CASE REPORT

Journal of Indonesian Thoracic Cardiac and Vascular Surgery *(JINATCVS)* 2024,
Volume 1, Number 1: 15-20

Published By :
The Indonesian Association of Thoracic and Vascular Surgeons

Management in a 5-year-old diagnosed with severe coarctation of the aorta, aortic arch hypoplasia, doubly committed sub-arterial ventricular septal defect, large patent ductus arteriosus, and severe pulmonary hypertension: Case report

Suprayitno Wardoyo[1*], Dhama Shinta Susanti[1], Stacia Manggala[1]

[1]Division of Cardiothoracic Surgery, Department of Surgery, Faculty of Medicine, Universitas Indonesia, Indonesia.

*Corresponding to:
Suprayitno Wardoyo;
Division of Cardiothoracic Surgery, Department of Surgery, Faculty of Medicine, Universitas Indonesia, Indonesia;
suprayitno.wardoyo04@gmail.com

Received: 2024-01-09
Accepted: 2024-02-15
Published:2024-03-16

ABSTRACT

Introduction: Coarctation of the aorta is a congenital heart disease often associated with other cardiac abnormalities, including aortic arch hypoplasia and ventricular septal defect. These multiple anomalies also contribute to pulmonary hypertension, which can significantly increase mortality and morbidity. Management strategies for aortic coarctation and other heart defects vary based on individual cases.

Case description: We present the case of a 5-year-old diagnosed with coarctation of the aorta, aortic arch hypoplasia, doubly committed sub-arterial ventricular septal defect, large patent ductus arteriosus, and severe pulmonary hypertension, who underwent staged surgery beginning in 2023. The initial surgery successfully addressed aortic coarctation and aortic arch hypoplasia, along with pulmonary artery banding. A second surgery, intended for ventricular septal defect closure and pulmonary artery de-banding, is scheduled with a one-year interval following the initial procedure. Close monitoring of the patient's condition, particularly pulmonary pressure, will be maintained throughout this period. The patient has been under antihypertensive medication since before the first surgery.

Conclusion: The primary goal of intervention is to manage severe pulmonary hypertension and improve circulatory function. Follow-up evaluations in the patient have revealed favorable outcomes, evidenced by the patient's improved hemodynamic status and echocardiography results.

Keywords: coarctation of the aorta, long aortic arch hypoplasia, pulmonary hypertension, staged surgery.
Cite This Article: Wardoyo, S., Susanti, D.S., Manggala, S. 2024. Management in a 5-year-old diagnosed with severe coarctation of the aorta, aortic arch hypoplasia, doubly committed sub-arterial ventricular septal defect, large patent ductus arteriosus, and severe pulmonary hypertension: Case report. *Journal of Indonesian Thoracic Cardiac and Vascular Surgery* 1(1): 15-20.

INTRODUCTION

Coarctation of the aorta is the narrowing of the upper descending aorta, found around the ductus arteriosus. The narrowing occurs only in part, but the aortic wall before and after the narrowing part remains continuous. Aortic coarctation generally occurs distal to the left subclavian artery but more proximally between the left common carotid and subclavian arteries. Coarctation with or without present patent ductus arteriosus (PDA) is named isolated coarctation.[1]

Coarctation of the aorta is found in 6-8% of infants who have congenital heart disease, or 0,06-0,08% of the population. Aortic coarctation is a common cause of systemic hypertension in children. Aortic coarctation also contributes to heart failure in around 11% of children aged 1-14 years. There are various degrees of severity of aortic coarctation. Hemodynamically significant stenosis reduces the cross-sectional lumen area by approximately 50%. However, a more extended narrowing segment can change the hemodynamic status regardless of the diameter of the narrowed lumen.[1,2]

In some cases, coarctation of the aorta also occurs with other heart defects. PDA is almost always present in neonates and infants who have a preductal type of aortic coarctation. PDA will help circulation to the distal part of the aortic coarctation since a part of the aorta is narrowed. Patients diagnosed with aortic coarctation without PDA or closure of the PDA bring worse outcomes due to decreased blood supply to the distal part of aortic coarctation. Around 82% of individuals with coarctation have no other abnormalities, 11% have coexisting ventricular septal defect (VSD), and approximately 8% have other heart abnormalities.[1]

Coarctation of the aorta in neonates is also commonly found in the presence of VSD. When compared with isolated coarctation of the aorta, the presence of VSD can increase mortality, regardless of the surgery approach. Management of aortic coarctation with VSD traditionally

requires staged surgery. The first surgery aims to repair the coarctation of the aorta and pulmonary artery (PA) banding via the left thoracotomy approach, and the second surgery includes VSD closure with a median sternotomy approach.[3]

Pulmonary hypertension in children is defined as mean pulmonary artery pressure (mPAP) >25mmHg at rest after several months of age. When present, pulmonary hypertension will cause significant morbidity and reduce life expectancy in children due to right ventricular failure. Pulmonary hypertension etiology includes congenital heart disease, lung disease, systemic disease, or idiopathic. Pulmonary hypertension due to congenital and idiopathic heart disease is commonly found.[4–7]

Pulmonary hypertension in children with congenital heart diseases is then classified into pulmonary artery hypertension (PAH) due to Eisenmenger syndrome, PAH associated with systemic-to-pulmonary shunt, PAH associated with small defects (VSD<2cm, ASD <1cm), and PAH after corrective surgery. The presence of increased left-sided pressure in patients with coarctation of the aorta also predisposes a portion of the population to pulmonary hypertension and RV dysfunction.[5–7]

This case report aims to discuss perioperative management for patients with coarctation of the aorta with large doubly-committed subarterial (DCSA) VSD, large PDA, and severe pulmonary hypertension.

CASE DESCRIPTION

A 5-year-old male patient presented to the hospital with his parents, reporting recurrent shortness of breath, easy fatigue with minimal activity, and difficulty in weight gain since infancy. Congenital heart disease (CHD) was diagnosed when the patient was 3.5 months old, following similar complaints since birth. Examination revealed severe cardiomegaly, prompting referral to Adam Malik Hospital for echocardiography. The diagnosis of congenital heart disease was confirmed. Additionally, the patient has a history of anal atresia and underwent colostomy at 4 days old. Subsequently, the patient was referred to Cipto Mangunkusumo Hospital for corrective surgery. The patient's medical history includes treatment with Ramipril 1x0.5mg, Furosemide 2x5mg, and Sildenafil 3x5mg.

The general physical examination revealed that the patient was conscious, with a respiratory rate of 24 breaths per minute, a heart rate of 111 beats per minute, and a temperature of 36.3°C. Non-invasive blood pressure (NIBP) measurements of the four extremities showed readings of 94/49mmHg and 93/54mmHg for the right and left upper extremities, respectively, and 90/68mmHg and 88/63mmHg for the right and left lower extremities, respectively. Oxygen saturation (SpO_2) in the right and left upper extremities was 99% and 96%, while in the right and left lower extremities it was 85% and 83%, respectively. Thoracic examination revealed precordial bulging, dilated chest veins, normal S1, and a loud S2 with an ESM grade of 3/6. No cyanosis was observed. The patient's body weight was 9.8 kg, height 95 cm, with body weight/age at 54.4%, body height/age at 87.16%, and body weight/body height ratio at 70%. These findings indicate the patient is malnourished with short stature.

Chest X-ray (anteroposterior) results showed cardiomegaly with prominent pulmonary segments and infiltration in both lung fields that suggested pulmonary edema as shown in Figure 1. Echocardiography examination in Cipto Mangunkusumo Hospital (8/3/2023) showed atrial solitus, mild tricuspid regurgitation with pressure gradient 45mmHg, large DCSA VSD, bidirectional shunt with dominant right-to-left shunt, pulmonary artery dilatation with mild pulmonary stenosis, large PDA bidirectional shunt with dominant right-to-left shunt, pressure gradient 20mmHg, long segment aortic coarctation with the length of 9mm and diameter of 4mm as shown in Figure 2.

Multi-sliced Computerized Tomography examination displayed coarctation of the aorta in pre-ductal type with a PDA diameter of 11mm, atrial situs solitus, ventricle D-loop, VSD, RVOTO (-) and LVOTO (-) as shown in Figure 3. There are suggestive findings of pulmonary hypertension with a PT/AAo ratio of 1.8. RPA and LPA are confluent;

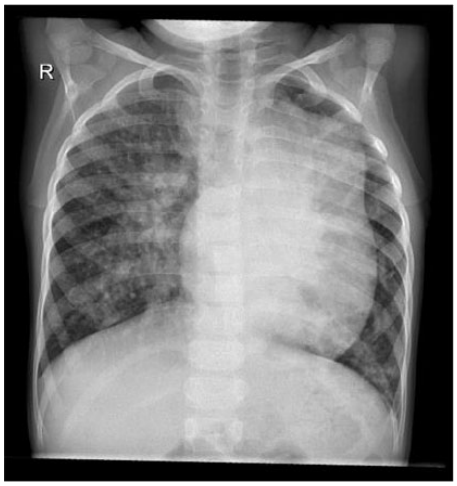

Figure 1. Chest X-Ray.

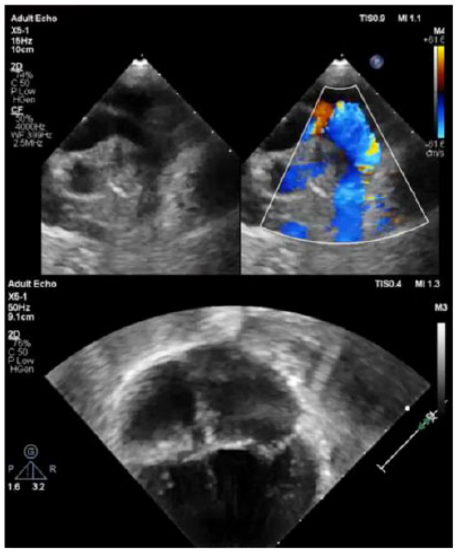

Figure 2. Echocardiography showed coarctation of the aorta and ventricular septal defect.

the RPA diameter was 17.2mm, and the LPA diameter was 14.7mm. CT Mc.Goon ratio was 2.9.

Based on the examinations conducted, the patient has been diagnosed with severe coarctation of the aorta, aortic arch hypoplasia, doubly committed subarterial ventricular septal defect, a large patent ductus arteriosus, and severe pulmonary hypertension. The patient was treated with Ramipril 1x0.5mg, Furosemide 2x5mg, and Sildenafil 3x5mg. The surgical strategy involved two stages with an interval of approximately 1 year between each surgery. Intraoperative Intervention is described in Figure 4. The first stage comprised correction of the coarctation of the aorta through the anastomosis of

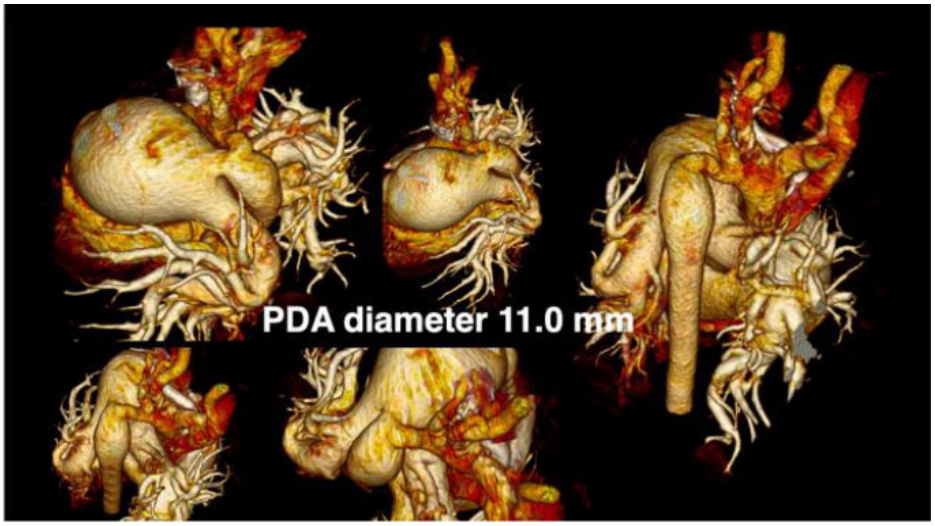

Figure 3. Multisliced Computerized Tomography showed coarctation of the aorta and patent ductus arteriosus.

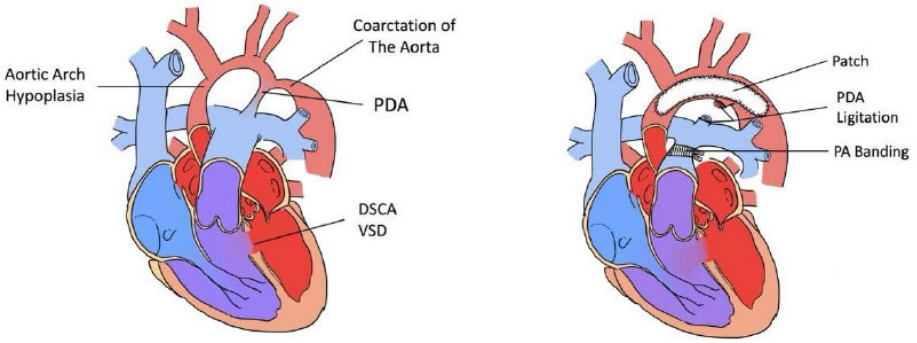

Figure 4. Intraoperative Intervention

the descending aorta to the ascending aorta, along with pulmonary artery (PA) banding. The second stage involved PA de-banding and ventricular septal defect (VSD) closure.

On December 21st, 2023, the first stage of surgery was conducted via median sternotomy utilizing a cardio-pulmonary bypass machine. Intraoperative findings revealed a significantly larger diameter of the main pulmonary artery (mPA) compared to the aorta, with the right pulmonary artery (RPA) exhibiting similar dimensions to the left pulmonary artery (LPA) and both confluences. An innominate vein was observed, along with a hypoplastic arch and pre-ductal coarctation of the aorta accompanied by a large patent ductus arteriosus (PDA). The PDA was ligated, and the aortic coarctation was repaired by incising distally to the ascending aorta, followed by anastomosis of the aortic arch and descending aorta with patch augmentation. The total duration of cardiopulmonary bypass (CPB) was 55 minutes, after which open chest management was initiated. The patient was then transferred to the Intensive Care Unit (ICU) with a radial blood pressure of 113/66 (mean arterial

Table 1. Echocardiography findings before and after the surgery

Echo (3/8/2023)	Echo (22/12/2023)	Echo (2/1/2024)
• Atrial solitus • Balanced four chamber • All pulmonary veins drain to left atrium • Mild tricuspid regurgitation, pressure gradient 45 mmHg • There is no mitral regurgitation • Large doubly committed sub-arterial ventricular septal defect, bidirectional shunt, dominant right-to-left shunt • Dilated pulmonary artery with mild pulmonary regurgitation • Large patent ductus arteriosus bidirectional shunt, dominant right-to-left shunt, pressure gradient 20mmHg • Long segment coarctation of the aorta 9mm, diameter 4mm • Good biventricular function	• Atrial solitus • Atrioventricular ventriculoarterial concordance • Dilated left atrium and left ventricle • Mild tricuspid regurgitation • Intact inter-arterial septum • Large subaortic ventricular septal defect 13mm bidirectional shunt • No patent ductus arteriosus seen • No left ventricular outflow tract obstruction • Main pulmonary artery banding pressure gradient 53mmHg • Left aortic arch • No residual coarctation of the aorta, aortic root dilation, pressure gradient 10mmHg • Fair contractility ventricle with paradoxical movement • Left ventricular ejection fraction 42% fractional shortening 20% • Right ventricle tricuspid annular plane systolic excursion 9mm • No pericardial effusion • No pleural effusion	• Balance four chamber • Stretch patent foramen ovale • Mild tricuspid regurgitation • Large malalignment perimembranous ventricular septal defect, bidirectional, dominant left-to-right shunt • Pulmonary artery band in place, pressure across pulmonary artery band is 35mmHg • Dilated pulmonary artery • Minimal residual coarctation of the aorta • No pericardial effusion • No pleural effusion • Good biventricular function

pressure 85) mmHg, a heart rate of 122 beats per minute, and central venous pressure (CVP) of 10 mmHg, supported by Dopamine at 5 mcg/kg/minute and Milrinone at 0.22 mcg/kg/minute. On December 22nd, 2023, sternal closure was performed. During the procedure, blood pressure was measured at 84/50 (mean arterial pressure 64) mmHg, and pulmonary artery pressure was recorded as 37/27 (mean 32) mmHg.

During the intensive care unit (ICU) stays, postoperative echocardiography was carried out (22/12/2023) with results of left atrium and left ventricle dilatation, mild tricuspid regurgitation, large subaortic VSD (13mm) bidirectional shunt. MPA banding with pressure gradient 53mmHg, no residual aortic coarctation, left ventricle ejection fraction 42%, fractional shortening 20%.

While in the ICU, patients also receive inotropic medication, including Adrenaline, Millirnone, and Dopamine, to support the patient's hemodynamic status during the recovery period.

The patient remained on the ventilator for a total of 96 hours and 50 minutes, starting on December 21st, 2023, at 13:10 and ending on December 25th, 2023, at 14:10. Following this, the patient was transferred to the High Care Unit (HCU) and stayed there until December 27th, 2023. Subsequently, the patient was relocated to an inpatient room until December 31st, 2023, when they were discharged home for outpatient treatment. Before discharge, the patient's final hemodynamic status showed a blood pressure of 94/67 mmHg, a heart rate of 92 beats per minute, a respiratory rate of 22 breaths per minute, and an oxygen saturation of 92% without any respiratory support. The urine output was also 2.1 cc/kg body weight/hour.

Echocardiography during outpatient (1/2/2023) showed large malalignment PM VSD, bidirectional, dominant L-to-R shunt, PA band with pG 35mmHg, dilated PA, minimal residual coarctation of aorta, good biventricular function. A summary of echocardiography findings is described in Table 1. The patient's current medications are sildenafil 3x5mg, Furosemide 3x5mg, Aldactone 1x6.25mg, and Ramipril 1x0.5mg.

DISCUSSION

The main strategy of aortic coarctation is cutting the narrowed segment, which can be done operatively or transcatheter. Surgery of aortic coarctation has improved since it was first described. Management of aortic coarctation cases with VSD involves several techniques and strategies. These cases can be managed by one one-stage approach or two-stage approach.[2,3,8]

A one-stage approach is achieved by aortic coarctation repair and VSD closure simultaneously through a median sternotomy and cardiopulmonary bypass with a single incision. But there are also some techniques using two incisions, through the median sternotomy and left thoracotomy, in a single procedure. The two-stage approach is achieved with two stages of surgery. The first surgery involves aortic coarctation repair and PA banding through a left thoracotomy and closing the VSD through median sternotomy in the second surgery.[3,8]

Some cases showed the presence of aortic coarctation with hypoplasia of the proximal transverse of the aortic arch. Several literatures describe extended end-to-end anastomosis procedures, assuming that the proximal arch will grow once the distal obstruction has been corrected.[8–10]

Another technique is median sternotomy and aortic arch augmentation to ensure no obstruction to left ventricle outflow. Research by Gray et al. explains that aortic arch augmentation through median sternotomy is a safe and effective procedure with low mortality and morbidity rates.[8]

Surgery management can be performed either by left thoracotomy or median sternotomy. The median sternotomy technique plays a role in cases when surgeons need to correct other intracardiac abnormalities accompanying aortic coarctation. This approach is also used when aortic coarctation is present with hypoplasia of the proximal transverse arch or when there is no proximal transverse arch because the left carotid and brachiocephalic arteries originate from the same origin. This is to ensure the repair of the hypoplasia of the aortic arch. Sakurai et al. found that using median sternotomy to repair the aortic arch was associated with a reduced re-intervention rate and re-coarctation risk.[1,3,8,11]

The patient in the case report had long-segment coarctation of the aorta accompanied by hypoplastic aortic arch, large PDA, DCSA VSD, and severe pulmonary hypertension. Due to the patient's condition, which showed severe pulmonary hypertension, it was more possible for the patient to carry out two-stage management. In comparison, the first stage consists of aortic coarctation repair and PA banding. The next stage plan is to do VSD closure, which will be carried out when the patient's subsequent follow-up shows the pulmonary pressure is less than two-thirds of the systemic pressure.

The patient's first stage of surgery was achieved through a median sternotomy and by using CPB during surgery, mainly due to the longer time needed for the aortic coarctation and arch hypoplasia repair. The first stage repaired the aortic coarctation, arch hypoplasia, PDA closure, and PA banding. The aortic coarctation and arch hypoplasia were repaired with an incision distal to the ascending aorta and patch augmentation.

In the first surgery, delayed sternal closure was carried out using draping to prevent disruption of heart contractions, which were still oedematous, and to monitor the occurrence of postoperative bleeding. Close monitoring was done 24 hours to monitor the hemodynamic status, saturation, electrolytes, blood gas analysis, and urine output. The assessment after 24 hours showed the patient was in stable condition. Therefore, the sternal closure was performed the following day.

Delayed sternal closure (DSC) is commonly performed after cardiac surgery in children to treat hemodynamic instability caused by cardiac compression due to sternum closure. Indications for DSC are variable and determined according to each individual. Some indications for DSC are based on hemodynamic status, lactate levels, myocardial edema, bleeding, need to repair the remains and the presence of pulmonary hypertension before the surgery or prevention of pulmonary hypertension in the postoperative period.[12,13]

DSC was shown to be a simple and effective technique for preventing postoperative cardiac compression and

has a role in patients with hemodynamic instability. Primary delayed sternum closure (PDSC) also shows better results than secondary delayed sternum closure (SDSC), where the sternum is closed initially and reopened. SDSC can be lifesaving, but the planned PDSC has the better outcome. DSC has the risk of infection in the sternum wound. However, the risk factors for DSC as a cause of infection are still specific and based on the patient's age and interval duration between DSC and definitive sternum closure.[12–14]

The patient in this case study suffered from severe pulmonary hypertension pre-operatively. Therefore, pulmonary hypertension management was carried out from before the surgery to after the surgery with antihypertensive medicine. The patient was diagnosed with pulmonary hypertension as a result of the first catheterization. Therefore, after the surgery, the patient was simultaneously given antihypertensive medicine. Before the surgery, the patient received Ramipril 1x0,5mg, Furosemide 2x5mg, and Sildenafil 3x5mg. In contrast, the medicine after surgery was continued with a regimen of Ramipril 1x0,5mg, Sildenafil 3x5mg, Furosemide 3x5mg, and Aldactone 1x6,25mg.

One of the causes of pulmonary hypertension is congenital heart disease and is often associated with the presence of a left-to-right-shunt. The presence of pulmonary hypertension can increase mortality and morbidity significantly in children. Therapy for pulmonary hypertension focuses on the specific medicine for pulmonary hypertension and supportive conventional medicine. Supportive conventional medications include diuretics, anticoagulants, and digoxin. Diuretics are generally used to relieve symptoms of right heart failure in patients with pulmonary hypertension associated with left ventricle diastolic dysfunction. In children, Hydrochlorothiazide and Furosemide can be used, sometimes combined with aldosterone antagonists such as Spironolactone. Digoxin is not routinely recommended and must be adjusted to individual cases.

Supportive treatment in the form of oxygen therapy is indicated in patients who are proven to have hypoxemia at rest (<92%) or during exercise. Oxygen will generally relieve symptoms in children with right heart failure due to pulmonary hypertension.[15]

Specific antihypertensive medications include calcium channel blockers (CCB), generally indicated in patients with pulmonary artery hypertension (PAH) who show positive acute pulmonary vasoreactivity testing (AVT) results. Amlodipine, Nifedipine, and Diltiazem are the most widely used drugs. Children with increased right atrial pressure (>8mmHg), right ventricular systolic dysfunction, or heart failure do not need to be given CCB. Verapamil was not given because of negative inotropic effects.[7,15]

PH-targeted therapy over the past 3 decades has improved in reducing mortality and morbidity rates in patients with pulmonary hypertension. The three molecular mechanisms this therapy targets are nitric oxide (NO), endothelin, and prostacyclin. The endothelium endogenously produces NO and causes smooth muscle in arterioles to relax and vasodilate by stimulating cyclic guanosine monophosphate. Exogenous NO can be administered via inhalation. PDE5 inhibitors are the most frequently prescribed long-term drugs and target the NO pathway.[4,5]

PDE-5 inhibitors such as Sildenafil and Tadalafil are drugs commonly used in children with pulmonary hypertension, with sildenafil being the most widely used drug.[15]

In this case, during the treatment in the ICU, the ventilator was slowly reduced until the patient was extubated, with a total ventilator time of 96 hours. After that, the patient was transferred to the HCU and inpatient ward for treatment. The final patient's status was blood pressure of 94/67mmHg, a heart rate of 92x/minute, a respiratory rate of 22x/minute, and oxygen saturation of 92% in four extremities without support. The urine output was 2.1cc/kg body weight/hour.

The patient was then sent home and was planned to undergo regular control with a catheterization evaluation in the sixth month to determine whether further action could be taken. The patient's control after 2 days showed stable hemodynamic status with echocardiography results showing the presence of a large VSD bidirectional dominant left-to-right shunt with PA to pG 35mmHg and good biventricular function.

CONCLUSION

There are several techniques used to handle patients with aortic coarctation accompanied by another cardiac defect, including VSD. As one-stage surgery has improved over the years, staged surgery is still used in some cases. In this case report, we use staged surgery in managing a patient with aortic coarctation accompanied by aortic arch hypoplasia, DCSA VSD, large PDA, and severe pulmonary hypertension. The first stage has been done with aortic coarctation repair and PA banding. During the procedure, we also use DSC to improve hemodynamic status. The surgery is also accompanied by the use of antihypertensive medicine to treat pulmonary hypertension. We aim to treat the patient's pulmonary hypertension and overall condition. The follow-ups show improved condition hemodynamically and the echo shows good biventricular function.

DISCLOSURES

Ethical Considerations

None.

Conflict of Interest

None declared.

Author Contribution

Suprayitno Wardoyo and Dhama Shinta Susanti were involved in concepting, designing, and supervising the manuscript. Stacia Manggala was involved in the data collection and drafting of the manuscript.

Funding

This work received no specific grants from any funding agency in the public, commercial, or non-for-profit sectors.

REFERENCES

1. Kouchoukos NT, Kirklin JW BBB. Kirklin: Morphology, diagnostic criteria, natural history, techniques, results, and indications. Philadelphia: Elsevier Saunders; 2013.
2. Law MA, Tivakaran VS. Coarctation of the Aorta. In Treasure Island (FL); 2024.

3. Plunkett MD, Harvey BA, Kochilas LK, Menk JS, St Louis JD. Management of an associated ventricular septal defect at the time of coarctation repair. Ann Thorac Surg. 2014 Oct;98(4):1412–8.
4. Abman SH, Hansmann G, Archer SL, Ivy DD, Adatia I, Chung WK, et al. Pediatric Pulmonary Hypertension: Guidelines From the American Heart Association and American Thoracic Society. Circulation. 2015 Nov;132(21):2037–99.
5. Ivy D. Pulmonary Hypertension in Children. Cardiol Clin. 2016 Aug;34(3):451–72.
6. Cinteza E, Nicolescu A, Filip C, Lupu A, Nicolae G, Duica G, et al. Pulmonary Hypertension in Children - a Practical Approach. Maedica (Buchar). 2015 Sep;10(3):237–42.
7. Friedberg MK. Aortic Coarctation is Right Out of Left Field: The Impact of Pulmonary Hypertension and Right Ventricular Dysfunction on Clinical Outcomes. Vol. 14, Circulation. Cardiovascular imaging. United States; 2021. p. 1109–11.
8. Gray WH, Wells WJ, Starnes VA, Kumar SR. Arch Augmentation via Median Sternotomy for Coarctation of Aorta With Proximal Arch Hypoplasia. Ann Thorac Surg. 2018 Oct;106(4):1214–9.
9. Wright GE, Nowak CA, Goldberg CS, Ohye RG, Bove EL, Rocchini AP. Extended resection and end-to-end anastomosis for aortic coarctation in infants: results of a tailored surgical approach. Ann Thorac Surg. 2005 Oct;80(4):1453–9.
10. Kaushal S, Backer CL, Patel JN, Patel SK, Walker BL, Weigel TJ, et al. Coarctation of the aorta: midterm outcomes of resection with extended end-to-end anastomosis. Ann Thorac Surg. 2009 Dec;88(6):1932–8.
11. Sakurai T, Stickley J, Stümper O, Khan N, Jones TJ, Barron DJ, et al. Repair of isolated aortic coarctation over two decades: impact of surgical approach and associated arch hypoplasia. Interact Cardiovasc Thorac Surg. 2012 Nov;15(5):865–70.
12. Elsisy MF, Dearani JA, Crestanello JA, Ashikhmina EA, Van Dorn CS, Stephens EH. Outcomes of Primary vs Secondary Delayed Sternal Closure in Pediatric Cardiac Surgery. Ann Thorac Surg. 2022 Apr;113(4):1231–7.
13. Elassal AA, Eldib OS, Dohain AM, Abdelmohsen GA, Abdalla AH, Al-Radi OO. Delayed Sternal Closure in Congenital Heart Surgery: A Risk-Benefit Analysis. Heart Surg Forum. 2019 Aug;22(5):E325–30.
14. von Stumm M, Leps Y, Jochheim L, van Rüth V, Gottschalk U, Mueller G, et al. Impact of delayed sternal closure on wound infections following neonatal and infant cardiac surgery. PLoS One. 2022;17(5):e0267985.
15. Gorenflo M, Ziesenitz VC. Treatment of pulmonary arterial hypertension in children. Cardiovasc Diagn Ther. 2021 Aug;11(4):1144–59.

This work is licensed under a Creative Commons Attribution