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The Outcome of Surgical Pulmonary Artery Banding in Patient with Complex Cyanotic Heart Disease Truncus Arteriosus Type I with Malalignment Ventricular Septal Defect: A Case Report

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1. Introduction

Truncus arteriosus is a cardiac malformation condition characterized by the presence of an arterial truncus that exits the heart through a single semilunar valve and goes directly to the coronary, systemic and pulmonary arteries. This condition is accompanied by the presence of a perimembranous infundibular Ventricular Septal Defect (VSD) under the truncus. The trunk valves may be bicuspid, tricuspid, or quadricuspid, and are often incompetent (Allen, 2001). This anomaly is rare, accounting for 2% of all congenital heart disease. This condition appears to result in congestive heart failure during infancy, up to the first few months of life. If good management is not carried out, about 70% of patients die within the first 3 months of life (Allen, 2001).

Diagnosis must be made comprehensively, starting from clinical assessment, to adequate supporting examinations. Non-invasive investigations such as echocardiography have become the main modality for diagnosing this condition, with addition of cardiac catheterization to confirm the diagnosis. Due to the large pulmonary flow and high pulmonary artery pressure, pulmonary vascular disease develops rapidly leading to increased mortality, exacerbated by the possibility of inoperability (Park, 2020). Conventional decongestive therapy is often inadequate to relieve the patient's condition during infancy. Therefore, surgical therapy in the form of pulmonary artery (PA) banding surgically or hemodynamic correction with an operative procedure with the Rastelli procedure are the alternative options. (Park, 2020).

This case report presenting a patient with truncus arteriosus type I (according to Collet and Edwards) with congestive heart failure and pulmonary hypertension. The patient conducted surgical PA banding to relieve the clinical condition. This report discusses from comprehensive diagnosis to management, including PA banding as the options of interventions and complications that occur after surgical intervention.

2. Case Illustrations

A 5 months old male baby, referred to the pediatric cardiac polyclinic at Prof. Hospital. Examined by a previous pediatrician due to suspicion of congenital heart disease during a physical examination. Patient with history of drinking milk intermittently since the age of 2 months. Appears to sweat excessively when drinking, is said to give the impression of rapid breathing and gets better on its own when not drinking breast milk. There are complaints of bluish skin a few days after birth, but it goes away on its own. There is a history of recurrent colds and fever since the age of 1 month, self-medication to the Primary Health Facility. Because these complaints continued to recur, the patient was referred to a pediatrician for further examination.

The patient was an only child, born normally assisted by a midwife, was said to be born crying immediately, history of fetal distress and rupture of membranes was denied. It is said the baby was born full term. Birth weight 2800 grams, birth length and head circumference are said to be forgotten. The patient's medical history was only given powder concoction medicine for his cold cough. History of diseases in the mother such as diabetes mellitus and hypertension, asthma, hepatitis B, TB, heart disease, and others during pregnancy was denied. History of exposure to heavy metals during pregnancy was denied. The use of drugs such as antihypertensives, antidepressants during pregnancy was denied by the mother. However, during pregnancy, the mother was exposed to cigarette smoke from her husband, who smokes every day. For the anthropometric status of the patient, the patient is malnourished, the average body weight is 5.1 kg, with an ideal body weight according to age of 6.5 kg (-3 – (-2) SD). The current body length is 52 cm, with an ideal age-appropriate body length of 55 cm (-1 - 0 SD).

At the time of examination the patient appeared calm, the face did not appear syndromic, there was no cyanosis of the oral mucosa or tongue. Pulse palpable 135 beats/minute, regular, strong lifting with axillary temperature 36.6C. upper and lower extremity saturation did not differ significantly 92-94% On physical examination the chest appeared symmetrical, there were no retractions. A single loud S2 heart sound was obtained, with a grade II/VI ICS II diastolic murmur on the left parasternal line. During examination, no abnormal lung sounds such as crackles or wheezing were found. On abdominal examination, there was no abdominal distention, the liver and spleen were not palpable, bowel sounds were normal. Examination of the extremities did not

reveal cyanosis, no clubbing finger, warm extremities and no leg edema.

On ECG examination (Figure 1) obtained with sinus rhythm with a rate of 135x/minute, normal axis. Lab tests were carried out with normal results from complete blood count, chemistry and electrolytes. AP chest x-ray examination (Figure 2) showed cardiomegaly with an enlarged right ventricle configuration, accompanied by pulmonary plethora and an impression of mediastinal widening.

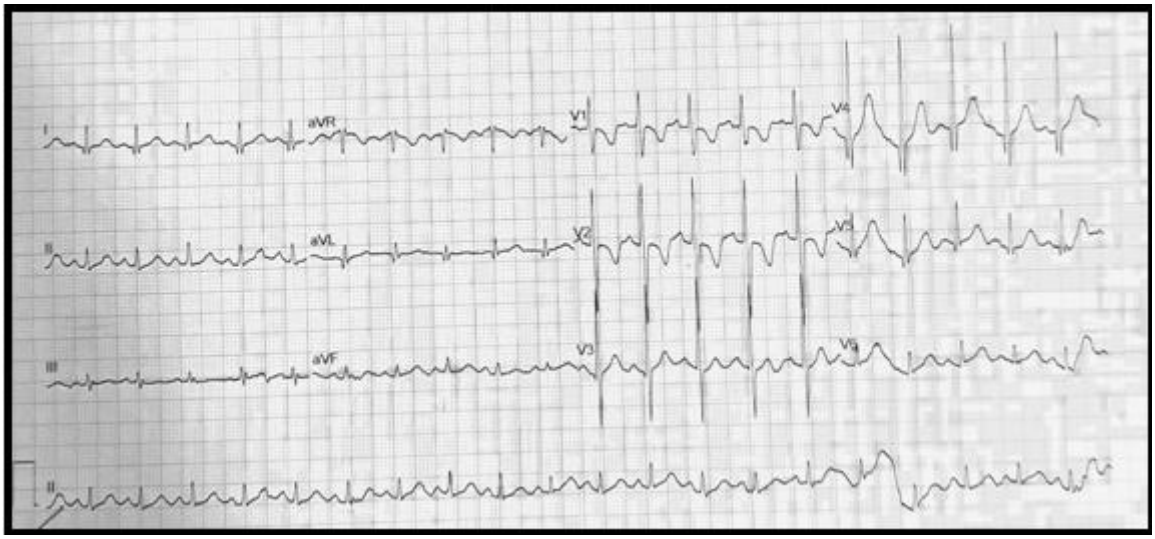


Figure 1: ECG. Normal sinus rhythm, Rate 135x/minute

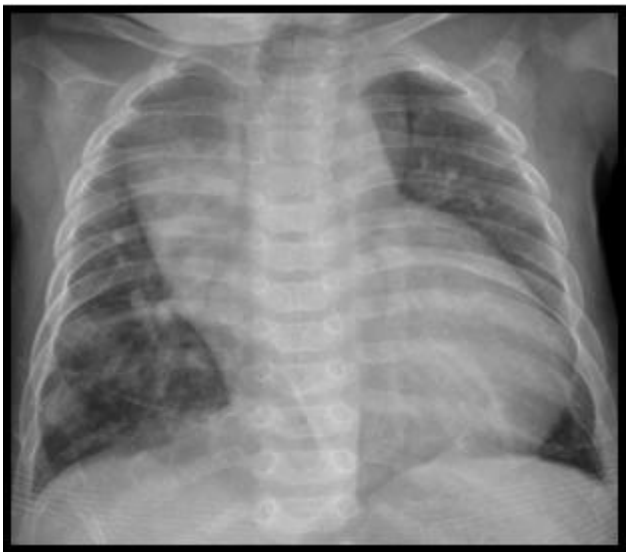
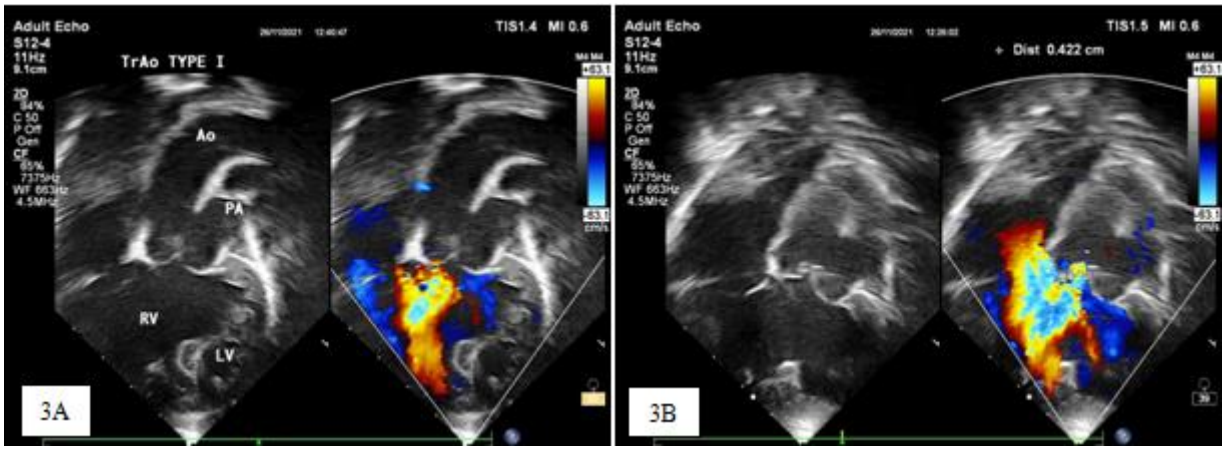


Figure 2: X-Ray Thorax AP. cardiomegaly wuth pulmonary plethora and widening mediastinum.

A transthoracic echocardiographic examination was performed (Figure 3) and obtained atrial situssolitus, normal systemic and pulmonary venous drainage, AV concordant, visible main pulmonary artery (MPA) originating from the ascending trunk with confluent pulmonary arteries. Also visible regurgitation of the truncus valve is moderate (vena contracta 0.42 cm). There is a large VSD malalignment with dominant left to right bidirectional shunts, with a diameter of 10 mm. Examination of left and right ventricular function was still within normal limits. So from echocardiography it suggests a truncus arteriosus type I (according to Collet and Edwards)



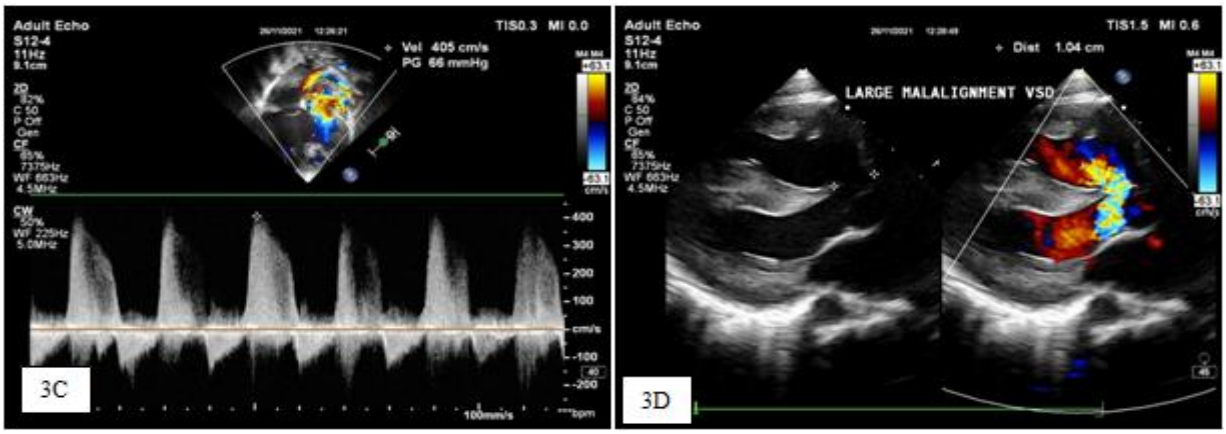


Figure 3: Transthoracis Echocardiography. **3A**. Truncus arteriosus type I seen with main pulmonary arteryoriginates from ascending truncus. **3B**dan**3C**. Moderate truncus regurgitation (VC 0.42 cm, PG 68 mmHg). **3D**. large VSD malalignment with bidirectional shuntsdominant left to right, with a diameter of 10 mm.

Based on the results of transthoracic echocardiography, the patient is planned for further examination cardiac catheterization. Results obtained from cardiac catheterization confirmed the diagnosis of truncus arteriosus type I with a patent foramen ovale (PFO) and pulmonary arterial hypertension (PAH). LV graphy was performed to obtain a large VSD, aortography and MPA graphy to confirm the findings of truncus arteriosus. In addition to assessing from an anatomical point of view, this examination also evaluates the pressure and saturation of each chamber of the heart and pulmonary arteries, so that a low flow ratio calculation (0.89) and moderate pulmonary artery resistance index (PARI) is obtained (5.27).

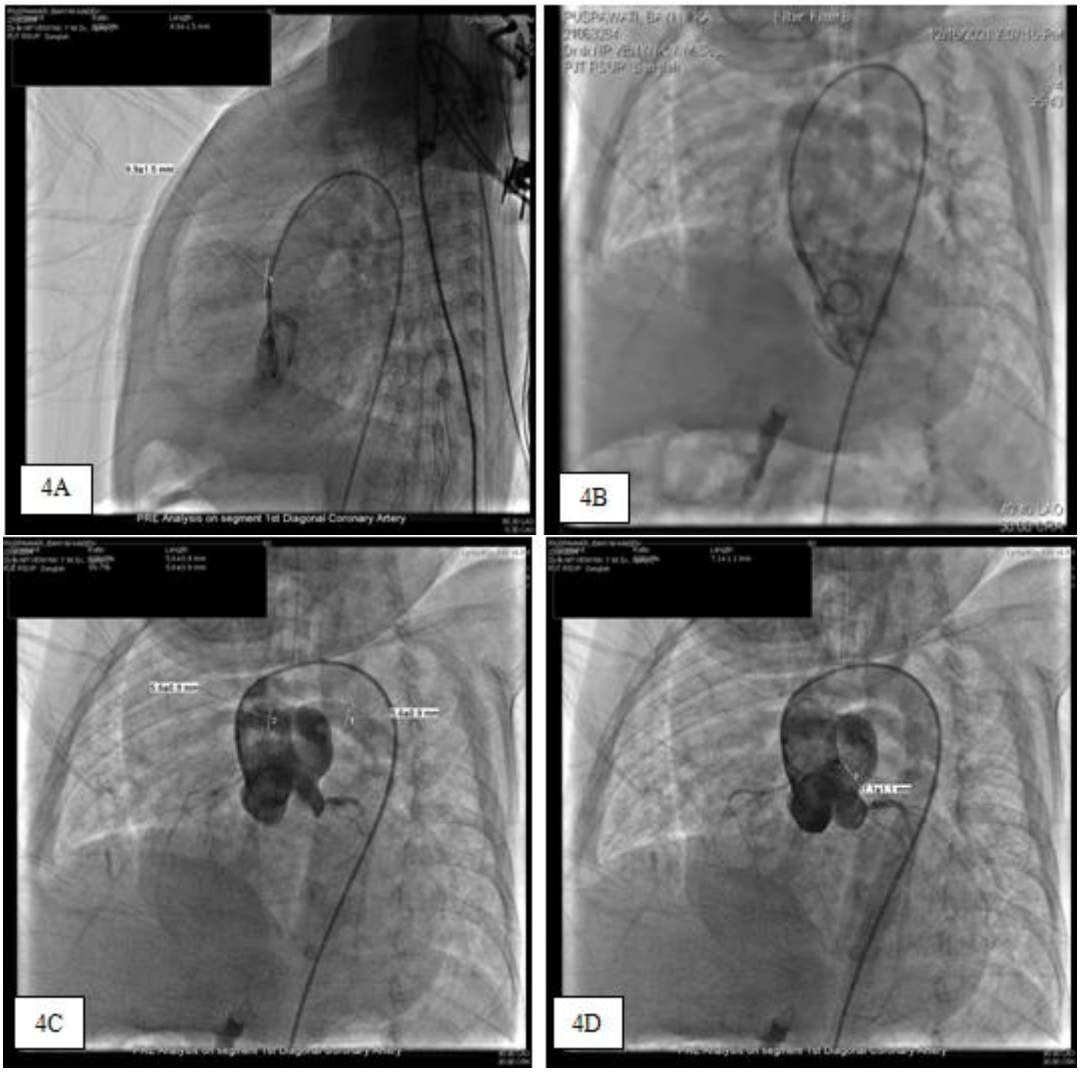


Figure 4: Cardiac Catheterization **4Adan 4B**. LV graphy showed large VSD (9.9±1.5 mm). **4C**dan**4D**. Aortography showed MPA originates from truncus ascendens, with confluents PA (RPA 5.6 ±0.9 mm, LPA 5.6 ±0.9 mm, MPA 7.1±1.1 mm)

After comprehensive diagnostic enforcement starting from anamnesis, physical examination and supporting examinations in the form of laboratory, EKG, chest x-ray, echocardiography and cardiac catheterization, it can be concluded that the patient has a complex cyanotic congenital heart disease in the form of truncus arteriosus type I, accompanied by clinical presence of congestive heart failure, PFO and pulmonary arterial hypertension. So that the patient was given treatment in the form of anti-heart failure with furosemide medication, spironolactone, captopril and digoxin.

The patient is also planned to have a joint conference with the cardiovascular thoracic surgery division for surgical intervention in the form of surgical PA Banding. The patient underwent surgical intervention a few weeks after the conference, with a PA banding procedure or pulmonary artery ligation aimed at reducing excessive flow to the pulmonary artery thereby delaying the development of severe pulmonary hypertension or pulmonary vascular disease (PVD). This procedure was carried out with a clinical evaluation during the operation, at that time a significant decrease in blood pressure and bradycardia was obtained when the 24 mm binding was performed, because the patient's condition had deteriorated, the patient also had a cardiac massage during the operation, when the condition was more stable it was decided to do a 27 mm binding, with plans to add anti-pulmonary hypertension drugs in the form of postoperative inhalation iloprost.

Re-evaluation of echocardiography after the procedure in the intensive care unit showed a moderate pulmonary stenosis gradient (pressure gradient 25.9 mmHg) due to PA banding, moderate regurgitation of the truncal valves, LV and RV systolic function was still good, with hemodynamics showing a decrease in cardiac output. Patient's condition improved with intensive monitoring. Clinical and echocardiography evaluation showed improvement of congestive state, clinically stable hemodynamic and after few days of intensive care, patient moved to general ward with good condition and patient was discharged with routine follow up.

3. Discussion

Epidemiology

Truncus arteriosus is a rare congenital heart malformation, accounting for approximately 0.7% of all complex congenital heart lesions. Truncus arteriosus usually occurs as an isolated cardiovascular malformation, although association with other organ anomalies has been reported, most commonly DiGeorge or velocardiofacial syndrome (chromosome 22q11.2 microdeletion). This anomaly has been reported in dizygotic twins, siblings, and relatives who have the disease. the same one. The truncal valve most often has three cusps or tricuspids (70% of cases), but may also be quadricuspid (21%) or bicuspid (9%); there have also been reported cases of having more than four cusps. Based on the classification by Collet and Edwards, truncus arteriosus has four types based on the origin of the pulmonary artery. Type I truncus has the highest prevalence, namely 48-68% of all reported truncus cases. Truncus type II has a prevalence of 29-48% and truncus type III 6-10% of all cases of truncus (Allen 2001). Most of the mortality in this patient population is caused by congestive heart failure and pulmonary hypertension, where the patient will fall into a state of heart failure at the age of approximately 3 months if not corrected (Cifareli, 2003).

Embryology

The embryonic truncus arteriosus lies between the conus cordis proximal to the aortic sac and distal to the aortic arch. Separation of the truncus arteriosus is closely related to conal and aortopulmonary septations. The enlarged trunk divides the trunk lumen into two channels: the proximal ascending aorta and the pulmonary trunk. As the proximal portion of the trunk septum fuses with the developing conal septum (originating from a conal swelling), a right ventricular origin from the pulmonary trunk and a left ventricular origin from the aorta are formed. The invagination of the roof of the aortic sac forms the aortopulmonary septum which eventually merges with the distal portion of the truncal septum. Thus, the right and left pulmonary arteries originate from the pulmonary trunk, and the aortic arch from the ascending aorta. This spiral path of the truncoaortic partition results in the normal course of the great arteries. If conotruncal or truncoaorticseptations do not progress normally, various congenital ventriculoarterial anomalies can occur. One of these anomalies is persistent truncus arteriosus, in which a single arterial trunk leaves the heart. In addition, conal (infundibular) septal deficiency results in a large VSD. Because the conal septum also contributes to the development of the anterior tricuspid leaflet and the medial tricuspid papillary muscle, these structures can also be malformed (Allen, 2001).

Pathophysiology and Clinical Manifestations

Truncus arteriosus is a cardiac malformation condition characterized by the presence of an arterial trunk that exits the heart through a single semilunar valve and goes directly to the coronary, systemic and pulmonary arteries. The pulmonary artery origin of this single artery can differentiate truncus arteriosus from pulmonary valve atresia (Park, 2020). Collett and Edwards divided the four types of truncus arteriosus based on the anatomical origin of the pulmonary arteries. In type I, the short trunk pulmonalis originates from the truncus arteriosus to form the two pulmonary arteries. It is said to be type II and III if the two pulmonary arteries are separated from the truncus arteriosus, if the two branches are close to each other then they correspond to type III, while type IV is if the two branches are far apart. Truncus arteriosus type IV is currently considered a form of pulmonary atresia with a defective VSD. Another classification described by Van Praagh with types A1 and A2 corresponds to the Collett and Edwards classifications I and II. The difference lies in Type A3 which is a type of truncus arteriosus without a truncal origin from a single pulmonary artery, with blood supply to the lungs from the ductus arteriosus or from collateral arteries. Finally, type A4 is associated with underdevelopment of the aortic arch, including tubular hypoplasia, discrete coarctation, or complete interruption I (Valpe, 2003).

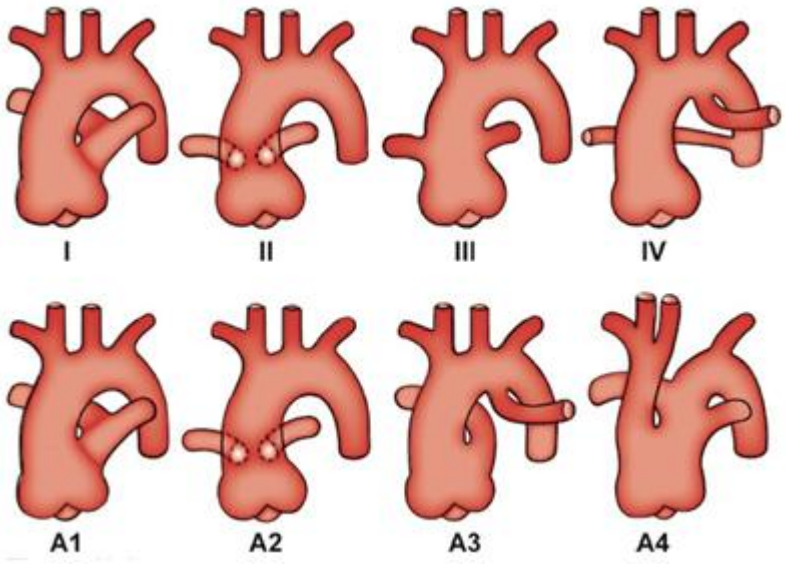


Figure 5: Truncus Arteriosus Classification. **Upper part.** Collet dan Edwards Classification. **Lower part**. Van Praagh Classification

The pathophysiology of truncus arteriosus is characterized by cyanosis and systemic ventricular volume overload. This malformation causes clinical manifestations especially occurring in the first few months of life. Early in the first weeks of life, there is still a persistent increase in pulmonary arteriolar resistance from the fetal stage, so mild cyanosis may occur with little evidence of cardiac decompensation. It occurs as a result of the common mixing of left and right ventricular output that occurs mainly during systole and at the truncal level. As the pulmonary resistance gradually decreases and the flow through the lungs increases, the cyanosis may decrease until it eventually disappears. However, tachypnea, tachycardia, excessive sweating, decreased nutrition, and signs of overflow in the pulmonary circulation may occur (Braunwald, 2001). Signs and symptoms of heart failure may also appear soon after birth if there is severe truncal valve insufficiency. Due to parallel systemic and pulmonary circulations, pulmonary blood flow can be at least 3 times higher than systemic blood flow, excessive pulmonary circulation and increased myocardial work increase resting oxygen requirements and decrease metabolic reserves. The more severe there is an excessive increase in volume load and dilatation of the cardiac chambers, aggravating cardiac decompensation. In infants with pulmonary artery stenosis, cyanosis may appear at birth and may worsen with age. However, stenosis can protect the child from excessive circulation in the lungs which would otherwise decrease pulmonary resistance. In patients with pulmonary artery stenosis and severe truncal valve insufficiency, severe cyanosis may occur, in addition to early signs of heart failure (Braunwald, 2001).

In this case, it is illustrated that a five-month-old patient has clinical congestive heart failure, characterized by tachypnea, diaphoresis, and failure to thrive. This condition can result from an excessive increase in pulmonary flow, which occurs with type I of the truncus arteriosus. The patient's condition is also exacerbated by truncus regurgitation which can cause an increase in myocardial workload and thus increase oxygen demand. The patient also has a history of blueness during the first weeks of life, but it gradually disappears on its own. This condition is compatible with the pathophysiology of truncus arteriosus where in the early weeks of life, pulmonary resistance is still high, and there is a pathophysiology of common mixing which causes cyanosis. As the baby grows, pulmonary resistance decreases, until finally the cyanosis decreases and disappears

Diagnosis Approach

The principle of establishing a diagnosis in this patient population must be carried out thoroughly from a general clinical evaluation and a specific diagnostic examination. In utero diagnosis of truncus arteriosus can be made as early as the first trimester. However, despite improvements in prenatal diagnosis, the rate of prenatal diagnosis of truncus arteriosus remains low (<50%). In addition, the distinction between truncus arteriosus and other congenital heart defects such as VSD, tetralogy of Fallot and pulmonary atresia is still difficult to distinguish in prenatal diagnostics (Taylor, 2000). Diagnostic approach starts from a comprehensive and directed anamnesis, in general from growth and development, nutritional or nutritional status, disease course and clinical symptoms must be properly evaluated. Evaluation of growth and development and nutritional status is very important, this data is essential in addition to supporting the presence of systemic disease, this data can also be a benchmark for the patient's prognosis. Patients with poor nutritional status can be characterized by disease progression that is already aggravating. In scoring heart failure with Ross' criteria, it is said that the patient has reached the criteria for moderate heart failure if he has experienced failure to thrive (table 1) (Park 2020).

The physical findings are closely related to the increased volume of pulmonary blood flow and the presence or absence of truncal valve insufficiency. Patients with increased pulmonary blood flow have little or no cyanosis. The peripheral pulse is palpable, the pulse pressure is usually increased because of runoff or runoff into the pulmonary vasculature during diastole, and is exacerbated by truncal valve insufficiency. On palpation examination

can also feel the thrill on the precordial. On auscultation of the heart, a single loud S2 heart sound was found, an S3 gallop could also be heard. Murmur examination can be identified by the presence of a loud pansystolic murmur on the left sternal line and radiating to the precordium. The patient with truncal valve insufficiency usually hears a high-pitched early diastolic murmur that is best heard along the left paraceternal line. Patients with heart failure may exhibit clinical signs typical of heart failure such as tachypnea, crepitus crackles, hepatomegaly, and distended neck veins. Meanwhile, signs of cyanosis or clubbing finger occur in patients with early presentation or patients who do not receive fast treatment resulting in pulmonary vascular disease (Allen 2001).

A simple but very important follow-up examination is the chest x-ray, in which the chest radiographic presentation can be a very essential finding. This is assessed by the presence of cardiomegaly and pulmonary vascular enhancement or plethora which commonly occurs. The aortic arch is on the right side in about one-third of patients, and the combination of the right aortic arch and increased pulmonary vascularity suggests a tendency to truncus arteriosus. Truncus arteriosus type I is often associated with a relatively superior position of the proximal left pulmonary artery, which can usually be distinguished on a frontal chest radiograph. The electrocardiogram (ECG) usually shows a normal QRS axis or minimal right deviation. In general, the EKG shows normal sinus rhythm, with normal conduction time. Generally often accompanied by biventricular hypertrophy. The presence of prominent left ventricular voltage is prominent in patients with increased pulmonary blood flow. In patients with normal or decreased pulmonary flow usually shows right ventricular hypertrophy (Park 2020).

Use of both 2D and color Doppler echocardiography is helpful in accurately determining cardiac anatomy, including cardiac function and hemodynamics. Echocardiography with cross-sectional analysis and flow Doppler is sufficient to confirm the diagnosis of truncus arteriosus and fully characterize the various anatomical features in most patients. Echocardiographic images are typical of the truncus arteriosus through visualization of the ascending truncus connection with the pulmonary artery.This image is obtained from the subcostal and apical views.Echocardiography can also evaluate for regurgitation and stenosis of the truncal valves, as well as assessing qualitatively and quantitatively.On the parasternal view can also assess VSD defects, including size, shunts and pressure gradients generated.With advances in echocardiography diagnostic tools that are accurate and measurable increased attention to surgical correction during early infancy before irreversible pulmonary vascular disease occurred.

Diagnostic cardiac catheterization and angiography are not always performed in patients with truncus arteriosus. However, cardiac catheterization can still be considered considering that pulmonary vascular disease can also occur early. Cardiac catheterization may be the modality of choice in truncus arteriosus patients during infancy to determine pulmonary vascular status prior to surgical intervention. Patients with truncus arteriosus who have two pulmonary arteries and a pulmonary arteriolar resistance of >8 units m2 are at a higher operative risk than patients with resistance below this level. Among the group with resistance >8 units m2, higher mortality was due to the progression of pulmonary vascular obstructive disease with secondary severe pulmonary hypertension and right ventricular failure (Cifareli, 2003). Rarely, patients with truncus arteriosus with associated compromise of the aortic arch or single pulmonary artery will require angiography to adequately delineate the aortic arch or pulmonary artery branch anatomy. In neonates, contrast cardiac CT scan is preferred to define anatomic problems not clearly demonstrated on echocardiography or to assist in surgical planning. In addition, magnetic resonance imaging (MRI) can also be used as an alternative modality in diagnosing truncus arteriosus. Cardiac MRI and CT scans can delineate sagittal and transverse views in the heart floor demonstrating the trunks of the great arteries with truncal valves covering the interventricular septum and the origin of the pulmonary arteries (Fig. 7) (Allen 2001).

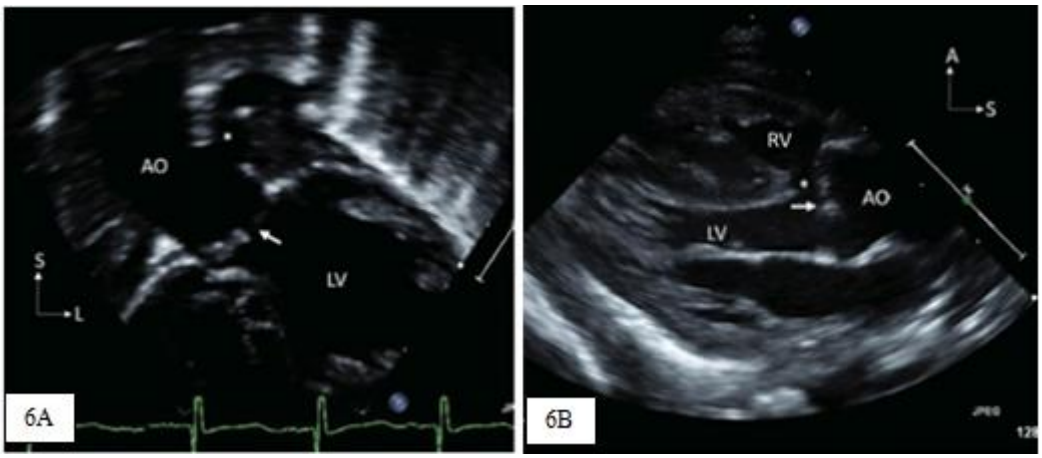


Figure 6: Transthoracic echocardiography. **6A**. Subcostal view with anterior angulation in a newborn with truncus arteriosus type I. Arrows indicate thickening of the truncal valves; aortic root (Ao) continues anteriorly/superiorly. **6B**. Long axis parasternal features in the newborn are similar to those of type I truncus arteriosus, with thickened truncal valves (arrow) and ventricular septal defect (asterisk) with thickened truncal valves (arrow).

In this case report, we found a patient with clinical heart failure and an excessive increase in pulmonary flow. Marked by clinical manifestations and supporting examinations in the form of plethora on chest X-ray. Subsequent investigations confirmed the findings. Echocardiographic examination showed the presence of MPA originating from the ascending trunk with confluent pulmonary arteries. Also visible regurgitation of the truncus valve is moderate (vena contracta 0.42 cm). There is a large VSD malalignment with dominant left to right bidirectional shunts, with a diameter of 10 mm. Examination of left and right ventricular function was still within normal limits. The patient then underwent cardiac catheterization to confirm anatomic abnormalities and evaluate pulmonary vascular status. Results obtained from cardiac tapping confirmed the diagnosis of truncus arteriosus type I with a patent foramen ovale (PFO) and pulmonary arterial hypertension (PAH). LV graphy was performed to obtain a large VSD, aortography and MPA graphy to confirm the findings of truncus arteriosus. From the results of evaluating the pressure and saturation of each chamber of the heart and pulmonary artery, it is obtained that the calculation of the flow ratio is low and the PARI is moderate.

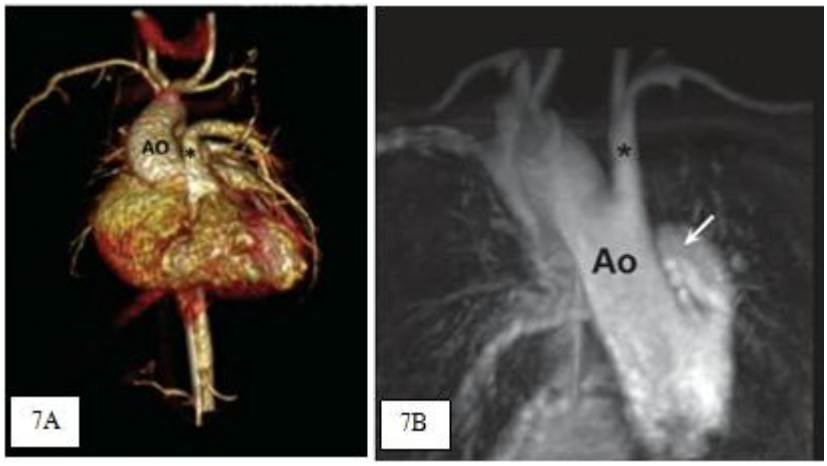


Figure 7: 7A. CT Angiography with 3D reconstruction, anterior view showing a type I trunk with a segment of the main pulmonary artery (asterisk) emerging from the aorta (AO). **7B**. MRI images with gadolinium contrast in a patient with type II truncus arteriosus. Anteroposterior view with pulmonary artery branches (arrows) originating from the left proximal aorta and showing right aortic arch view

The best treatment for this case so far is surgical intervention. Although medical therapy can help improve the patient's congestion problem, this treatment is not the main target. Total correction is highly recommended in the first weeks of life, by optimizing medical therapy in the intensive care unit after surgery can reduce patient mortality. Delay in surgery can cause chronic ischemia in the myocardium which is hypertrophied due to desaturated blood perfusion due to low diastolic perfusion. This results from pulmonary artery runoff and trunk regurgitation. This ventricular dysfunction explains why patients aged 6 to 12 months have a higher mortality rate than those at younger ages. Pulmonary vascular obstructive disease may also develop earlier, so early correction is highly recommended. Pulmonary vascular obstructive disease is strongly associated with increased surgical mortality in infants who undergo repair after 6 months of age (Lund, 2011).Various modifications of the Rastelli procedure can be performed. Ideally, surgery should be performed within the first week of life. When diagnosis is delayed, surgery should be performed as soon as 2 to 3 days after clinical stabilization. For all types of truncus arteriosus, the VSD must be closed until the left ventricle has supplied the truncus. The presence of coronary artery anomalies and prevention of coronary artery intervention must be evaluated, this must be considered by the surgical operator (Rajasinghe, 1997). For type I, the operator can use an aortic homograft or conduit with an internal diameter of 9-11 mm placed between the RV and PA. For types II and III, excision or removal of the abnormal pulmonary artery branch can be performed, and connecting the conduit between the right ventricle and pulmonary artery. Then aortic reconstruction was performed with proximal and distal aortic anastomoses. Additionally, truncal valve repair or replacement may be indicated in significant truncal valve insufficiency.

PA Banding Outcome and Prognosis

The management of surgical correction should be based on a clear conference. Discussion between the surgical and cardiology departments is essential to determine the next course of action. The preferred operation is total correction during the neonatal period. In truncus arteriosus, the pulmonary vessels are subjected to very high flows and pressures and pulmonary vascular disease tends to develop rapidly. These patients should undergo repair in early infancy, preferably before the age of 3 or 4 months (Konstantinov, 2006).

Pulmonary artery banding is a palliative surgical procedure used to treat functionally univentricular hearts, multiple ventricular septal defects and complete atrioventricular septal defects. Pulmonary artery banding has recently gained in interest for left ventricular retraining and hypoplastic left heart malformations. The indication for pulmonary artery banding is limited by several factors: difficulty of determining optimal band tightness, influence of perioperative variables with mutual interference, agerelated variability of the ventricular adaptive response, difficulty of securing sufficiently tight banding in older children, repeated surgical procedures needed to adjust the band

perimeter, long periods under intensive respiratory and/or pharmacological support, and the frequent need for reconstruction of the pulmonary artery at intracardiac repair.

Although surgical PA banding can provide palliation for young patients with truncus arteriosus, there are significant risks and complications. In addition, successful appeals do not guarantee that these patients will be good candidates for later correction. From some literature it is evident that even when patients undergo banding in infancy, only 30% will be able to reach this age to have a complete correction. Similarly, 30% of patients with large VSDs who were operated on after 2 years of age developed pulmonary vascular disease. Even after banding, progression of pulmonary vascular obstructive disease occurs in approximately 10% of patients until they are inoperable. Some data show that patients with right pulmonary artery-ventricular discontinuity who require a conduit or conduit can be operated safely in infancy with a mortality of 10% to 20%, which is much better than pulmonary artery banding followed by complete repair (Lund, 2011).

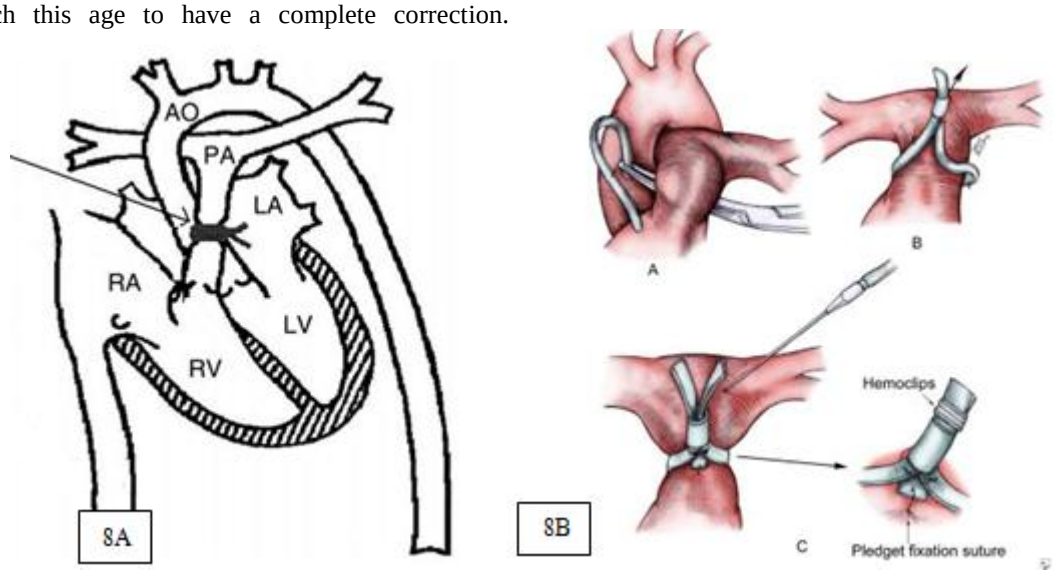


Figure 8: 8A-B. Operative technique of Pulmonary Artery Banding / Ligation

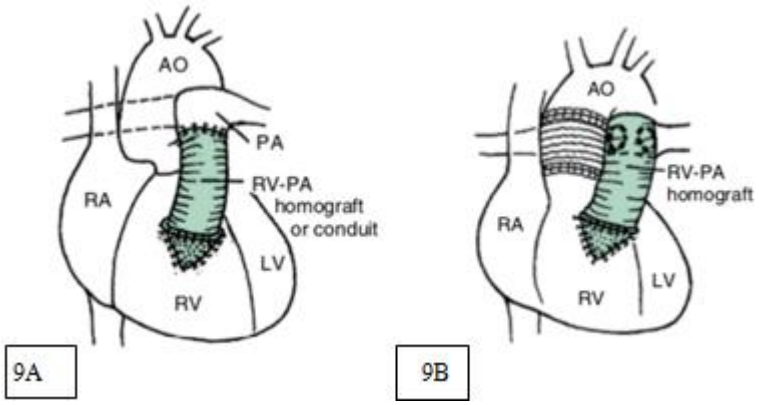


Figure 9: Operative technique on truncus arteriosus. **9A**. In type I truncus arteriosus, using a homograft or conduit directly connects the right ventricle and pulmonary artery. **9B**. In truncus arteriosus types II and III, the pulmonary artery branch is excised and repaired and then connected to a homograft or conduit.

In this case report, a conference was held between surgeons and cardiologists, to determine the next course of action. The patient was decided to undergo surgical PA appeal based on the results of the conference decision. The choice of this action was based on limited facilities and experience at Sanglah Hospital. There are several complications during the operation. There was a significant decrease in blood pressure and bradycardia until cardiac resuscitation was performed in the form of cardiac massage. After the procedure, the patient was closely observed in the intensive care unit, with the plan to add anti-pulmonary hypertension medication in the form of postoperative inhaled iloprost. Re-evaluation of echocardiography after the procedure showed a moderate pulmonary stenosis gradient (pressure gradient 25.9 mmHg) due to PA banding, there was moderate regurgitation of the truncus valve. As stated in some literature, pulmonary vascular disease is more difficult to predict and can occur more quickly. This depends on the severity of pulmonary outflow, the patient's systemic condition or the presence of comorbidities. The literature also says that surgical PA banding has a high mortality rate, accounting for up to 50% to 70%. It has been suggested that pulmonary artery occlusion can lead to rapid and severe hemodynamic instability. Treatment after surgery in the intensive care unit with close monitoring is highly recommended in this patient population, and may be the key to successful treatment. So that careful preparation, including preparation for post-action such as anti-pulmonary hypertension drugs is essential (Volpe, 2003).

4. Summary

A 5 months old male baby has been reported with a history of intermittent breastfeeding since 2 months of age. On physical examination, clinical mild heart failure was found. Follow-up examinations including echocardiography and cardiac catheterization confirmed the diagnosis of truncus arteriosus type I (according to Collet and Edwards) and were given medical treatment for heart failure but the symptoms still persist. Furthermore, the patient underwent surgical PA banding with good result. Patient's condition improved with intensive monitoring. Clinical and echocardiography evaluation showed improvement of congestive state, clinically stable hemodynamic and after few days of intensive care, patient moved to general ward with good condition and patient was discharged with routine follow up. Although according to literatures PA Banding is a risky procedure, from this case report it can also be learned that careful preparation from before to after surgery is needed, including the preparation of medicines that are important in patient's management, thusproviding optimal and comprehensive management.

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