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A CASE REPORT : ANAESTHETIC MANAGEMENT OF EISENMENGER SYNDROME FOR NON-CARDIAC SURGERY.

Anaesthesiology

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ABSTRACT

Background: Eisenmenger syndrome is one of leading cause of perioperative death in patient undergoing non cardiac surgery which is upto 19%. With improved management of congenital cardiac disease, Eisenmenger's syndrome is becoming very rare. However, these patients occasionally present for incidental non-cardiac surgery. Anaesthetic management of patient with shunting lesion undergoing non cardiac surgery varies with severity of the lesion.

Case Description: We describe a case of a 23year old female presenting with history of dyspnea NYHA grade II with pallor (+1), grade II clubbing, pansystolic murmur with X ray and 2D-ECHO finding suggestive of Eisenmenger syndrome with ventricular septal defect of 2.5mm with bi directional shunt and mild Pulmonary arterial hypertension. We describe anaesthesia considerations and management of patient of this syndrome posted for open cholecystectomy under general anaesthesia.

Conclusion: Patient with Eisenmenger syndrome for non cardiac surgery can be managed with meticulous preanesthetic evaluation, vigilant perioperative monitoring and definitive anaesthetic management with special intraoperative considerations to prevent the risk of paradoxical air embolism, myocardial depression, sudden hemodynamic changes and maintaining adequate arterial blood pressure at the same time.

KEYWORDS

Eisenmenger syndrome, Right to left shunt, General anaesthesia, Cholecystectomy.

INTRODUCTION

Eisenmenger syndrome (ES) is a constellation of symptoms that arise from a congenital heart defect and result in large anatomical shunts. Initially there is left-right shunt, which develops into severe pulmonary arterial hypertension (PAH) and elevated vascular resistance and ultimately left-to-right shunt becomes right-to-left shunt, resulting in significant hypoxemia and cyanosis. In 1897 Victor Eisenmenger coined the term Eisenmenger complex, which included large ventricular septal defect and pulmonary hypertension.[1] Wood redefined this in 1958 as pulmonary hypertension with reversed or bidirectional shunt associated with septal defects or patent ductus arteriosus.[2] With improved management of congenital cardiac disease, Eisenmenger's syndrome is becoming very rare. However, these patients occasionally present for incidental non-cardiac surgery. We describe the successful management of a patient with this syndrome presenting for open cholecystectomy.

Case Description

A 23year old female weighing 53 kilograms scheduled for elective open cholecystectomy was referred for pre-anaesthesia evaluation. She had a history of dyspnea grade II for last 2 months and was not on any treatment. On general physical examination pallor (+1) and grade II clubbing were present with no signs of central and peripheral cyanosis, pedal edema, raised jugular venous pressure. Blood pressure was 110/68 mmHg right arm supine position, pulse rate 80 beats per minute, regular, low volume with no radio-radial and radio-femoral delay. Respiratory rate was 18 per minute with 94% saturation on room air.

CNS was normal. CVS examination- Auscultation revealed a pansystolic murmur in the left lower sternal border. Respiratory system- normal vesicular breath sounds were audible Complete blood counts, renal and liver function tests were within normal limits. Chest X-ray (Fig:1) suggestive of Boot shaped heart (s/o Congenital Heart disease). ECG showed sinus rhythm, enlarge 'p' wave with right axis deviation.2-D ECHO with colour doppler was done after Cardiology opinion which showed small ventricle septal defect of 2.5mm diameter with bidirectional shunt and a good left ventricular function with ejection fraction 55% and mild pulmonary arterial hypertension. Mild Tricuspid valve regurgitation was present. Aortic and Mitral valves function appeared normal. She was diagnosed as CHD with Left to Right shunt , NHYA Grade 2, in Sinus rhythm, without Cyanosis. Patient was accepted under ASA grade II for surgery. Airway examination- MP class 1. Patient was re-examined an evening prior to surgery, appropriate consent was obtained and advised to be kept nil per oral overnight. Tab. ranitidine 150 mg was prescribed at night and 6 AM morning.

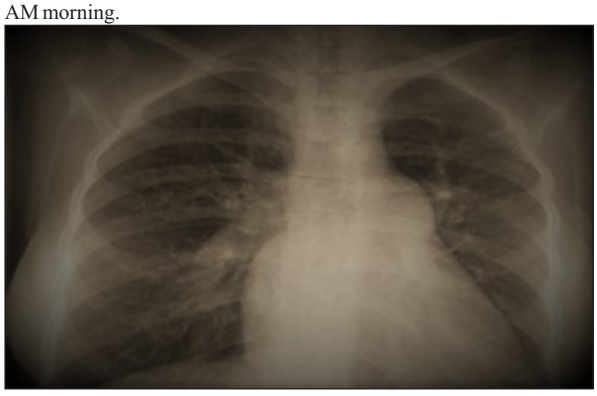

Fig 1: Chest X-ray

On the day of surgery, after written informed consent for surgery and anaesthesia was obtained, she received infective endocarditis prophylaxis with injection ampicillin 2g intravenous 30 minutes prior to surgery. Patient was taken on operation table, standard ASA monitors were attached (ECG, SPO2, NIBP) intravenous cannulation was done with 18 guage after free flow of blood was observed and connected to the ringer lactate tubing in order to prevent any air bubble entry into the intravenous set. Premedication was started with intravenous injection glycopyrrolate 0.2 mg, Morphine 4mg and Midazolam 2mg. Induction was done with injection 1.5mg/kg Ketamine and 0.1 mg/kg vecuronium muscle relaxant used then patient was ventilated with bag and mask (IPPV) and patient was intubated with appropriate sized endotracheal tube. Anaesthesia was

maintained with 40 % oxygen and 60 % air with inhalational agent Isoflurane. Intraoperative vitals were- Spo2 98% on 100% oxygen, blood pressure ranged between 110-100/80-60 mmHg, heart rate 80 – 90 beats per minutes. Intraoperative 800ml crystalloid for fluid replacement and injection paracetamol 1 gram for analgesia were given. Intraoperative continuous ECG monitoring (lead II and V) was done. Perioperative period was uneventful and patient was reversed with neostigmine and glycopyrrolate. Patient was extubated successfully and total duration of anaesthesia was 40 minutes. Minimal blood loss was observed. Post Operative period was unremarkable.

DISCUSSION

Eisenmenger syndrome is one of leading cause of perioperative death in patient undergoing non cardiac surgery which is up to 19%.[3] Anaesthetic management of patient with shunting lesion undergoing non cardiac surgery varies with severity of the lesion. Severity of cyanosis, right ventricle dysfunction and tricuspid regurgitation plays an important role in determining perioperative risk. It would be reasonable to assume that in this condition the pulmonary vascular resistance would remain relatively constant. However, normal changes in the systemic vascular resistance would be expected to occur. Attention should be paid to minimizing any alterations in peripheral vascular resistance and maintaining arterial blood pressure. The anaesthetic technique aimed to avoid a fall in arterial blood pressure by maintaining cardiac output and systemic vascular resistance[4]. Care should be taken to avoid air bubbles in all iv lines to avoid paradoxical emboli (migration of air from right to left heart via inter septal defect) formation which can lead to heart attack and strokes. Goal of monitoring is to detect and prevent sudden hemodynamic changes and to prevent further complications. Drugs used for induction and maintenance of general anaesthesia causes myocardial depression and decreases systemic vascular resistance. We used Morphine because it causes minimal cardiac depression with no sensitization of the heart to cathecholamines and no interference with autonomic or cardiovascular drug action. We used ketamine as an induction agent as it does not reduce systemic vascular resistance. Ketamine has been recommended as the drug of choice for induction because it has little effect on pulmonary, but moderately increases systemic vascular resistance for short duration in the dosage used. Whereas thiopentone and propofol decrease systemic vascular resistance.[3] Vecuronium is devoid of circulatory effects like histamine release association with blood pressure changes have been reported after administration of atracurium. Morphine increases heart rate and Vecuronium known to decrease heat rate, combination helps to stabilize the HR. Inhaled nitrous oxide was avoided as induces pulmonary vasculature constriction and may significantly worsen pre-existing pulmonary hypertension, and expansion of air in case of any event of air embolism. Isoflurane was used because it preserves cardiac output as the result of an active carotid baroreceptor reflex and decrease afterload. Prolong fasting and volume depletion should be avoided before surgery and alpha adrenergic agonist Ephedrine or Mephenteramine should be used in case of systemic arterial hypotension and hypovolemia should be treated with intravenous fluid replacement.[5] Pain causes stress and will increase SVR, PVR and oxygen requirements of the myocardium and this must be avoided in Eisenmenger's syndrome as the right heart is constantly under strain and at risk of decompensation.

CONCLUSION

Safe anaesthetic management of patients with Eisenmenger's syndrome requires meticulous preanaesthetic evaluation, vigilant perioperative monitoring and a definitive anaesthetic management with special intraoperative goals to prevent risk of paradoxical air embolism, myocardial depression, sudden hemodynamic changes and maintaining adequate arterial blood pressure at the same time. Familiarity with all anaesthetic drugs and inhalational agents to maintain cardiovascular stability and its effect on pulmonary and systemic vasculature is of utmost importance. Adequate planning and implementation of above said principles had helped us in successful anaesthetic management of this rare case.

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