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Case Report

Anaesthetic Management of a Patient with Large Atrial Septal Defect with Severe Pulmonary Hypertension and Moderate Tricuspid Regurgitation for Total Nephrectomy: A Case Report

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Abstract

A 65 year old male patient posted for left nephrectomy was diagnosed to have large ASD with severe pulmonary arterial hypertension (PAH) and moderate tricuspid regurgitation (TR). We report the successful management of this case using general anaesthesia (GA) along with epidural analgesia. Our main objective during GA was to prevent untoward effects like fluid overload, hypotension, hypoxemia, hypercarbia leading to reversal of shunt. Careful monitoring and vigilant care during the perioperative period led to an uneventful recovery and discharge on the 6th postoperative day.

Keywords: *Atrial Septal Defect; Nephrectomy; Severe PAH; General Anaesthesia; Epidural Analgesia*

Introduction

Atrial Septal Defect (ASD) is the most common congenital acyanotic cardiac anomaly seen in grown up Congenital Heart Diseases (GUCHD). ASD has three type of defects namely osteum primum, osteum secundum and sinus venosus. The osteum secundum is most common and accounts for 70% of cases with ASD with a male to female ratio of 1:2 [1]. Pulmonary arterial hypertension (PAH) is defined as a mean pulmonary arterial pressure greater than 25 mmHg at rest or greater than 30 mmHg during exercise [2]. ASD causes left to right intracardiac shunt resulting in right ventricular volume overload and hypertrophy, increased pulmonary blood flow with pulmonary hypertension. Complications of uncorrected ASD include severe PAH, right sided heart failure, atrial fibrillation and Eisenmenger's syndrome [3]. Changes in systemic vascular resistance have important implications in the perioperative period in patients with ASD. We present a detailed management of a patient with large ASD, severe PAH and moderate TR who underwent left sided nephrectomy under general anaesthesia combined with epidural analgesia.

Case Report

A 65 year old male, weighing 35 kgs, presented with bilateral renal parenchymal disease, left poor functioning kidney with mid-ureteric stones since two months. The patient was posted for left nephrectomy after four cycles of haemodialysis.

Patient had a history of recurrent respiratory tract infection and exertional dyspnea NYHA grade II since 10 years and a history of palpitations and occasional chest pain since 5 months. On examination, heart rate was 160/minute, irregular, blood pressure was 130/90

mmHg, oxygen saturation was 98% on room air and jugular venous pressure was raised. Respiratory system examination revealed crepitations in lower zones of both lungs. CVS examination revealed loud P_2, Pansystolic murmur grade IV/VI in pulmonary and mitral area.

Biochemical and hematological investigations showed Hb 9.3 gm/dl, TLC count 18,400/cumm and Serum Creatinine 1.55 mg/dl following four episodes of haemodialysis while other tests were within normal limits. Chest X-ray revealed prominent central pulmonary vessels, cardiomegaly and multiple calcifications while ECG revealed a Right Bundle Branch Block. 2D Echo showed grossly dilated right atrium and ventricle, ASD (17 mm) with left to right shunt, moderate TR, severe PAH with an estimated right ventricular systolic pressure of 60 mmHg and left ventricular ejection fraction of 60%. Pulmonary function tests revealed FEV_1 35% with severe restrictive disease and insignificant improvement in functions following bronchodilator therapy.

Anaesthetic management

A thorough pre anaesthetic check-up was done and high risk written informed consent was obtained. Patient was kept nil orally for 8 hours before surgery. Venous access was secured with an 18 G cannula and Ringer Lactate was started as maintenance fluid in the pre-operative hold area. Heart rate, non-invasive BP, ECG, SpO_2, and end tidal CO_2 were noted pre-operatively as baseline and monitored throughout the perioperative period.

Preoperatively, Epidural catheter was inserted in L2-L3 epidural space after confirming loss of resistance by saline technique under strict aseptic precautions for intraoperative and postoperative analgesia. Patient was premedicated with glycopyrrolate 4 mcg/kg, ondansetron 0.1 mg/kg and Fentanyl 2 mcg/kg following preoxygenation for 3 - 5 minutes with 100% oxygen and anaesthesia was induced with Etomidate 0.25 mg/kg and Atracurium 0.5 mg/kg. Endotracheal intubation was done orally with 8.0 mm cuffed endotracheal tube and anaesthesia was maintained with air and oxygen (50:50), sevoflurane, atracurium (0.1 mg/kg) and controlled ventilation. Normocarbia was maintained with $ETCO_2$ of 32 - 35 mmHg.

Intraoperatively, persistent tachycardia (HR varied between 130-170 beats/min) and hypotension (NIBP varied between 90/60-70/50 mmHg) was present. Therefore, Inj. Noradrenaline infusion was started @ 0.5 - 1 mcg/kg/min to maintain BP at baseline values. The epidural catheter was dosed with Ropivacaine (0.125%)in 12 ml doses hourly. Inj metoprolol 2 mg was given to control tachycardia. Meanwhile, ECG showed an irregularly irregular rhythm suggestive of atrial fibrillation (AF). AF was reverted with a single dose of Amiodarone 150 mg IV over 10 minutes, BP was also well maintained with the Norepinephrine infusion. Total fluid given intraoperatively was 1500 ml and urine output was 300 ml after 2 hours of surgery. Neostigmine and glycopyrrolate were given for reversal of neuromuscular block following completion of surgery.

Postoperatively, patient was conscious, responding to verbal commands, muscle power was adequate and BP was stable on Noradrenaline infusion after extubation. Patient was shifted to ICU for monitoring where ECG showed episodes of atrial fibrillation. On advice of Cardiologist, Tab Digoxin 0.25 mg stat and Tab. Sildenafil 20 mg twice a day was started and continued for 3 days.

Noradrenaline infusion was tapered and stopped after 4 hours of surgery when BP was maintained within normal limits. Postoperatively, analgesia was maintained with epidural top - up of 0.125% Ropivacaine (12 ml) and 50 mcg fentanyl 8 hourly along with Inj Paracetamol 1000 mg/6 hourly for 48 hours. ECG showed regular rhythm a day after surgery with a HR of 80 - 90 beats/min. Patient had an uneventful recovery and was discharged on 6th postoperative day.

Discussion

Atrial Septal Defect (ASD) accounts for 6% - 10% of all congenital heart diseases [4]. ASD is progressive in nature, usually asymptomatic in childhood but symptoms appear during later ages due to reversal of shunt. Untreated patients can develop symptoms like exertional dyspnea, fatigue, palpitations, and arrhythmias [5].

Pulmonary hypertension is classified as mild (36 - 49 mmHg), moderate (50 - 59 mmHg) and severe (> 60 mmHg) based on the estimated right ventricular systolic pressure calculated by echocardiography [6]. Pulmonary hypertensive crises is defined as a sudden increase in PVR that results in pulmonary pressure exceeding mean arterial pressure, compromising cardiac output and producing hypoxemia [7].

The common problems which can be encountered during general anaesthesia (GA) for these patients include paradoxical air embolism during the vascular access, heart block, dysrhythmias, heart failure and infective endocarditis. Shunting has an important effect on anesthetic management. All intravenous lines must be meticulously deaired to decrease the risk of systemic air embolization in patients with intracardiac shunts. Inadequate anesthesia and sympathetic nervous system stimulation might increase SVR, exacerbate left to right shunting, and reduce systemic cardiac output in a patient with a large atrial septal defect.

An abrupt rise in PVR may precipitate acute right ventricular failure causing decreased cardiac output, progressing to severe bradycardia and cardiac arrest. Pulmonary hypertensive crises can be avoided by hyperventilation, avoid sympathetic stimulation, maintain normothermia, and minimize intrathoracic pressure. Inhaled nitric oxide should be available in the operating room for use in high risk patients requiring treatment for drastic rise in PVR [8].

GA provides better hemodynamic stability and epidural catheter provides adequate analgesia during intraoperative and postoperative period. Hypercarbia can be avoided by mechanical ventilation and adequate adjustment of tidal volume and respiratory rate. Inflation of lungs with intermittent positive pressure ventilation (IPPV) leads to release of endogenous nitric oxide and prostaglandins, which causes pulmonary vasodilatation [9]. Our primary goal during GA was to minimize increases in PVR, maintain systemic vascular resistance (SVR), maintain optimum cardiac contractility along with close monitoring to detect atrial arrhythmias and avoid paradoxical air embolism as these changes may lead to hypoxemia, cyanosis and myocardial ischemia.

Intravenous agents used for the induction of anaesthesia may depress myocardial contractility and decrease SVR that can lead to adverse effect on tissue oxygen delivery during induction of anaesthesia. We used injection Etomidate as induction agent for better hemodynamic stability. Intraoperatively, we managed hypotension with Noradrenaline infusion and tachycardia was managed with adequate analgesia and IV Metoprolol. Nitrous oxide may increase PVR and may increase the size of air bubble so we avoided the use of nitrous oxide.

Atrial and ventricular dysrhythmias are common in adults with CHD. The most common form of tachyarrhythmia observed is intraatrial reentrant tachycardia. Atrial tachyarrhythmias are often resistant to pharmacological treatment and can result in rapid hemodynamic deterioration. Ventricular dysrhythmias are most frequently encountered in patients who have significantly decreased right or left ventricular function. Some patients require a permanent pacemaker to treat bradycardia secondary to postoperative atrioventricular block [10]. In our case, Atrial Fibrillation occurred intraoperatively which was managed by a single bolus dose of Amiodarone injection.

Invasive arterial blood pressure monitoring can be essential in patients with intracardiac or systemic to pulmonary shunts, undergoing major surgery and who are susceptible to sudden changes in SVR/PVR. Transesophageal echocardiography may be useful in such patients for the monitoring of intravascular volume status and ventricular function [11]. Preoperatively, 2D Echo was done to evaluate the size of ASD, grading of PAH and ventricular function Intraoperatively, we monitored the patient with SpO_2, non-invasive blood pressure monitor, ECG, and $EtCO_2$ monitoring to avoid pulmonary hypertensive crises and subsequent hypoxemia, hypothermia, hypercarbia.

Conclusion

Patients with large ASD and PAH can undergo non-cardiac surgery by a detailed preoperative assessment, balanced intraoperative anaesthetic management, intraoperative and postoperative analgesia to avoid changes in SVR/PVR, tachycardia, hypotension, hypercarbia, hypoxemia and hypothermia.

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