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Total intravenous anaesthesia in a patient with eisenmenger syndrome: Case report

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ABSTRACT

Eisenmenger's syndrome is defined as the process in which a left-to-right shunt caused by a congenital heart defect causes increased flow through the pulmonary vasculature, causing pulmonary hypertension which causes increased pressures in the right side of the heart and reversal of the shunt into a right-to-left shunt. It can occur with complex congenital cardiac malformations, septal defects, and patent ductus arteriosus (PDA). Patients with Eisenmenger's syndrome are at high risk for peripartum morbidity and mortality[1]. We report the administration of intravenous ketamine with use of upper extremity pulse oximetry in a patient with Eisenmenger's syndrome secondary to a VSD undergoing endometrial curettage.

KEYWORDS

CASE REPORT

A 23-yr-old woman, gravida 1, para 0 with incomplete abortion was admitted to our hospital with chief complaint of intermittant bleeding per vagina. She had a recurrent history of dyspnoea on exertion and episodes of cyanosis since 6 yr of age. Her activity of daily life was normal, and she was evaluated as New York Heart Association (NYHA) class I.

Patient was on treatment on tab diltiezem and tab digoxin since 10 year of age which she had left one year back. Her weight was 54 kg and her height was 154 cm. Blood pressure was 140/90 mm Hg and heart rate was 102 beats /min. Lungs were clear. Peripheral cyanosis with clubbing in both upper and lower limbs was present. Maternal hemoglobin concentration was 11.0 g/dl and hematocrit was 43%. Blood chemistry and coagulation studies were within normal limits.

Echocardiogram showed severe right atrial and right ventricular enlargement, pulmonic regurgitation, a large muscular defect VSD (21 mm), ejection fraction 69% and stoke volume 40 ml. The lesion was considered inoperable because of severe pulmonary hypertension.

An endometrial curettage was planned by the obstetrician. Monitoring included an electrocardiogram, pulse oximeter probe on the right hand and blood pressure. Infusion line was taken by 18 G intracath, special precaution was taken to avoid any air entrapment, to avoid paradoxical air embolism. 500 ml of ringer lactate was started. While breathing room air, arterial oxygen saturation (Sao2) was 64%. Supplemental oxygen (8 L/min by high oxygen mask) was given to patient which increased the Sao2 to 86%.

Patient is premeditated with glycopyrolate 0.2 mg. No preoperative sedation was given. Initial hemodynamic measurements, obtained while the patient was receiving 8 l/min of supplemental oxygen via face mask, included an arterial blood pressure of 130/90mm Hg, heart rate of 103 beats/ min, respiratory rate of 28 breaths/min, Spo2 of 86%.

Patient was kept on high oxygen flow through bain circuit and Ketamine 30 mg IV was given which was later on maintained with two additional doses of 10 mg. Operative time was around 15 minutes. 400 ml of crystalloid was given. Patient intraoperative hemodynamic status was stable. After then patient was shifted to post operative anaesthesia unit on high oxygen flow through venturi mask with vitals heart rate 110 beats/min, BP 140/96 mmHg and Sao2 was 90%. She had no adverse events postoperatively.

DISCUSSION

The Eisenmenger syndrome is a form of cyanotic congenital heart disease not usually amenable to corrective surgery. It is, however, compatible with leading an active life in early adulthood and due to advances in medical therapy it is not uncommon for patients with this syndrome to live to 30 years or more[2].

In general, therapy of Eisenmenger syndrome is supportive. Patients should avoid intravascular volume depletion, heavy exercise, and high altitude[3]. The hemostatic changes associated with the syndrome may lead to thromboembolic events, cerebrovascular complications, or the hyperviscosity syndrome. Occasionally, therefore anaesthetists and surgeons will be required to care for these patients when they present for incidental surgery.

In Eisenmenger syndrome there is decreased cross-sectional area of the pulmonary arteriolar bed with, usually irreversible, pulmonary hypertension. The pulmonary hypertension precludes corrective surgery as the elevated pulmonary vascular resistance persists or worsens after surgical closure of the defect. Occasionally, in patients with Eisenmenger's syndrome, the increased resistance is not fixed and an attempt to correct the underlying pulmonary- systemic connection may lead to an improved haemodynamic status[2].

In Eisenmenger's syndrome the pulmonary and systemic vascular resistances are approximately equal and the shunt is balanced. The factors which favour the development of pulmonary hypertension are hypercarbia, acidosis, hypoxia, high left atrial pressure and a high pulmonary blood flow. Although attempts to decrease the pulmonary vascular resistance in the Eisenmenger syndrome are usually unsuccessful, any fall in systemic vascular resistance will be adverse, as it increases the right-to-left shunt and thus arterial hypoxaemia. An increase in the systemic vascular resistance will increase the left-to-right shunt and pulmonary blood flow, at the expense of a further increase in pulmonary arterial blood pressure[4].

Anesthetic management of Eisenmenger's syndrome is often difficult. The anesthetic goal is to avoid hemodynamic changes that can worsen hypoxemia through an increase in right-to-left shunt. The priniciple of any anaesthetic technique chosen for a patient with Eisenmenger's syndrome is to avoid a fall in arterial blood pressure by maintaining both cardiac output and systemic vascular resistance[5]. Factors reducing cardiac output are: direct myocardial depression or loss of sympathetic drive to the heart, extreme changes in heart rate and a decrease in venous return.

Anaesthetist should be very careful in relation to infusion lines to avoid the hazards of paradoxical air embolism and by the surgeon in relation to the opening of large veins or venous sinuses with the patient in a position where vascular air entrainment is possible.

Induction of anaesthesia presents the time when a fall in systemic vascular resistance and hypotension are likely to occur. Arm-brain circulation time is short, due to the right to- left shunt, and therefore agents given intravenously will act very quickly. Barbiturates cause hypotension by a combination of reduced cardiac output as well as decreasing the tone of the systemic capacitance vessels[6].

Since pulmonary blood flow is decreased in Eisenmenger syndrome, the rate of rise of the arterial concentration of volatile anaesthetics is much decreased. Inhalational induction will therefore be slower and higher concentrations of volatile anaesthetics may be needed. Nearly all inhalational anaesthetics cause a degree of hypotension due to varying effects on SVR and myocardial contractility and thus are not advisable. Nitrous oxide was avoided because it is a potent pulmonary vasoconstrictor.

In Eisenmenger's syndrome the amount of right-

Short Communication

to-left shunt depends in part on the ratio of systemic vascular resistance (SVR) to pulmonary vascular resistance (PVR). Ketamine has advantages as it does not reduce systemic vascular resistance[7] and thus it is safe and effective anesthesia in a patient with Eisenmenger's syndrome. This was also considered safe because ketamine allows maintenance of spontaneous respiration and intact laryngopharyngeal reflexes which permit the patient to protect his own airway.

It is also well documented that the patient's pulmonary vasculatures dilate in response to oxygen. Both acute and chronic administration of oxygen may decrease PVR thus reducing right to left shunt in patients with congenital heart disease, primary pulmonary hypertension, and chronic bronchitis. Thus in our patient increase in oxygen saturation was both due to supplemental oxygen and administration of ketamine.

Aside from oxygen administration, patients may be treated with various specific medications to reduce PVR and improve oxygenation[8]. With long-term oxygen administration; the use of specific medications in patient management is generally palliative. Sildenafil and L-arginine are effective in decreasing PVR. L-arginine is converted into nitric oxide in the body which decreases PVR. Bosentan, an endothelin receptor antagonist, has been used and it decreases PVR by 25%[9]. More recently, IV epoprostenol has been successful in patients with congenital heart disease[10].

Phenylephrine, norepinephrine and metaraminol have been recommended to prevent a decrease in SVR during anaesthesia[11], but they also increase PVR and hence they were not used in our patient. Prophylactic use of vasopressors has not been recommended because of associated side effects on these patients with an already compromised cardiovascular system and an unpredictable and probably increased response to potent vasoactive drugs. However, their immediate availability must be ensured in the perioperative period[12].

Postoperatively, a rapid return to consciousness and avoidance of hypoxia, which would increase the pulmonary vascular resistance, are essential. In addition, avoidance of extreme changes in heart rate, oxygen therapy and early mobilisation is recommended. Patients with the syndrome should undergo routine follow-up at a tertiary care center that has physicians and nurses with special expertise in congenital heart disease.

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