ANESTHESIA FOR PATENT DUCTUS ARTERIOSUS LIGATION SURGERY IN ADULT

Kurniaji[1], Juni Kurniawaty[2]
[1]Fellowship Program of Cardiovascular Anesthesia in Department of Anesthesiology and Intensive Care, Gadjah Mada University, Indonesia
[2]Department of Anesthesiology and Intensive Care, Gadjah Mada University, Indonesia
e-mail: mediacanest@gmail.com

Abstrak
Patent Ductus Arteriosus (PDA) adalah suatu kondisi jantung bawaan yang terjadi ketika pembuluh darah normal janin yang disebut ductus arteriosus, yang menghubungkan arteri pulmonalis dan aorta di dalam rahim, gagal menutup setelah bayi lahir. Laporan kasus ini berfokus pada manajemen anestesi pada PDA dewasa yang menjalani operasi ligasi PDA. Seorang wanita berusia 33 tahun, mengalami gejala kelelahan, sesak napas, dan intoleransi latihan yang memburuk sejak pertama kali didiagnosis 12 tahun lalul. Setelah pemeriksaan menyeluruh, pasien didiagnosis dengan PDA, hipertensi pulmonal (PAH) aliran tinggi resistensi tinggi, dan tes oksigen reaktif. Prosedur pembedahan pasien meliputi penutupan PDA melalui median sternotomi dengan *cardiopulmonary bypass* (CPB). Anestesi yang digunakan yaitu anestesi berbasis opioid dengan *deep hypothermia circulatory capture* (DHCA) selama 9 menit. CPB dan DHCA, bersama dengan tindakan neuroprotektif dan pemantauan yang tepat, dapat menjadi metode anestesi yang aman untuk pasien dewasa penderita PDA dan hipertensi pulmonal yang menjalani operasi ligasi PDA.

Kata Kunci: ***Cardiopulmonary bypass; Deep hypothermic circulatory arrest; Patent ductus arteriosus***

Abstract
Patent ductus arteriosus (PDA) is a congenital heart condition that occurs when the normal fetal blood vessel called the ductus arteriosus, which connects the pulmonary artery and aorta in utero, fails to close after birth. This case report focuses on anesthesia management in adult PDA undergoing PDA ligation surgery. A 33-year-old female had symptoms of fatigue, shortness of breath, and exercise intolerance that worsen since first diagnosed 12 years ago. After thorough evaluation, the patient was diagnosed with PDA, high flow high resistance PAH, and reactive oxygen test. The patient's surgical procedure involved PDA closure via median sternotomy with cardiopulmonary bypass (CPB). Opioid based anesthesia was used, supported with deep hypothermia circulatory arrest (DHCA) for 9 minutes. CPB and DHCA, in addition to neuroprotective measures and proper monitoring, can be secure anesthesia method for adult patients with PDA and pulmonary hypertension undergoing PDA ligation surgery.

Keywords: ***Cardiopulmonary bypass; Deep hypothermic circulatory arrest; Patent ductus arteriosus***

PENDAHULUAN

Patent ductus arteriosus (PDA) is a congenital heart condition that occurs when the normal foetal blood vessel called the ductus arteriosus (DA), which connects the pulmonary artery and aorta in utero, fails to close after birth.[1] The DA is an important component of fetal circulation, directing cardiac output away from the lungs toward the placenta to sustain systemic oxygenation. The DA is no longer required after birth since the placental circulation is cut off and removed, the pulmonary vascular bed's resistance lowers, and the lungs take over as the primary source of gas exchange and oxygenation. The DA shuts in >90% of term newborns by 48 hours and in 100% by 96 hours of age. In a small number of individual, a PDA will remain open into later childhood and adult life.[2]

PDA is uncommon in adults. Longitudinal cohort studies suggest that the incidence of "silent" PDA, those cases discovered by cardiac imaging in the absence of clinical manifestations, approach 1 in 20 births. American Heart Association/American College of Cardiology guidelines emphasize that considerations for ductal closure among older patients be made in the context of evaluation of left-to-right shunting and hemodynamic assessment for PAH.[3] Typically, closure through surgery or device placement is performed in children. However, closure becomes more challenging if complications like infective endarteritis, congestive heart failure, pulmonary hypertension, and calcification develop.[4]

This case report focuses on anesthesia management in an adult patient with a patent ductus arteriosus undergoing surgery.

KASUS

The patient, a 33-year-old female, presented with symptoms of fatigue, shortness of breath (SoB), and exercise intolerance. She was first diagnosed 12 years ago during an echocardiography examination on her pregnancy. She got oral medication since than but the symptoms have been worsen. Her daily medication was including bisoprolol 5 mg once daily, furosemide 40 mg once daily, sildenafil 20 mg 3 times a day, and spironolactone 25 mg once daily. The patient is a housewife. She couldn't do household chores without the feeling of SoB. She was able to walk about 500 m without breathlessness. After a thorough evaluation, including cardiac imaging, the patient was diagnosed with a patent ductus arteriosus, high flow high resistance pulmonary arthery hypertention (PAH), and reactive oxygen test. The patient's surgical procedure involved closure of the patent ductus arteriosus via median sternotomy with cardiopulmonary bypass (CPB).

During the preoperative evaluation, physical examination found that the patient was underwight (body weight 29 kg, BMI 12). There was grade 2 systolic murmur, no rhales, ronkhi nor wheezing on her lungs. A chest X-ray showed pulmonary hypertension and cardiomegaly. Transoesophageal echocardiography examination revealed the PDA diameter was 1.3 cm, bidirectional shunt, left atrium and left ventricle dilatation, eccentric left ventricular hypertrophy and right ventricular hypertrophy. Global and segmental systolic function were both good with EF 55%. Grade Il diastolic dysfunction was found, as well as pulmonary hypertension, mild pulmonary and tricuspid regurgitation.

The patient underwent a ductus arteriosis ligation procedure via sternotomy. Anesthesia procedure midazolam 0.05 mg/kgBB, fentanyl 4 mcg/kgBB, rocuronium as a muscle relaxant.

Because there was an aortic rupture, DHCA procedure was performed to obtain good exposure of surgery field. The temperature was maintaned at 20^{o}C. the cross-clamping duration was 60 minutes, whether duration of CPB was 140 minutes. During DHCA, methylprednisolone 30 mg/kg, sodium thiopental 20 mg/kg, and ice packs around head and carotid artery were used for brain protection. The patient was weaned from bypass with heart rate of 115/min and blood pressure 110/55 mmHg in normal sinus rhythm, with inotropic support of injection milrinone 0.4 µg/kg/min with good urine output.

Left femoral artery was cannulated and right internal jugular venous catheter was also inserted. Body warmer was used to prevent hypothermia and blood glucose levels

were also checked periodicaly to prevent hypoglycemia. Patient was shifted to the intensive care unit and extubated 48 hours postoperatively.

DISKUSI

The prevalence of adult patients with congenital heart disease (CHD) is on the rise, leading to an increased likelihood of them undergoing cardiac and non-cardiac surgeries. Understanding the complex cardiovascular anatomy and physiology of CHD is essential for addressing the anesthetic implications of the defect. Anticipation of the unique challenges faced by adults with congenital heart disease undergoing surgeries can significantly reduce perioperative risks.[5]

Pre-operatively, echocardiogram should be available. Review of ventilator settings, the fraction of inspired oxygen concentration (FiO2), infusions, and vascular access is also very important prior to providing an anesthetic.[1]

Adults with CHD may develop pulmonary hypertension for a variety of reasons. Potential etiology includes pulmonary venous hypertension secondary to elevated ventricular end diastolic pressure, elevated pulmonary venous atrial pressure, or pulmonary vein stenosis.[6] Many patients also continue to have decreased oxygen saturation secondary to residual shunts, poor lung function. and persistent decreased of pulmonary flow.[1] Our patient had symptoms that were worsen so that needed a surgery.

In this case, the patient was induced with intravenous fentanyl, midazolam, and rocuronium. opioid-based anesthesia was used to reduce stress response and improves surgical outcomes.[5] Anesthesia was maintained with oxygen: air, sevoflurane, fentanyl, and rocuronium. Ventilatory management in a PDA ligation is aimed to maintain pulmonary vascular muscle tone. TheFiO2 and PaCO2 was minimizing between 35-40 mmHg, so that blood flow does not tend to the pulmonary circulation.

Deep hypothermic circulatory arrest (DHCA) is a well-known method utilized in certain surgeries where blood flow stops in all blood vessels as the patient's core body temperature is significantly lowered.[7] Repairs of the aortic arch or congenital repairs involving the aortic arch, may require DHCA.[8] In some emergency cases, DHCA may be performed unplanned for example to repair laceration in the right ventricle when opening the sternum.[7,9]

DHCA is essential for cardiac surgeries where standard cannulation of the proximal aorta cannot achieve cerebral perfusion. A systemic decrease in temperature can facilitate the cessation of blood flow, which is called circulatory arrest.[8,9] Circulatory arrest allows the surgeon to operate in a bloodless environment with better visibility since no cannula or clamps are needed.[9] The duration of DHCA considered as safe is still controversial. A retrospective study showed an increased risk of stroke when the duration of DHCA was longer than 40 minutes. Acceptable duration ranges from 30-40 minutes due to the increased risk of brain injury. Animal studies show significant brain tissue damage if it lasts longer than 45 minutes.[8,10] At normal temperature (37°C) the duration of cessation of blood circulation is only permitted for 5 minutes. Cerebral metabolism decreases by 6-7% for every 1°C decrease in temperature from 37°C, which implies that brain cooling results in a reduction in oxygen requirements.[4] So, if body temperature can be lowered to 15°C then the duration is permitted up to 31 minutes. The maximum duration of stopping blood circulation is 40 minutes because of increasing the risk of stroke. Despite its benefit, prolonged can result in a number of

problems of coagulopathy, post ischemic hypothermia and cerebral microembolism. Profound hypothermia is associated with Dysrhythmias due to electrolyte imbalance, increased plasma viscosity and erythrocyte rigidity, metabolic acidosis, hyperglycemia and altered drug distribution and elimination.[10]

When CPB is resumed after a period of DHCA, hypothermic perfusion should be maintained for 10–20 min before start to do a rewarming to reduce the risk of raised intracranial pressure. Once rewarming commences, the gradient between core and peripheral temperatures should be less than 5^{o}C. Excessively rapid rewarming with perfusion temperatures more than 37^{o}C may induce cerebral ischaemia secondary to an imbalance between oxygen supply and demand. Similarly, cerebral hyperthermia should be avoided as this may exacerbate neurological injury and increase the risk of adverse neurological outcomes.[10]

In this case, there was an aortic rupture during surgery. DHCA was performed for 9 minutes. It has been suggested that patients can tolerate up to 30 minutes of DHCA without significant neurological dysfunction.[3] Simultaneously, deep hypothermia reduces cerebral metabolism and oxygen consumption, allowing for a longer period to perform surgery during interrupted cerebral perfusion. As the brain is the most vulnerable organ to ischemia, ensuring adequate cerebral protection necessitates protecting other vital organ systems as well. In our situation, the patient's temperature was maintained at 20°C during the entire period of deep hypothermic circulatory arrest. To protect the brain, external cooling was also utilized using cooling blanked and ice-packs around the carotid artery.

KESIMPULAN

The utilization of CPB and DHCA, in addition to neuroprotective measures and proper monitoring, can be a secure anesthesia method for adult patients with PDA and pulmonary hypertension undergoing PDA ligation surgery.

DAFTAR PUSTAKA

Kritzmire SM, Boyer TJ, Singh P. Anesthesia for Patients With Patent Ductus Arteriosus. StatPearls [Internet]. 2022;1–11. Available from: https://www.ncbi.nlm.nih.gov/books/NBK572063/.

Backes CH, Hill KD, Shelton EL, Slaughter JL, Lewis TR, Weisz DE, et al. Patent Ductus Arteriosus: A Contemporary Perspective for the Pediatric and Adult Cardiac Care Provider. J Am Heart Assoc [Internet]. 2022 Sep 6;11(17):e025784. Available from: https://doi.org/10.1161/JAHA.122.025784.

Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, et al. 2018 AHA/ACC Guideline for the Management of Adults With Congenital Heart Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;139(14):e698–800.

Shinde SR, Basantwani S, Tendolkar B. Anesthetic management of patent ductus arteriosus in adults. Ann Card Anaesth. 2016;19(4):750–1.

Kwanten LE, O'Brien B, Anwar S. Opioid-Based Anesthesia and Analgesia for Adult Cardiac Surgery: History and Narrative Review of the Literature. J Cardiothorac Vasc Anesth [Internet]. 2019;33(3):808–16. Available from: https://doi.org/10.1053/j.jvca.2018.05.053.

Baum VC, Souza DG de. Congenital heart disease in adults. In: Kaplan JA, editor. Kaplan's Essentials of Cardiac Anesthesia for Cardiac Surgery. 2nd ed. Philadelphia: Elsevier; 2018. p. 385–400.

EM E, FD R. Deep hypothermic circulatory arrest: alternative strategies for cerebral perfusion. A review article. Perfusion. 2011;26(1 Suppl):27–34.

Patel PA, Augoustides JGT, Pantin EJ, Cheung AT. Thoracic Aorta. In: Kaplan DL, editor. Kaplan's Essentials of Cardiac Anesthesia for Cardiac Surgery. 2nd ed. Philadelphia: Elsevier; 2018. p. 411–2.

Ikenga CS, Sahu B, Okwulehie V, Umeh F. Emergency deep hypothermic circulatory arrest in a 1-year-old undergoing cardiac surgery in a nigerian hospital-anaesthesia and critical care interventions. Pan Afr Med J. 2020;37(221):1–6.

Conolly S, Arrowsmith JE, Klein AA. Deep hypothermic circulatory arrest. Contin Educ Anaesthesia, Crit Care Pain. 2010;10(5):138–42.