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Case Report

PERIOPERATIVE ANAESTHETIC MANAGEMENT IN REPAIR DIAPHRAGMATIC HERNIA WITH ATRIAL SEPTAL DEFECT AND PULMONARY HYPERTENSION

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ABSTRACT

Introduction: Patients who had suffered disease of atrial septal defect (ASD) coming by pulmonary hypertension (PH) often present a clinical dilemma. Both of these disorders are congenital anomalies that often appear in pediatrics. Anaesthetic management in diaphragmatic hernia repair with this comorbidity requires precision and accuracy to avoid morbidity and mortality during surgery. The right management and care of anesthetic procedures is needed for patients who will be operated on with these two disorders. **Objective:** To describe the anesthetic management of a diaphragmatic hernia repair patient with comorbid ASD and pulmonary hypertension. **Case report:** The patient, a 1.5-month-old female baby born, has presented with shortness of breath complaints since the birth. Those were born spontaneously at the midwife's office and did not cry immediately, and a history of blueing and decreased consciousness was admitted for 20 days. Based on the examination, the diagnosis of diaphragmatic hernia from echocardiography foundASD and PH with a left ventricular ejection fraction of 64%. The patient was planned for diaphragmatic hernia repair under general anesthesia. Induction of anesthesia was performed with 5 mcg of fentanyl and inhalation anesthetic 3.5 vol% sevoflurane. After the endotracheal tube (ETT) was attached, the patient was desaturated to 50%, then the hyperventilated oxygenation was performed and positioned with knee chest position, and then milrinone at a dose of 1 mcg/min was given, saturation rose to 100%. During intraoperative ventilation control with manual bagging and maintenance anesthesia with inhalation ansethetic sevoflurane of 3.2 vol%. After surgery, the patient was admitted and observed in the pediatric intensive care unit for 2 days before extubation. **Conclusion:** Appropriate perioperative management in ASD patients with PH can reduce perioperative morbidity and mortality.

Keywords: Anaesthetic Management; Atrial Septal Defect (ASD); Diaphragmatic Hernia; Pulmonary Hypertension (PH); Human and Health

ABSTRAK

Pendahuluan: Pasien dengan atrial septal defek (ASD) disertai hipertensi pulmonal (PH) sering menimbulkan dilema klinis. Kedua kelainan ini termasuk kelainan kongenital yang sering muncul pada pasien pediatri. Manajemen anastesi pada repair hernia diafragma dengan komorbid ini memerlukan ketelitian dan kecermatan agartidak terjadi morbiditas dan mortalitas selama operasi. Diperlukan manajemen anastesi khusus dan cermat pada pasien yang akan dilakukan operasi dengan kedua kelainan ini. **Tujuan:** Pada laporan kasus ini penulis akan memaparkan manajemen anastesi pada pasien repair hernia diafragma dengan komorbid ASD dan hipertensi pulmonal. **Laporan kasus:** Melaporkan Pasien Bayi perempuan 1.5 bulan lahir cukup bulan datang dengan keluhan sesak nafas sejak lahir. Pasien lahir spontan di bidan tidak langsung menangis dan riwayat membiru dan penurunan kesadaran dirawat 20 hari. Berdasarkan pemeriksaan, pasien di diagnosis dengan hernia diafragma dari pemeriksaan echokardiografi ditemukan adanya ASD dan PH dengan ejeksi fraksi ventrikel kiri 64%. Pasien kemudian direncanakan operasi repair hernia diafragma dengan anastesi umum. Induksi anastesi dilakukan dengan menggunakan agen analgetik fentanyl 5 mcg dan anastesi inhalasi sevofluran 3.5 vol%. Setelah selang endotrakeal tube (ETT) terpasang dengan baik, pasien mengalami desaturasi hingga 50%, sehingga dilakukan oksigenasi hiperventilasi dan pasien diposisikan dengan *knee chest position* diberikan milrinon dosis 1 mcg/menit, saturasi naik hingga 100%. Selama intraoperasi dilakukan kontrol ventilasi dengan manual bagging. Maintenace anastesi dengan menggunakan anastesi inhalasi sevofluran 3.2 vol%. Setelah operasi selesai pasien dirawat dan diobservasi di ruangan intensive pediatrik selama 2 hari sebelum akhirnya dilakukan ekstubasi. **Kesimpulan:** Manajemen perioperatif yang tepat pada pasien ASD

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dengan PH dapat menurunkan morbiditas dan mortalitas perioperatif.

Kata Kunci: Manajemen Anastesi; Atrial Septal Defek (ASD); Hernia Diaframa; Hiperteni Pulmonal (PH); Manusia dan Kesehatan

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INTRODUCTION

Atrial septal defect (ASD) is a common congenital heart disease with a prevalence of 1.6 per 1000 births and accounts for 8-10% of congenital heart defects (CHD). Pulmonary hypertension (PH) is defined as systolic pulmonary artery pressure (PASP) ≥40 mmHg and occurs in 6% to 35% of patients withASD, of which 9%-22% of cases are moderate to severe. PH in ASD can be caused by several etiologies. Post calpillary PH can be due to increased left ventricular end diastolic pressure (LVEDP). Pre-capillary PH occurs due to the presence of a large shunt (1).

Diaphragmatic hernia is a complex and severe condition, but it is rare. It occurs in 1: 2000 - 5000 pregnancies, male to female ratio is 2: 1. In this hernia, there is a defect in the diaphragm muscle that allows intra-abdominal organs to enter the chest cavity. It is more common in the left hemithorax, with a prevalence of 85%-90%. However, most cases occur before or during the period after birth. Management delays will increase morbidity in these patients. Anesthetic management in diaphragmatic hernia repair patients with congenital heart defects such as ASD and PH is different from those without such defects. This case report will present the perioperative management of a patient undergoing diaphragmatic hernia repair with comorbid ASD and PH (1).

CASE REPORT

The patient was a 1.5 months old female infant with complaints of breathlessness since birth. She was born spontaneously at full term in a private clinic. At birth, the baby did not cry immediately with blue lips and fingertips, and resuscitation was carried out and the baby cried and started to turn red. After 5 days at home, the patient returned to blueness and shortness of breath. The patient did not want tobreastfeed and every time the patient vomited and turned blue. Those who suffered were hospitalized and reported to the perinatology room with a diagnosis of atrial septal defect, pulmonary hypertension and diaphragmatic hernia. The patient was consigned to surgery and anesthesia for emergent laparotomy, exploratory adhesiolysis, repair diaphragmatic hernia surgery.

The patient was stabilized and treated for 30 days and given furosemide 2x1mg, Sildenafil 2x1 mg, and ramipril 1x0.25mg. From the physical examination of the patient with a body weight of 2900 grams, alert consciousness, respiratory frequency of 60 x/min, heart rate of 172 x/min, and peripheral oxygen saturation 96%-98% using a nasal cannula 1 liter per minute. Examination of the thoracic wall found intercostal retraction (+), left hemithorax movement is left, on auscultation vesicular breathing sound weakened in the left hemithorax, ronkhi and wheezing are not found, and regular heart sounds with mur-mur (+). The patient's laboratory examination showed HB 10.4 g/dL, other values were within normal limits (Table 1).

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Table 1. Laboratory Value

Parameter	Value
HB	10.4 g/dL
WBC	10.000 /uL
Platelet	453.000 /uL
HCT	31.9 %
PT	14.5 seconds
APTT	33.2 seconds
INR	1.02
AST	35 U/L
ALT	22 U/L
Random Blood Glucose	89 mg/dL
Albumin	4.1 g/dL
Ureum	9 mg/dL
Creatinine	0.38 mg/dL
Na	139 mmol/L
K	5.1 mmol/L
Cl	97 mmol/L

On chest X-ray, the heart size was difficult to assess, with multiple cavities in thesinistra hemithorax suspected of sinistradiaphragmatic hernia (Figure 1). Echocardiography found a small secundum ASD of 2.6 mm, a left to right shunt, moderatetricuspid regurgitation, a pressure gradient (PG) of 64 mmHg, left ventricular function with an ejection fraction of 64%, and systolic function of 32%. Figure 2 depicts the impression of a small secundum ASD in conjunction with pulmonary hypertension.

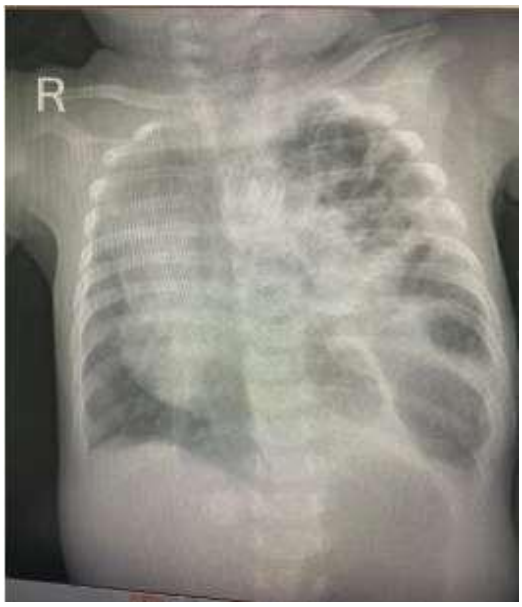

Figure 1. Chest X-ray

Based on the examination, the patient was diagnosed with a primary diagnosis of diaphragmatic hernia and was planned to undergo laparatomy exploration and repair hernia under general anesthesia. On pre-anesthetic evaluation, the patient was labeled American Society of Anesthesiologists (ASA) Physical Status III with pulmonary hypertension and an atrial septal defect.

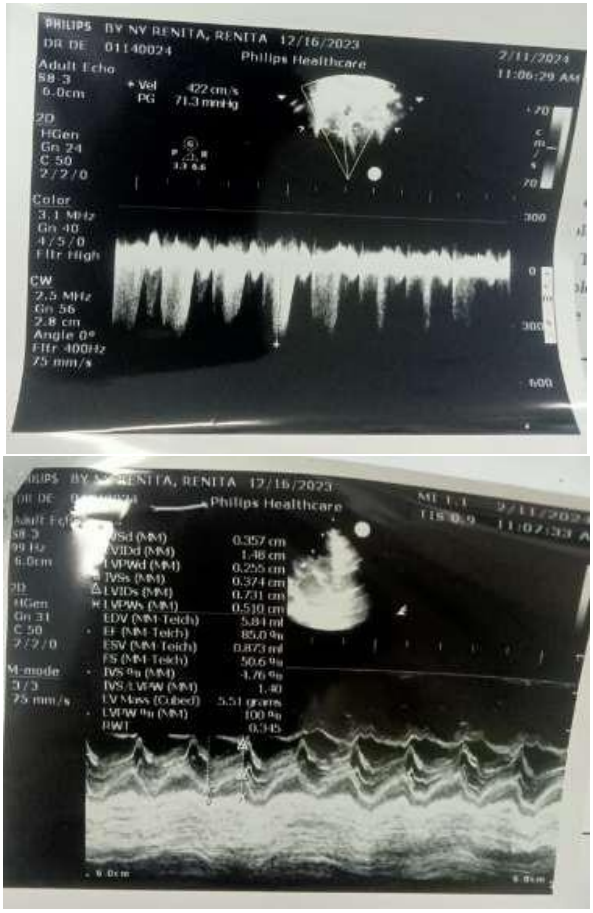

Figure 2. Echocardiographic

The surgery was performed after 1 month of pediatric cardiology therapy. Before surgery, the patient was fasted for 6 hours, and the therapy from cardiology was continued during perioperative. The operating room was fitted with monitoring devices for oxygen saturation (SpO2), heart rate (HR), echocardiogram (ECG), blood pressure (BP), and end tidal CO2 (ETCO2). The patient was preoxygenated for 3 minutes, then induced with 5 mcg fentanyl.

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The patient was sedated with 3.5 vol% sevoflurane and the patient were cuffed for 2 minutes. After deep sleep and apnea the patient was intubated with an ETT no. 3 uncuff.

Table 2. Post-operative Laboratory (14/02/2024)

Parameter	Value
HB	9.5 g/dL
WBC	22.440 /uL
Platelet	389.000 /uL
HCT	29.4 %
pH	7.18
pCO2	88.5 mmhg
pO2	172 mmhg
HCO3	32.9 mmol/L
TCO2	36 mmol/L
BE	4 mmol/L
SaO2	99%
CRP	74 mg/L

After intubation, the patient was desaturated to 50% and had blue lips. The patient was placed in knee-deep position and oxygenated with hyperventilation, then administered milrinoneat a dose of 1 mcg/min. During intraoperative ventilation control with manual bagging, maintenance anesthesia with sevoflurane 3.2 vol%, oxygen 60%, water 40%, and a flow rate of 4 liters per minute. The operation lasted 75 minutes with stable hemodynamics during surgery without the need for vasopressors or inotropic agents. After the surgery was completely finished, one was sent into pediatric intensive care unit (PICU) with ETT retention. The patient was monitored for 2 days and evaluated for a thoracic X-ray (Figure 3), laboratory, and postoperative blood gas analysis (Table 2). While in thePICU, the patient was alert and underwent gradual ventilator weaning, periodic suctioning due to sputum retention causing hypercarbia, and a 40 cc PRC transfusion to correct anemia. On the 3rd postoperative day, extubation was performed and observed for 24 hours, the patient stabilized and moved to the regular room.

Table 3. Post Transfusion Laboratory (16/02/2024)

Parameter	Value
HB	11.7 g/dL
WBC	15.640 /uL
Platelet	393.000 /uL
HCT	37.3 %
pH	7.24
pCO2	80.5 mmhg
pO2	150 mmhg
HCO3	34.8 mmol/L
TCO2	37 mmol/L
BE	7 mmol/L
SaO2	99%

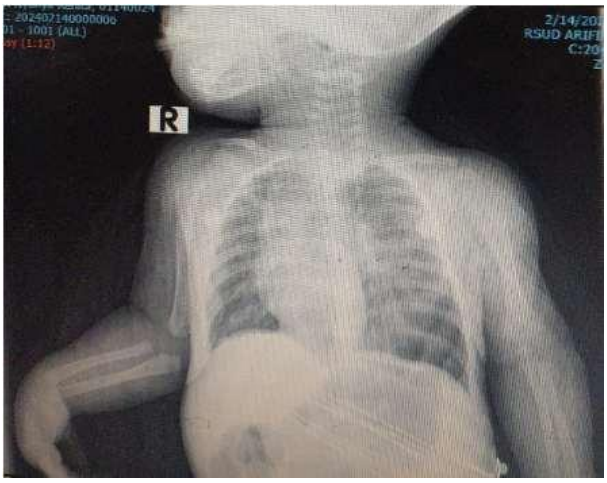

Figure 3. Post-operative Chest X-ray

Table 3 shows improvement in laboratory values; Hb increased and leukocytes decreased, and blood gas analysis seemed to have improved even though it was not significant. Figure 4 shows hemodynamic monitoring during surgery. There was a decrease in blood pressure and saturation at the beginning of the operation, but after management, blood pressure and oxygen saturation stabilized, and there was no significant decrease until the operation was completed.

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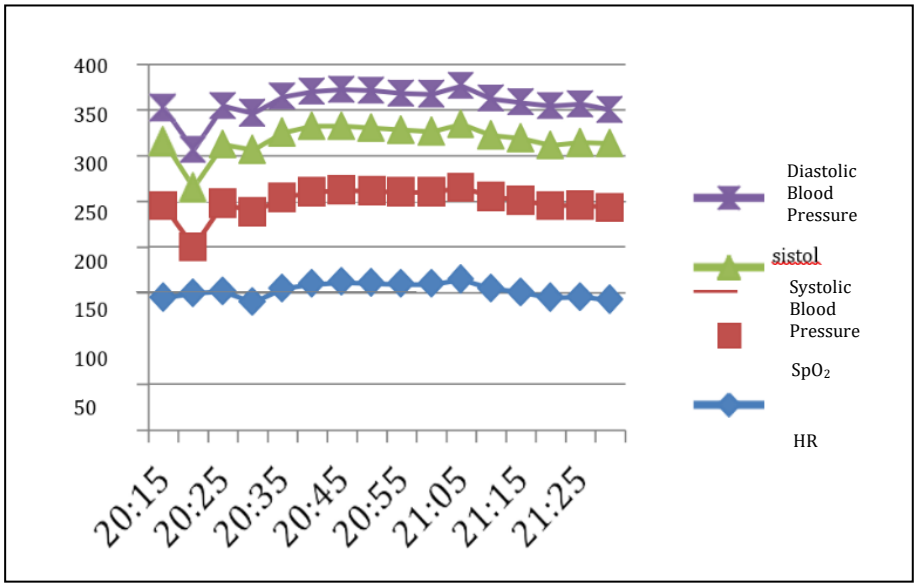

Figure 4. Intraoperative Hemodynamics

DISCUSSION

Congenital diaphragmatic hernia occurs in about 1 in 2500 to 5000 births. The male to female ratio is 2:1, and it is more common in the left diaphragm than the right. The diaphragm may develop defects during development because embryologically, it consists of the union or fusion of multiple tissues (1,2). According to current guidelines, hernia repair is customized according to expertise, resources, and elements related to the patient's condition, including comorbidities such as heart disease (3). In the case of non-cardiac surgery patients, comorbid heart disease is the most significant factor in severity and mortality (4). Patients with PH often undergo non-cardiac surgical procedures. In a study in the US, PH was admitted in a range of 0.81% of cases that had a prevalence improving from 0.4% in 2004 to 1.2% in 2014 (5). Perioperative complicationsthat occur include respiratory failure (28%), cardiac arrhythmias (12%), failure of congestive heart (11%), failure of acute renal (7%), septic shock (7%), and also postoperative death (7%) (6).

Optimization of PH Patients for Surgery

Two to four weeks before the surgery process becomes such areasonable moment for a patient visit in order to help determine therisk stratification components that are related to the surgery process. This can include an assessment of diagnostics as well as functional status. PH management in patients who will undergo surgery is necessary before the procedure in order to minimize the risk of perioperative complications. To address the pulmonary vascular abnormalities found in pH, many therapies have been developed, often referred to as pulmonary vasodilators. However, their mechanism of action is complex, including antiproliferative effects (7).

There are three classes of drugs for specific therapy of PH: (1) Nitric oxide (NO) pathway mediators: phosphodiesterase 5 (PDE5) inhibitors: tadalafil and also sildenafil; soluble guanylate cyclase stimulators: riociguat. (2) Endothelin receptor antagonists include ambrisentan, bosentan, and macitentan. (3) Prostacyclin pathway prostacyclins: agonists: treprostinil (subcutaneous, oral, inhaled, or even intravenous), epoprostenol (intravenous

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or inhaled); iloprost (inhaled), and also prostaglandin I2 receptor agonists: selexipag (oral). Every patient who had PH had to get alarmed in order to take diuretics prior to surgery to prevent acute cardiac decompensation during the perioperative period. In this case, the patient received selective therapy consisting of sildenafil taken orally for 20 days and furosemide taken twice a day at a dose of 1 mg. (8).

Intraoperative Anesthetic Management Hemodynamic Targets

Intraoperative hemodynamic targets in PH patients lead on purpose to help anticipating the acute dysfunction of RV and also keeping the cardiac index in order to make sure the process of perfusion of adequate end-organs. These are general guiding principles we are able to get to prevent systemic hypotension. Avoid using pulmonary vasoconstrictive agents not inotropic to the RV, such as phenylephrine when hypotension occurs. Maintain sinus rhythm. β-blocker and calcium canal blocker therapy should be continued preoperatively, but intraoperative initiation should generally be avoided due to negative inotropic effects (9). Reduce or avoid factorsto help improve PVR as like acidosis, hypoxia, hypothermia, hypercarbia, and pain. Avoid increasing the airway pressure and positive end expiratory pressure (PEEP). These are able to get titrated for maximal exchange of gas, hemodynamics, and also function of RV. Recommended ventilator settings are tidal volume 6 to 8 mL/kg ideal body weight, PEEP 5 to 10 mmHg, respiratory rate that is titrated to Paco2 30 to 35 mmHg, pH > 7.4, and also inhaled oxygen fraction that is titrated to Spo2 > 92% (10). It is important to maintain the baseline condition of RV filling. Hypovolaemia is going to emerge from the decrease inside MAP and also perfusion of RV, while hypervolemia makes overloading of RV and wall tension, making the function of RV get worse. RV hypertrophy may lead to a narrowing of preload volume, and volume overload becomes hardly tolerated (11). Optimize pressure of the central venous, preload, and RV through diuretics and also PAH targeted therapy. In an ideal way, optimization is made in the preoperative setting, but acute transformations of the intraoperative moment could need titration of those processes of therapy. In the intraoperative setting, inhaled (NO or prostacyclin) or even parenteral (subcutaneous or intravenous prostacyclin) therapy for PH is able to be done to acutely minimize the overload of RV. Immediate hemodynamic effects are able to be filled by changing the position of the body (e.g., the Trendelenburg position and also the elevation of the leg are going to improve the preload, while the reverse position of the Trendelenburg is going to make such an acute decrease in the preload).

General Anesthesia

Significant hemodynamic changes that contribute to acute RHF may be associated with the use of anesthetic agents in induction. Etomidate (0.15-0.3 mg/kg) has no significant influence on contractility, heart rate, or systemic PVR in patients with PH; there has been no comparative research or studies about the maximal induction agent (12). Nevertheless, the continuous process of infusion or repeatedetomidate administration is able to reduce morbidity and mortality. Ketamine is related to improved PVR in adults and is also best anticipated if monitoring of PVR or vasodilation of the pulmonarydid not get concurrent. Propofol may directly or indirectly influence the contractility of RV and could be applied with caution as it tends to require concurrent administration from

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vasopressor agents or even inotropes (13,14). Opioids, when reported alone, give minimal influence toward the circulation of the pulmonary and minimize the response to stimulation of sympathetic, but they also cause unfavorable bradycardia in larger and higher doses. Premedication that has been done through benzodiazepines and also opioids are going to be judiciously done; their co-administration will lead to an acute hypoxia-induced improvement in PA pressure and also hypercarbia. A general option to be done is to gain such an induction in rapid sequence through ventilation of the mask in order to reduce the hypercarbiaand hypoxia periods that lead to such a significant improvement in the afterload of RV. It matters to be done in order tomake a sufficient breathing moment while having ventilation of the mask, as high intrathoracic pressures cause such an acute drop in preload through the severe process of hypotension. Any inhaled agent is able to be applied for anesthesia care unless for nitrous oxide because of its influence on the impact of improving the PVR (15–18). There is a lack of comparative data about the influence of other generally used inhaled agents on PVR. If total intravenous anesthesia is chosen, a propofol infusion (50- 150 μg·kg-1·min-1) with or with no opioidscan be used. In accordance with the theory, this patient was induced with 5 mcg of opioid fentanyl and the patient was sedated with the inhalation agent sevoflurane with prior preoxygenation.

Selective Pulmonary Vasodilators

NO and also epoprostenol become such vasodilators in selective pulmonary surgery, most generally done in the non-cardiac process of surgery (19). NO is able to be given at doses of 1 to 80 ppm,even though most centers have a maximum dose of 20 to 40 ppm. Inhaled epoprostenol (iEPO) can be such as an aerosol recipe of epoprostenol and also be given in doses in the range of 10 and 50 ng/kg/min of NO and iEPO has efficacy and safety profiles in various small observational research, but procuring the cost, NO can be known to be higher than iEPO. Both tend to exhibit short half-lives, that let these agents attractive in the perioperative moment at hemodynamic objectives can changerapidly (20,21).

Vasopressors and inotropes

There are no found any comparative research and studies of inotropes and also vasopressors in those who suffered in PH cases. Norepinephrine and also vasopressin are commonly recommended over phenylephrine because they are able to help anticipate vasoconstriction of pulmonary, improvement of PVR, and also reflex bradycardia (22). Animal data reported that vasopressin has a more minimal influence on PVR than another vasopressor. Vasopressin in high doses (>0.08-0.1 U/min) is not recommended because it may exest an influence between coronary arteries that is brought to RV ischemia (23). The inodilator subclass of inotropes, like milrinone (25-50 μg/kg bolus over 15 minutes,0.25-0.75 μg/kg/min infusion) and also dobutamine (2.5-10 μg/kg/min), is able to be given in order to help increase the contractility of RV and minimize overloading RV. Both milrinone and dobutamine have systemic inotropic effects, and pulmonary vasodilator impacts that can cause severe hypotension of the systemic vascular system, thus sometimes needing more vasopressors, such as norepinephrine or terlipressin, to help maintain adequate MAP. Milrinone boluses are able to make significant systemic hypotension and thus have to be reduced in those who suffer from hypotension or hypovolemia. Both inodilators are also able to cause arrhythmias (24). In this patient,

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milrinone was used intraoperatively at a dose of 1 mcg/min, with stable hemodynamics during surgery. After surgery, milrinone was stopped, and the patient resumed preoperative therapy.

Congenital Heart Disease

Patients who have been corrected and also have uncorrected congenital heart disease are able to pose unique challenges in anesthetic management. This management will be given to every patient that has congenital heart disease which is still based on certain factors of physiology. Patients that have ventricular or atrialdefects can be given such a passive venous return, so it matters to help maintain preload and prevent improving PVR through high PEEP and intrathoracic pressure avoidance. For patients that had shunts it matters to anticipate an improvement in right-to-left shunts, that is able to be found even in those who had shunts of baseline left-to- right, as such shunts can lead to hypoxemia, acidosis, and systemic hypotension. In these patients, maintaining a low PVR:SVR ratio by avoiding increased PVR and decreased SVR, as well as maintaining contractility, preload, and cardiac output, is crucial (23, 24). The main limitation of this study is that we only included one patient, making our findings difficult to generalize to a broader population. In addition, the absence of a comparator also makes it impossible to compare outcomes between those exposed and unexposed.

CONCLUSION

Anesthetic management in pediatric patients with pulmonary hypertension and congenital heart disease requires special and more stringent attention, especially on PVRand SVR. During perioperative care, prevention of increased PVR and decreased SVR will provide a better outcome for the patient. The choice of anesthetic technique, anesthetic drugs, and pulmonary vasodilators, systemic vasopressors or inotropic drugs during perioperative care should also be considered individually depending on the patient'scondition. This case report only describes anesthesia management in one patient with comorbid pulmonary hypertension. It is hoped that further research can compare 2 or 3 cases with different anesthesia management techniques in patients with similar cases and comorbidities.

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Conflict of Interest

The authors declare that there is no conflict ofinterest.

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Authors' Contribution

M Irvan Noorrahman contributed in conceptualization, data collection, data analysis and interpretation, and manuscript preparation;

Pratama Ananda and Novita Anggraeni contributed in supervision, critical review, and final approval of the manuscript.

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