Cardiology in the Young

cambridge.org/cty

Non-operating room anaesthesia for embolisation of ductus closure device to the pulmonary artery in a child with patent ductus arteriosus

Yeliz Kılıç and Mehmet Ali Harbelioğlu

Department of Anesthesiology and Reanimation, Faculty of Medicine, Eskişehir Osmangazi University, Eskişehir, Turkey

Brief Report

Cite this article: Kılıç Y and Harbelioğlu MA (2019) Non-operating room anaesthesia for embolisation of ductus closure device to the pulmonary artery in a child with patent ductus arteriosus. *Cardiology in the Young* **29**: 725–726. doi: 10.1017/S1047951119000921

Received: 10 January 2019
Accepted: 30 March 2019
First published online: 3 June 2019

Key words:
Complication; non-operating room anaesthesia; patent ductus arteriosus

Author for correspondence:
Yeliz Kılıç, MD, Department of Anesthesiology and Reanimation, Faculty of Medicine, Osmangazi University, Büyükdere Mah., 26040, Odunpazarı, Eskişehir, Turkey.
Tel: +90 222 2392979; Gsm: +90 505 3573180;
E-mail: yeliz_kilic3@hotmail.com

ABSTRACT

Patent ductus arteriosus is among the most common congenital heart diseases. With the increasing use of transcatheter closure procedures, the incidence of complications related to the procedure has increased. Embolization of the ductus closure device to the pulmonary artery is a very rare complication. Since those procedures are often performed under non-operating room anesthesia, anesthetic management of such patients is of great importance. Herein, anesthetic management of embolization of the ductus closure device to the pulmonary artery in a little girl was presented. This is the first case regarding the anesthetic management of such complication.

Patent ductus arteriosus, defined as the failure in closing of the ductus arteriosus, is among the most common congenital heart diseases.[1] Treatment options include fluid restriction, diuretic agents, transcatheter closure, and surgical repair. Today, the first therapeutic option is transcatheter closure, which has been widely used since 1939 with high success.[2] This procedure is often performed under general anaesthesia outside the operating theatre, called non-operating room anaesthesia. With the increasing use of transcatheter closure procedures in recent years, the incidence of intraoperative complications has increased. Herein, we aimed to report our anaesthetic management of a rare complication of this procedure – embolisation of the ductus closure device to the pulmonary artery – in a child.

Case presentation

A 4-year-old girl, 12 kg in weight (10–25 percentile) and 96 cm in length (50 percentile), underwent transcatheter closure for patent ductus arteriosus at paediatric interventional laboratuar under non-operating room anaesthesia. She was stable haemodynamically (tension arterial, 90/60 mmHg; respiratory rate, 28 per minute; hearth pulse, 104 beats/minute). Blood tests were normal. No abnormality was observed in her physical examination. She had not any syndrome associated with patent ductus arteriosus. Her Mallampati score was consistent with American Society of Anesthesiologists score 2. She was given 50 mg/kg ampicillin/sulbactam before the procedure.

Following standard monitoring, induction was slowly performed with propofol (3 mg/kg), remifentanil (0.2 mcg/kg), and rocuronium (0.4 mg/kg). The patient was then intubated with using 5 mm cuffed endotracheal tube. Anaesthesia was maintained with using 50%/50% (air/O_2) and 2–3% sevoflurane without any problem. Pulmonary artery pressure was measured as 26 mmHg. Vasoreactivity test with ilioprost was accepted as positive. Then, patent ductus arteriosus closure was successfully performed with amplatzer duct occluder II device. After providing extubation criteria, the patient was successfully extubated without using a muscular relaxant antagonist.

Control transthoracic echocardiography was performed before the patient was removed from the operating table, and it was revealed that the device was dislocated and embolised to the left pulmonary artery. Thereupon, the patient was re-intubated with the administration of propofol (3 mg/kg), remifentanil (0.1 mcg/kg), and rocuronium (0.4 mg/kg). Thanks to extubation without reverse agent, no problem was observed during the second induction. However, instant hypoxia, hypo/hypercarbia, hypotension, and arrhythmias developed due to the multiple manipulations. Norepinephrine infusion (0.05–0.10 mcg/kg per minute) was started for persistent hypotension. Metabolic acidosis was treated by the administration of bicarbonate (16.8 mEq intravenous and 8.4 mEq infusion for 6 hours). Despite all manipulations lasting 4 hours, the device could not be replaced into the correct position (Fig 1a and b).

CAMBRIDGE UNIVERSITY PRESS

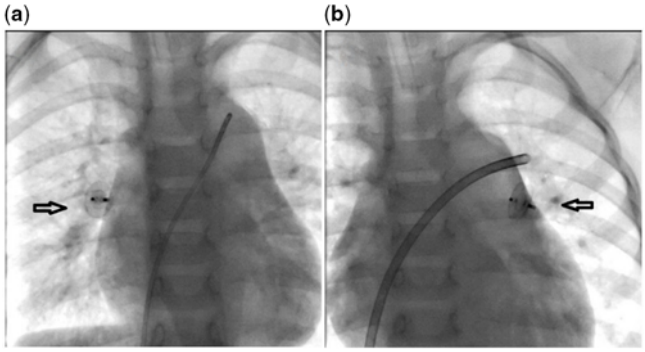

Figure 1. (*a*) The closure device embolised to the right pulmonary artery (arrow). (**b**) The closure device embolised to the left pulmonary artery (arrow).

Subsequently, paediatric cardiologists decided to terminate the procedure and referred the patient to surgery. She was successfully extubated with using 4 mg/kg sugammadex, and was taken to the intensive care unit. The patient was referred to the external medical centre the next day. Patent ductus arteriosus repair and removal of the device were performed surgically there.

Discussion

Increased positive expiratory pressure (>5 mbar) and short inspiration time (0.35 second) are recommended as the ventilation strategy in order to correct impaired gas exchange, reduce left-to-right shunt, and keep systemic blood flow high. The presence of a left-to-right shunt causes an increased risk of pulmonary hypertension, which is a higher risk for perioperative cardiovascular complications, including cardiac arrest, pulmonary hypertensive crisis, and death compared with normal population.[3,4] This risk is considered to be independent of patient's age, anaesthetic or airway technique, but to be related to the duration of anaesthesia. In anaesthesia management, hypoxia, hypercarbia, and acidosis should be prevented, and pain management should be optimal. Hypovolemia may result in a decrease in cardiac output and pulmonary blood flow. Systemic hypotension or reduction in systemic vascular resistance together with a decrease in coronary artery blood flow may lead to biventricular ischaemia. We did not see any clinical pulmonary hypertension in our patient, but it should be always kept in mind.

Balanced anaesthesia should be preferred. In the premedication, benzodiazepines are appropriate because of their less haemodynamic side effects. Inhalation agents may impair the perfusion compliance by reducing hypoxic pulmonary vasoconstriction and are also known to decrease pulmonary vascular resistance by pulmonary vasodilatation.[5] Although volatile anaesthetics cause prolongation of the QT interval, it was not considered clinically significant.[6] Etomidat and fentanyl can be preferred, whereas thiopentalin is considered as a non-suitable agent due to its systemic hypotension effect and myocardial depression. The general opinion is that slow use of propofol in low doses should be preferred due to its impact on reducing systemic vascular resistance and contractility.[7]

Pulmonary gas exchange abnormalities firstly occur after embolism of pulmonary device. Increase in dead space, formation of right-to-left shunt, and ventilation/perfusion incompatibility cause this situation.[6] Blood flow is directed to the remaining lung tissue instead of a clogged pulmonary artery, and pulmonary edema, alveolar haemorrhage, and surfactant loss may develop due to this increased flow.[8] In case of persistent embolism, left ventricular preload and subsequently left ventricular output decrease, which cause a systemic hypotension. Supportive therapies are in the forefront of anaesthesia management, and blood pressure should be increased with right ventricular stroke volume and systemic vasoconstriction, and coronary perfusion pressure should be preserved. Norepinephrine is recommended as a first inotropic agent.[9] It provides an increase in venous return, blood pressure, and coronary perfusion pressure by increasing the systemic vasoconstriction via α-1 receptors. In addition, norepinephrine increases both right ventricle and cardiac output through the stimulation of β-1 receptor. As an alternative to norepinephrine, dopamine, epinephrine, and dobutamine can also be used.[9] Additionally, it is unreliable to check the ventilation of patients by end-tidal carbon dioxide, because these patients have a higher physiological dead space that may cause incorrect readings. The blood sample taken from the arterial catheter inserted during the procedure can be used to determine partial carbon dioxide pressure values and be helpful for ventilation strategies.

Conclusion

Device embolisation to the pulmonary artery is a very rare complication of transcatheter closure of patent ductus arteriosus, which carries additional risks in the anaesthetic management of such patients. Therefore, we consider that this case report may be helpful and informative for all anaesthesiologists.

Financial Support. This research received no specific grant from any funding agency or from commercial or not-for-profit sectors.

Conflicts of Interest. None.

Ethical Standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human medical regulations and with the Helsinki Declaration of 1975, as revised in 2008.

References

1. Baruteau AE, Hascoët S, Baruteau J, et al. Transcatheter closure of patent ductus arteriosus: past, present and future. Arch Cardiovasc Dis 2014; 107: 122–132.
2. Kenny D, Morgan GJ, Bentham JR, et al. Early clinical experience with a modified Amplatzer ductal occluder for transcatheter arterial duct occlusion in infants and small children. Catheter Cardiovasc Interv 2013; 82: 534–540.
3. Taylor CJ, Derrick G, McEwan A, Haworth SG, Sury MR. Risk of cardiac catheterization under anaesthesia in children with pulmonary hypertension. Br J Anaesth 2007; 98: 657–661.
4. Carmosino MJ, Friesen RH, Doran A, Ivy DD. Perioperative complications in children with pulmonary hypertension undergoing noncardiac surgery or cardiac catheterization. Anesth Analg 2007; 104: 521–527.
5. Friesen RH, Williams GD. Anesthetic management of children with pulmonary arterial hypertension. Paediatr Anaesth 2008; 18: 208–216.
6. Hayman M, Forrest P, Kam P. Anesthesia for interventional cardiology. J Cardiothorac Vasc Anesth 2012; 26: 134–147.
7. Türktan M, Hatipoğlu Z. Anesthetic management of pediatric pulmonary arterial hypertension. Arch Med Rev J 2015; 24: 149–158.
8. Desciak MC, Martin DE. Perioperative pulmonary embolism: diagnosis and anesthetic management. J Clin Anesth 2011; 23: 153–165.
9. Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest 2002; 121: 877–905.

https://doi.org/10.1017/S1047951119000921 Published online by Cambridge University Press