia, hypercapnia, hypothermia and increased intrathorasic pressure (atelectasis, positive end expiratory pressure) should be avoided. Pulmonary vascular resistance can be decreased by increasing Pa02, hypocapnia, alkalemia, minimizing intrathorrasic pressure and pharmacological methods like isoproterenol and phophodiesterase III inhibitors. In our patient, we proffered general anaesthesia with epidural anaesthesia as it provides better haemodynamicstability and post operative analgesia. Goals during management of our patient were toavoid factors that can increase the shunt, maintained adequate preload and cardiac contractility, a near normal heart rate, SVR and PVR. Precautions were taken to avoid air embolism during vascular access. Arterial blood gas analysis was done to check for hypercapnia. Nitrous oxide was avoided because of a risk of paradoxical air embolism. High FiO2decreases pulmonary vascular resistance and increases pulmonary blood flow and left to right shunt. Sevoflurane was used for its cardioprotective features.

CONCLUSION

Proper preoperative evaluation and good intraoperative and postoperative analgesiasurgical procedures may be undertaken in such patients with greatly reduced risk.

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