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Anesthetic Management in Patient with Suspected Brain Abscess, Atrium Septal Defect, and Eisenmenger Syndrome

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Abstract
Introduction: Patients with congenital heart disease especially with systemic shunting between systemic and pulmonary circulation often develop pulmonary hypertension and left-to-right shunt (Eisenmenger syndrome) if left untreated. These patients are at risk of developing spontaneous brain abscess due to brain infarction caused by polycythemia, impaired immune function, and loss of lung phagocytosis. Such patients were often admitted to the emergency room with signs of increased intracranial pressure (ICP), and needed specific consideration during surgery. Case: a 31-year old female diagnosed with intracranial space occupying lesion (SOL) due to suspected brain abscess with concurrent heart defects (atrial septal defect / ASD and Eisenmenger syndrome) was consulted to the operating theatre for emergency burrhole aspiration. The surgery was performed for an hour and the postoperatively the patient was admitted to the intensive care unit (ICU). Conclusion: perioperative management of patients with brain abscess and concurrent ASD and Eisenmenger syndrome consists of preoperative management, methods of anesthesia, monitoring, and interventions to prevent the worsening of left-to-right shunt and increasing intracranial pressure. These managements consist of optimal pain management, perioperative oxygen therapy, and prevention of precipitating factor that increases left-to-right shunting.

Keywords: Neuroanesthesia, Intracranial, Space Occupying Lesion, Eisenmenger Syndrome

1. Introduction

Patients with congenital heart defects, especially heart defects with systemic and pulmonary shunts often develop pulmonary hypertension and reverse right-to-left shunts, known as Eisenmenger syndrome. Such patients have an increased risk for developing intracerebral abscesses due to infectious processes that spread hematogenically (most common from the pulmonary circulation) or trauma (Effendi, 2018).

Cyanotic congenital heart disease (CHD) has associated with more than 60% of cases of brain abscess. The incidence rate of brain abscess contributes to about 8% of intracranial masses in developing countries and 1-2%

in Western countries with varying clinical manifestations related to the size and location of the space-occupying lesion (SOL). Brain abscesses often cause increased ICP and have a significantly high morbidity and mortality rate (Atchabahian & Gupta, 2013; Bokhari & Mesfin, 2021).

Cyanotic CHD is characterized by a right-to-left intracardiac shunt that allows distribution of desaturated blood into the systemic circulation resulting in arterial hypoxemia (Raha et al., 2012). Eisenmenger syndrome is a complex pathophysiological condition that includes: (1) clinically significant cyanosis, (2) lntracardiac shunting of (ASD, ventricular septal defect / VSD, or aorticopulmonary anomaly), and (3) pulmonary hypertension due to irreversible elevation of pulmonary vascular resistance (PVR) (Arif et al., 2017). Eisenmenger syndrome occurs in more than 50% of adults with abnormal interventricular or aorticopulmonary communication, but only in 9% of patients with secundum ASD (Puri et al., 2011).

Patients with cyanotic CHD often to be at increased risk for cerebral abscess due to polycythemia-induced cerebral infarction, impaired immune function, and bypass of pulmonary phagocytosis (Hall et al., 2016). Cyanotic CHD accounts for nearly 13- 70% of all cases of brain abscess and usually, SOL with dimension >2 cm is indicated for surgical intervention (Hall et al., 2016; Raha et al., 2012). Patients with signs and symptomps of increased ICP should receive therapy to reduce ICP, and receive specific consideration when undergoing the surgery (Ozawa et al., 2018). Anesthesiologist should prevent increasing the ICP even further during preoperative, intraoperative, and postoperative periods (Pasternak & Lanier, 2018).

This case report presented a 31-year-old female diagnosed with supratentorial intracranial SOL at right frontoparietal region due to suspected brain abscess concurrent with ASD and Eisenmenger syndrome underwent burrhole aspiration.

2. Case Report

A 31-year-old female with body weight of 48 kg, diagnosed with supratentorial intracranial SOL at right frontoparietal region due to suspected brain abscess concurrent with ASD and Eisenmenger syndrome was consulted to the operating theatre for burrhole aspiration. She had history of left-sided hemiparesis, seizures, and fever. She had coexisting disease of CHD 2 years ago and history of laparotomy 3 years ago. She had received ceftriaxone 2x1gr IV, metronidazole 3x500mg IV, and gentamycin 2x100mg IV preoperatively.

Before the surgery, she was completely awake and aware with slight tachycardia and otherwise stable vital signs (blood pressure 122/87 mmHg, heart rate 110 beats per minute, respiration rate 24-28 breaths per minute, temperature 36,7^{o}C, and oxygen saturation (SaO2) of 63-65% with oxygen 3 liter per minute via binasal cannula). Her lung examination was clear without additional abnormal breath sound, and a grade II cardiac murmur at atrial region. Her abdomen was normal with cool extremities, peripheral cyanosis, clubbing finger, and left-sided hemiparesis (motoric function one over five).

Her blood lab result was unremarkable with hemoglobin 16.1 g/dL, hematocrit 48.6%, leukocytes 8,540 cells/dL, platelets 221,000, normal PT, APTT, and INR. Urea 21 mg/dL, creatinine 0.55 mg/dL, normal liver function, blood sugar and electrolytes. Her blood gas analysis was acidotic (pH 7.327) with pO2 52.2 mmHg, pCO2 29.2 mmHg, HCO3 15.4, base excess -8.4, and SaO2 81.5.

Her echocardiography was performed preoperatively with signs of ASD and right-to left shunt. Her head CT scan showed hypodense lesion at right frontoparietal region with dimension of 3x3x3 cm, with perifocal edema and minimal midline shift (<5 mm to the left side).

Anesthetic risk assessment was considered an ASA III case. General anesthesia was planned for the patient. She has fasted for 6 hours preoperative, and monitoring was performed with ECG, blood pressure, heart rate, temperature, SaO2, and arterial line. Intravenous maintenance was given Ringer Lactate 100ml/hour. Preparation of vasoconstrictor agents was done to prevent worsening left-to-right shunt. Postoperatively, patient was planned to be admitted to the ICU.

Preinduction vital signs were normal (blood pressure 118/76 mmHg, heart rate 107 beats per minute, respiration rate 24 breaths per minute, temperature 36.8^{o}C, and SaO2 65-68% with non-rebreathing mask and oxygen 10 liters per minute. The patient was positioned supine and preoxygenated with 100% oxygen. Induction achieved by administration of fentanyl 150 mcg, midazolam 5 mg, and rocuronium 50 mg. intubation was performed smoothly with a spiral ETT size 7.0 in one attempt. Anesthetic maintenance was achieved with sevoflurane 0.5-1 vol% in 50% inspired oxygen fraction.

Intraoperatively, the patient was stable vital signs of systolic blood pressure varying from 92-125 mmHg, diastolic blood pressure from 68-86 mmHg, heart rate varying 92-116 beats per minute, and SaO2 from 63-78%. The operation lasted for one hour, and her urine output was 50 ml per hour, with hemodynamic support norepinephrine 0.05-0.1 mcg/kgbw/minute. Postoperatively she regains complete consciousness with blood pressure 116/74 mmHg, heart rate 94 beats per minute, respiration 22 breaths per minute, and SaO2 68% with non-rebreathing mask and oxygen 10 liters per minute. Postoperative monitoring included vital signs, urine output, and signs of increased intracranial pressure. Postoperative analgesia was achieved with fentanyl 25 mcg/hour IV and paracetamol 1gr/6 hour IV. The patient was admitted to the ICU postoperatively.

Discussion

CHD is an acquired heart defect with varying clinical manifestations (mild to severe). Patients with mild CHD may exhibit no symptom and no abnormalities are found on clinical examination whereas in severe CHD, symptoms often appeared immediately since birth. CHD is divided into 2 groups, namely cyanotic CHD and non-cyanotic CHD, characterized by central cyanosis due to right-to-left shunts (for example tetralogy of Fallot, transposition of the great arteries, tricuspid atresia). Noncyanotic CHD were often found as a leakage of the heart septum accompanied by a left to right shunt including ventricular septal defects, atrial septal defects, or persistent patent blood vessel such as in persistent ductus arteriosus. In addition, non-cyanotic CHD was also found in ventricular outflow tract obstruction such as aortic stenosis, pulmonary stenosis and coarctation of the aorta.

Eisenmenger syndrome is a complication often occurs in non-cyanotic CHD marked by increased blood flow to the lungs. As a result, the pulmonary capillaries will react by increasing their resistance so that the pressure in the pulmonary artery and in the right ventricle may increase. If the pressure in the right ventricle exceeds the pressure in the left ventricle, an inverted shunt occurs from right-to-left so that the patient begins to be cyanotic (Djer & Madiyono, 2016). In patients with cyanotic CHD, veno-arterial blood flow in the heart causes recirculation of desaturated venous blood into the systemic circulation. This condition causes hypoxia in the systemic circulation and body tissues including the brain which may form necrotic areas that predispose to brain infection. Hypoxia also causes reactive polycythemia, increases blood viscosity and decreases cerebral blood flow which may worsen necrosis in the brain. Cerebral abscess formation in cyanotic CHD patients is also influenced by brain exposure to bacteremia often originated from pulmonary circulation due to bypassed pulmonary circulatory system. In addition, the heart defect itself predisposes to the formation of heart valve vegetation. Vegetative embolus may form which may enter the systemic circulation and embolize certain brain regions creating necrotic areas of the brain that can develop into a cerebral abscess (Ontoseno, 2004).

Anesthetic consideration in patients with cyanotic CHD begins with a thorough pre-anesthesia examination that documents their current cardiac and neurological status, previous surgeries, previous anesthetic management, complications, as well as current treatment. Consultation with the cardiology department, 2-dimensional (2D) echocardiography and assessment of the coagulation profile are required for the patient. Vomiting, fever, poor oral intake, and diuretics can lead to dehydration and a pre-existing increase in blood hyper viscosity. Prolonged preoperative fasting should be avoided and consumption of clear fluids up to 2 hours before surgery is recommended. Severe cyanotic patients with hematocrit 60% are at risk for coagulopathy, so phlebotomy can be performed before surgery (Lei, 2015).

Right-to-left shunt will cause poor pulmonary perfusion resulting in chronic hypoxemia and cyanosis. Compensatory mechanisms that follow include polycythemia, vasodilation, hyperventilation, and chronic respiratory alkalosis. Anesthesia problems that arise include perioperative hemodynamic instability, cyanotic spells, coagulation disorders due to polycythemia, paradoxical air embolism, fluid and acid-base imbalances,

congestive heart failure, prophylaxis of infective endocarditis (IE), and maintaining intracranial dynamics. Adequate hydration can help reduce increased blood viscosity, sludging and thromboembolic events.

Anesthesia management in patients with Eisenmenger syndrome undergoing noncardiac surgery is similar to anesthetic management in patients with other forms of severe pulmonary hypertension (Steppan & Maxwell, 2021). The two main principles of perioperative risk management are prevention of systemic hypotension and avoidance of increased PVR (PH crisis) (Nashat et al., 2017). By maintaining systemic vascular resistance (SVR) which is relatively higher to PVR, right-to-left shunts will be minimal. A sudden drop in oxygen saturation without a change in ventilation is considered an early sign of decreased systemic vascular resistance. Hypoxia, hypercarbia and acidosis can cause a significant increase in PVR. Therefore, hyper ventilation with 100% O2 without PEEP may reduce PVR.(Raha et al., 2012; Wajekar et al., 2015) Norepinephrine can help maintain systemic vascular resistance and epinephrine and/or vasopressin may also be used (Bennett, 2021; Lei, 2015). Ketamine as sole anesthetic induction is contraindicated in intracranial surgery and total intravenous anesthesia (TIVA) with fentanyl may be chosen for its cardiostability and short duration of action.

Early extubation can prevent an increase in PVR due to prolonged ventilation (Lei, 2015). Postoperative management includes intensive cardiac monitoring, oxygenation, appropriate analgesia, fluid management, and prophylaxis of vomiting and seizures (Raha et al., 2012; Wajekar et al., 2015). Patients should be normovolemic in the postoperative period and oral intake should be resumed as soon as possible. Pain control is essential to maintain adequate ventilation, but opioids must be administered with caution to avoid oversedation and hypoxia (Lei, 2015). Hypoventilation and hypoxemia, arrhythmias, and inadequate postoperative pain control can lead to exacerbation of the patient's shunt, which can lead to heart decompensation (Bennett, 2021).

Conclusion

The main goals of anesthetic management in patients with cerebral abscess with congenital heart disease with Eisenmenger syndrome are as follows: Maintaining adequate SVR. Good maintenance of intravascular volume, venous return, pain prevention, hypoxaemia, hypercarbia, and acidosis which can lead to an increase in pulmonary vascular resistance (PVR). Anesthesiologists should also prevent myocardial depression during general anesthesia and prevent increasing the ICP.

A well-executed general anesthetic with hemodynamic maintenance aimed at preventing the occurrence of worsening right-to-left heart shunts, and maintaining good intracranial pressure has a fairly good outcome in patients with suspected cerebral abscess with congenital heart disease ASD with Eisenmenger syndrome. Preparation for emergency events is always needed and done to reduce the morbidity and mortality of these cases.

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