Indonesian Journal of Chemical Science and Technology (IJCST)

State University of Medan, https://jurnal.unimed.ac.id/2012/index.php/aromatika

IJCST-UNIMED 2026, Vol. 09, No. 1, Page; 180 – 197

Received : Jan 24th, 2026 Accepted : Jan 30th, 2026 Web Published : Jan 31st, 2026

Right Heart Failure Atrial Septal Defect (DSA) Secondary With Tricuspid Regurgitation

Doharjo Manullang[1*], Menang B. Tarigan[2], Salomo G.U. Simanjuntak[3], Ivonne RVO Situmeang[4], Evi Rosa Simanjuntak[5]

[1,2,3,4,5] Faculty of Medicine, Methodist University of Indonesia

*Corresponding author : doharjomanullang@yahoo.com

ABSTRACT

Congenital heart disease is a structural or functional abnormality of the cardiovascular system that is present at birth, although it can be found later in life. And the prevalence of ASD is around 9.8%. ASD is a condition where damage occurs in the inter-atrial septum, causing a connection between the left and right atrium. ASD is divided into 4 types: secundum type Atrial Septal Defect, primum type Atrial Septal Defect, sinus venosus type Atrial Septal Defect, and finally coronary sinus type Atrial Septal Defect. Secundum Atrial Septal Defect is the most common type. And generally, more often occurs in women than men with a ratio of 2:1. And the examination modalities in establishing Atrial Septal Defect include Chest X-ray, Electrocardiography and Echocardiography. The following is a case report of a 41-year-old man diagnosed with Secundum Atrial Septal Defect (ASD) + TR. This patient was diagnosed at the age of 41. In its natural course, ASD will cause pulmonary artery pressure to increase with age. ASD closure before age 24 can reduce the risk of long-term mortality to the same level as the ASD-free population. This case study aims to provide insight into diagnosis, management, and patient education to achieve optimal therapy and quality of life.

Keywords: *Atrial Secundum Defect, Classification, Diagnostic Tests and Treatment*

1. INTRODUCTION

Congenital heart disease is a structural or functional abnormality of the cardiovascular system that is present at birth, although it may be discovered later in life. The actual incidence of cardiovascular abnormalities is difficult to determine accurately, as some conditions are not detected at birth, such as bicuspid aortic stenosis and mitral valve prolapse. The incidence of adult congenital heart disease (ACHD) in the form of atrial septal defect (ASD) is 9.8%.[1]

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Atrial septal defect is a condition in which there is damage to the septum between the atria, causing a direct connection between the left and right atria. There are two main types of ASD, namely secundum atrial septal defect and sinus venosus type. Secundum atrial septal defect is more common in females with an incidence ratio of 2:1 between females and males, while in the sinus venosus type, the incidence ratio is 1:1. ASD often goes undetected until adulthood because it tends to be asymptomatic and does not show any specific physical symptoms. Usually, ASD is discovered incidentally through routine examinations such as chest X-rays or echocardiography. The most common early symptoms involve shortness of breath and fatigue, along with frequent lung infections. [1]

In managing secundum atrial septal defect (ASD), a holistic approach tailored to the patient's condition is key to providing effective care. Routine monitoring, clinical evaluation, and symptomatic management are the first steps in understanding the patient's condition. Symptomatic therapy, prophylactic antibiotics, and interventional treatments, including the use of device occluders via cardiac catheterization, may be options depending on each patient's needs. Surgical procedures, such as patch placement on the atrial septum, may also be a solution. Cardiac rehabilitation, periodic follow-ups, patient education, and psychosocial support are important parts of providing holistic care. Infection prevention, particularly through good dental care, is also an aspect that should not be overlooked. Overall, the management of secundum ASD should be based on collaboration among a medical team involving cardiologists, surgeons, and other healthcare professionals, with a focus on the patient's physical recovery and psychosocial well-being. [1,2]

The following is a case report of a 41-year-old man with Secundum Atrial Septal Defect (ASD). In its natural course, ASD will cause pulmonary artery pressure to increase with age. Closure of ASD before the age of 24 can reduce the risk of long-term mortality to match that of the population without ASD. This case is presented to enhance understanding in diagnosing, managing, and educating patients to achieve optimal therapy and quality of life for this patient, considering that he was only diagnosed at the age of 41, where mortality risk may increase if proper management and follow-up are not conducted in the future. Hopefully, this case can add to our collective knowledge.

II. Definition

Atrial Septal Defect (ASD) is a congenital heart disease in which there is a hole connecting the left and right atria, causing shunting from the left atrium to the right atrium with an increase in volume load in the right atrium and ventricle, which can persist into adulthood.[1]

III. Epidemiology

Atrial septal defect is one of the most common types of congenital heart defects, occurring in approximately 25% of children. This congenital heart disease is often discovered in adulthood because it is often asymptomatic in infants and children. The prevalence of congenital heart disease and ASD has increased over the past 50 years. In the 1930s, congenital heart disease was diagnosed in less than 1 per 1,000 live births. In recent years, congenital heart disease has been diagnosed in 9 per 1000 live births. Atrial septal defect was identified between 1945 and 1949 in less than 0.5 live births per 1000 live births. More recent epidemiological data show that ASD occurs in 1.6 per 1000 live births.2. Congenital heart disease is more frequently diagnosed in patients in developed countries with higher incomes.[16]

Although atrial septal defects occur as single defects, ASDs are associated with Mendelian inheritance, aneuploidy, transcription errors, mutations, and maternal exposure. Atrial septal defects are found in patients with Down syndrome, Treacher-Collins syndrome, absent radius-thrombocytopenia syndrome, Turner

e-ISSN : 2622-4968, p-ISSN : 2622-1349

syndrome, and Noonan syndrome; these syndromes occur due to Mendelian inheritance. Maternal exposure to rubella and drugs, such as cocaine and alcohol, can also affect the unborn fetus, leading to ASD. .[3]

Atrial septal defect occurs in conjunction with other congenital heart defects, namely ventricular septal defect. In some patients with congenital heart disease, communication between the left and right heart circulations is vital for survival. [4]

ASD in adults is mostly detected in the fifth decade of life. At age 40, 70% of untreated patients will experience symptoms of shortness of breath during activity, fatigue, evidence of congestive heart failure, pulmonary hypertension and Eisenmenger syndrome, atrial arrhythmia, or stroke associated with paradoxical embolism.[1,4]

IV. Classification

Secundum ASD is the most common type of defect (incidence rate of 80%). This abnormality occurs in the fossa ovalis due to one or more defects in the primary septum. Small secundum ASDs need to be distinguished from patent foramen ovale because recent studies have shown that patent foramen ovale is not an abnormality of the atrial septum tissue. Patent foramen ovale is a condition in which there is a tunnel between the primum septum and the secundum septum located in the anterosuperior part of the atrial septum. A patent foramen ovale defect is a subclass of a secundum ostium defect, so the foramen ovale is not a defect in the "actual septum." Most secundum defects do not fuse with the veins, coronary sinuses, or atrioventricular valves.[5,21]

1. Secondary ostium defect

This defect occurs when there is increased reabsorption of the primary septum in the atrial roof, or the secondary septum does not block the secondary ostium. Secondary ostium defects are associated with pediatric syndromes such as Treacher-Collins syndrome and absent radius-thrombocytopenia syndrome.6

The natural history of atrial septal defects varies according to their anatomical form. In secundum defects, spontaneous closure often occurs. In a large retrospective study involving 200 consecutive children, who were followed up for 6 months, 34% of defects closed spontaneously and 28% reduced to a diameter of 3 mm.7

Based on similar observations, Radzik et al. suggest that infants born with atrial septal defects less than 3 mm do not require cardiac follow-up because spontaneous closure may occur. Conversely, for atrial septal defects larger than 8 mm, families should be informed about the possibility of the need for closure at a later date, either percutaneously or surgically, and therefore require regular echocardiographic monitoring.8

For secundum atrial septal defects that do not close spontaneously, the diameter may change over time. A total of 104 patients, with an average age of 4.5 years, were examined at Texas Children's Hospital over a period of 3 years. The size of the defects varied; 33% were small (between 3 and 6 mm), 38% were medium (between 6 and 12 mm), and 29% were large (12 mm or more). The diameter of the atrial septal defect increased in 68 patients (65%), with 31 patients (30%) experiencing an increase of at least 50%. Additionally, 13 patients had defects that grew to over 20 mm.[20]

2. Primum ostium defect

Also known as partial atrioventricular septal defect, with an incidence of 15% of all ASDs, it is the third most common atrial septal defect that occurs due to the failure of the primum septum to fuse with the endocardial cushion. This defect occurs between the inferior border of the fossa ovalis and the atrioventricular valve, without any defect in the ventricular component. The atrioventricular valve in this defect is almost always abnormal.[6,21]

3. Sinus venosus defect

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Superior and inferior defects occur, and neither involves the actual membranous septum. This defect is usually found at the mouth of the vena cava. The most common is superior venous sinus defect (incidence 5%) due to the lack of tissue separating the right pulmonary vein from the superior vena cava. The pulmonary vein from the right lung is often connected to the superior vena cava in venous sinus defects.

4. Coronary sinus defect

The coronary sinus is a blood vessel that runs along the groove between the left atrium and left ventricle and collects veins that represent the venous return of the heart muscle. It normally flows into the base of the right atrium above the leaflet of the tricuspid valve. A defect or hole in the common wall between the left atrium and the coronary sinus (called "unroofing" of the coronary sinus) creates communication between the right and left atria.

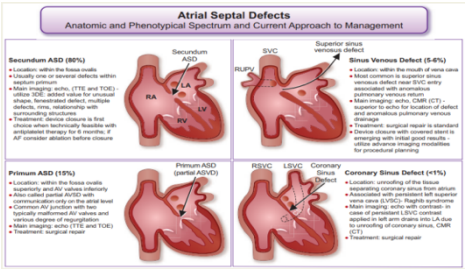


Figure 1. Classification of Atrial Septal Defects

V. Diagnosis

Most patients with secundum ASD have asymptomatic symptoms, especially during infancy and early childhood. Growth and development are usually normal, but if the shunt is large, growth may be impaired. In older children, intolerance to some physical exercises may be observed. If the defect is large enough, patients may experience shortness of breath and frequent lung infections. Physical examination of patients with ASD reveals a normal first heart sound (S1), a wide fixed split second heart sound (S2), and a systolic ejection murmur heard in the pulmonary area (mid-left and upper sternal line).[7,8,9,10.]

Many ASDs remain undiagnosed until adulthood, therefore treatment, especially for large defects, is often delayed. Untreated large defects can cause fatigue and shortness of breath during activity, cardiac arrhythmias, palpitations, increased incidence of pneumonia, pulmonary hypertension, and increased mortality.15 Eisenmenger syndrome is rare, but it is a complication of untreated ASD. When vascular resistance increases, right atrial pressure approaches systemic pressure. When right atrial pressure exceeds systemic pressure, shunt flow reversal occurs. [18]

Patients with defects smaller than 5 mm may not experience any symptoms, while patients with heart abnormalities measuring between 5 and 10 mm will develop symptoms in their fourth or fifth decade of life. Patients with larger defects develop symptoms earlier, in their third decade of life. Approximately 20% of adult patients experience atrial tachyarrhythmia before surgery. Evidence of stroke or transient ischemic attack, especially after diagnosis of peripheral blood clots, should raise suspicion of ASD.[11]

Supporting tests that can be performed to aid in the diagnosis of ASD include chest X-rays, electrocardiography, and echocardiography. Chest X-rays may show a prominent right atrium with a prominent pulmonary cone. Chest X-rays may also show a slightly enlarged heart and increased pulmonary vascularization corresponding to the size of the shunt. Chest X-ray findings are not particularly helpful in

e-ISSN : 2622-4968, p-ISSN : 2622-1349

diagnosis, but chest X-rays can help monitor clinical status by identifying cardiomegaly and enlargement of the pulmonary artery. Enlargement of the heart is often seen on lateral chest X-rays because the right ventricle protrudes anteriorly as its volume increases. [2,6,7]

Transthoracic echocardiogram is the gold standard imaging modality. This examination allows one to detect the size of defects, see the direction of blood flow, find related abnormalities (involvement of the endocardial cushion and atrioventricular valves), examine the structure and function of the heart, estimate pulmonary artery pressure, and estimate the pulmonary/systemic flow ratio (Qp/Qs).[11]

Although echocardiography is the gold standard for evaluating ASD, other diagnostic modalities that can be performed are cardiac CT and MRI. Exercise testing can help determine the reversibility of shunt flow and the response of patients with pulmonary arterial hypertension to activity. [11]

An electrocardiogram can show right bundle branch block (RBBB) in 95% of cases of secundum ASD, indicating right ventricular volume overload. In secundum ASD, there is a right axis deviation of the QRS complex. Right ventricular hypertrophy may be found, but enlargement of the right atrium is rarely found.[2,6,7] Color Doppler echocardiography can clearly show the shunt from the left atrium to the right.2,3 Patients with ASD do not always show abnormalities on physical examination and characteristic electrocardiogram results, so echocardiography is needed to show the presence of left-to-right shunting in ASD.[2,6,7]

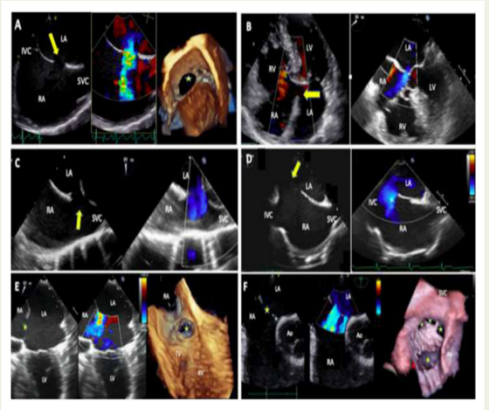


Figure 2. Echocardiography image of atrial septal defect

VI. Management

The main management of ASD that cannot close spontaneously is to close the defect. Closing the ASD will eliminate the left-to-right shunt that causes reduced heart function, reduce the volume load on the right atrium and ventricle, and restore pulmonary blood flow to normal.8 The management options for closing ASD are surgical intervention and non-surgical cardiology intervention.

Patients with atrial septal defects measuring less than 5 mm often experience spontaneous closure of the defect within the first year of life. Defects larger than 1 cm are likely to require medical/surgical intervention to close the defect.12 Monitoring of adult patients with small defects and no signs of right heart failure is essential. Echocardiography every 2 to 3 years can be performed to evaluate right heart function and structure. A history of TIA or stroke requires more aggressive monitoring and possible surgical intervention.

If ASD requires closure, options include percutaneous and surgical interventions. Indications for treatment include stroke, significant hemodynamic shunting, and evidence of systemic oxygen desaturation.

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Percutaneous transcatheter closure poses less risk to patients, but is only useful for closing secundum ostium defects. Percutaneous transcatheter ASD closure has a 7.2% risk of post-procedural complications compared to a 24% risk of post-surgical complications.

Complications associated with percutaneous closure include arrhythmia, AV block, cardiac erosion, and thromboembolism. Contraindications to percutaneous closure are small ASDs that have no hemodynamic impact, primum ostium defects, sinus venosus and sinus coronary defects, and secundum defects with advanced pulmonary hypertension. When atrial septal defects are closed percutaneously, patients require antiplatelet therapy for the following 6 months. Women with large ASDs and Eisenmenger syndrome should avoid pregnancy due to the risk of worsening existing pulmonary arterial hypertension and an increased incidence of dysrhythmias.[17] Surgical closure of atrial septal defects requires placement of a patch over the lesion through an incision in the right atrium.[13]

VII. Prognosis

Most small defects will close spontaneously within the first year of life, but large defects associated with systemic-to-pulmonary shunting and systemic oxygen desaturation require percutaneous surgical intervention. A subsequent study by Murphy et al. specifically examined 123 adults who underwent surgical repair of ostium secundum or sinus venosus atrial septal defect. When operated on before the age of 25, they had low mortality and morbidity rates. Conversely, those who underwent surgery at an older age had higher mortality and morbidity rates.

Independent predictors of long-term survival were age at the time of surgery and systolic pulmonary artery pressure before surgery. Twenty-seven late deaths were reported, with 18 cases caused by cardiovascular disease, death from heart disease, and 5 cases of death from stroke.[8] **2. CASE REPORT**

2.1. Anamnesis (Autoanamnesis and Alloanamnesis)

2.1.1 Identification

Mr. BS, 41 years old, Muslim, works as a construction worker, address in Sepancar Lawang Kulon village, Baturaja, Ogan Komering Ulu. Treated in room Komering 1.1 bed 2, RSMH Palembang since November 4, 2023, with Atrial Septal Defect (ASD).

2.1.2 Main Complaint

Shortness of breath has worsened since 3 months prior to admission.

2.1.3 Additional Complaints

Easily fatigued for the past 3 months prior to admission.

2.1.4 Medical History

For the past 3 months prior to admission, the patient has complained of shortness of breath, which is particularly noticeable after working as a construction worker. The symptoms improve with rest, and the patient sleeps comfortably with one pillow. Shortness of breath is not affected by weather or emotions. There are no complaints of waking up at night due to shortness of breath, no wheezing, no coughing, no chest pain, no palpitations, no tremors or sweaty palms, swelling of the legs is absent, cyanosis of the lips and fingertips/toes is absent, fever is absent, nausea is absent, vomiting is absent, epigastric pain is absent, weakness is absent, dizziness is absent, unexplained bruising is absent, nosebleeds and bleeding gums are absent, weight loss is absent, appetite is normal.

e-ISSN : 2622-4968, p-ISSN : 2622-1349

No complaints regarding bowel movements or urination. The patient initially sought treatment at a clinic and was diagnosed with stomach pain, but the symptoms did not improve. The patient then sought treatment at a private hospital in Baturaja, where he was treated for 5 days and diagnosed with heart disease. The patient returned home with persistent shortness of breath, especially when performing daily activities such as working as a construction laborer. The patient returned home with medication, but forgot the name of the drug. The complaints were felt to have decreased.

One month later, the patient was readmitted to the hospital due to shortness of breath. The shortness of breath was influenced by activities such as walking 20 meters and decreased when the patient rested. Chest pain was denied, cold sweats were denied, and palpitations were denied. There were no complaints of weakness or swelling in the legs. There were no complaints regarding bowel movements or urination. The patient was then referred to Siti Fatimah Hospital in Palembang, where a heart examination revealed a heart valve abnormality. The patient was then referred to RSMH for further management.

2.1.5 Past Medical History

- History of shortness of breath and fatigue during activity or exercise since childhood, but without cyanosis. Shortness of breath decreased and disappeared with rest.
- History of congenital heart disease discovered 1 month ago
- History of hypertension denied
- History of asthma denied
- History of pulmonary tuberculosis denied

2.1.6 Family Medical History

History of heart disease in the family denied The patient was born normal, full-term, but small in stature. History of infection or use of drugs and herbal medicines during the patient's mother's pregnancy denied

2.1.7 Socioeconomic History and Habits

- The patient works as a construction worker, has a high school education, and earns approximately Rp. 2,000,000 per month. Impression
- Lower-middle socioeconomic status.
- The patient eats three times a day with rice, various side dishes such as eggs, tofu, tempeh or fish, but the patient does not like to eat vegetables.
- The patient eats one plate per meal.

2.2. Physical Examination

General Condition:

Appears moderately ill

- Sensorium: Compos mentis
- BP: 100/70 mmHg
- Pulse: 80 x/m, regular, adequate volume and tension
- RR: 24 x/m, rapid and shallow
- T: 36.6ºC
- TB: 163 cm
- BB: 48 kg
- BMI: 18.1 kg/m2 (normal weight)

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Specific Findings:

- Head: Pale palpebral conjunctiva (-), icteric sclera (-), angular cheilitis (-), tongue papilla atrophy (-), exophthalmos (-), stomatitis (-)
- Neck: JVP (5-0) CmH2O, enlarged KGB (-), goiter (-)

Thoraks

Cor:

- I: Cardiac impulse visible at left LAA ICS VI
- P: Cardiac impulse palpable at left LAA ICS VI, thrill (+)
- P: Upper border of heart at ICS II, right LPS dextra, left LAA sinistra ICS VII
- A: HR: 80 x/m regular, systolic murmur (+) maximum punctum at ICS II parasternal sinistra, radiating laterally, grade 3/6, lifting (–), gallop (–)

Pulmo Anterior:

- I: Symmetrical static, right lung dynamic = left lung dynamic
- P: Right stem fremitus = left stem fremitus
- P: Sonorous in both lungs
- A: Vesicular (+) normal, rhonchi (-), wheezing (-)

Pulmo Posterior:

- I: Symmetrical static, dynamic right lung = left lung
- P: Right stem fremitus = left stem fremitus
- P: Sonorous in both lungs
- A: Vesicular (+) normal, rhonchi (-), wheezing (-)

Abdomen:

- I: Flat
- P: Weak, liver not palpable, spleen not palpable, epigastric tenderness (-)
- P: Tympanic, no *shifting dullness*, no CVA percussion pain
- A: Bowel sounds (+) normal

Extremities:

Pretibial edema (-/-), pale extremities (-), tremor (-), clubbing fingers (-), koilonychia (-).

2.3 Supporting Examinations

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Examination

Darah rutin	Hasil Pasien	Nilai Normal	Satuan
Hemoglobin (Hb)	13.7	13.48 - 17.40	g/dL
Eritrosit	4.56	4.40 - 6.30	
$10^6/mm^3$			
Leukosit	5.740	4.73-10.89	
$10^3/mm^3$			
Ht	39	41-51	%
Trombosit	172	170-396	$10^3\mu L$
MCV	85.1	85-95	fL
MCH	30	28-32	pg
MCHC	35	33-35	g/dL
Basofil	0	0-1	
Eosinofil	2	1-6	
Neutrofil	65	50-70	
Limfosit	27	20-40	
Monosit	6	2-8	

Kesan: Normal

Faal hemostasis	Hasil Pasien	Nilai Normal	Satuan
PT Kontrol	13.20		
PT Pasien	15.2	12-18	detik
APTT Kontrol	28.7		
APTT Pasien	31.3	27-42	detik
Fibrinogen Kontrol	326		
Fibtongen Pasien	357	200-400	detik
INR	1.12		
D-dimer	0.27	< 0.5	
mcg/ml			

Kesan: Normal Faal hemostasis

RSMH (19/10/2023)

Kimia Darah	Hasil Pasien	Nilai Normal	Satuan
AST/SGOT	17	0-32	U/L
AST/SGPT	11	0-31	U/L
Ureum	18	16.6-48.5	mg/dL
Kreatinin	1.07	0.50-0.90	mg/dL
Albumin	4.5	3.5-5.0	
Natrium	142	135-155	mg/dL
Kalium	4.1	3.5-5.5	mg/dL
Ca	9.0	8.5-10.2	mg/dL
Hbsag	Non reaktif		
Anti HCV	Non reaktif		
Anti HIV	Non reaktif		

Kesan: Normal Kimia Klinik

a. Electrocardiography at RSMH December 6, 2023

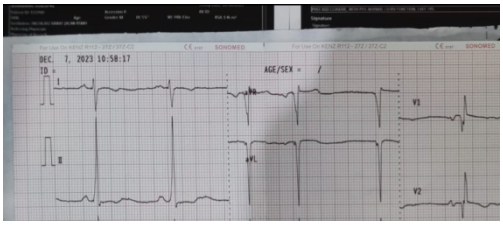


Figure 3. Patient's ECG

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Sinus rhythm, right axis, HR 80 bpm, normal P wave, PR interval 0.22, QRS complex 0.06 seconds, R/S in V1 >1, SV1 + RV5V6 < 35, LV strain.

Impression: **Sinus bradycardia, inverted T waves in V1-V6**

b. **Rontgen Thorax RSM**

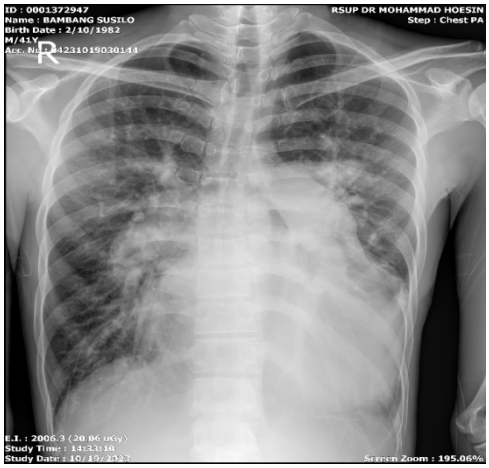


Figure 4. Chest X-ray, October 19, 2023

The chest X-ray examination revealed:

- Enlarged heart.
- Aorta is normal.
- Superior mediastinum is not widened.
- Trachea is in the center.
- Right and left hilar regions are widened.
- Increased bronchovascular markings in both lungs.
- No infiltrates or nodules visible in either lung.
- Smooth diaphragm.
- Right and left costophrenic sinuses are sharp.
- Bones and soft tissues of the chest wall are normal.

Impression: Cardiomegaly with pulmonary congestion

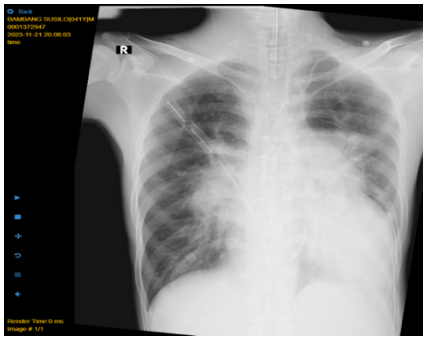


Figure 5. Chest X-ray, November 21, 2023

e-ISSN : 2622-4968, p-ISSN : 2622-1349

The chest X-ray examination revealed:

- CTR difficult to evaluate, impression of enlarged heart.
- Aorta normal.
- Trachea in the center, superior mediastinum not widened.
- Right and left hilar widened.
- Increased bronchovascular markings in both lungs.
- No infiltrates or nodules visible in either lung.
- Smooth diaphragm.
- Right costophrenic angle sharp, left sharp angle obscured by cardiac shadow.
- Bones and soft tissues of the chest wall normal.

Impression: Cardiomegaly

c. Echocardiography at Siti Fatimah Hospital 11/10/23

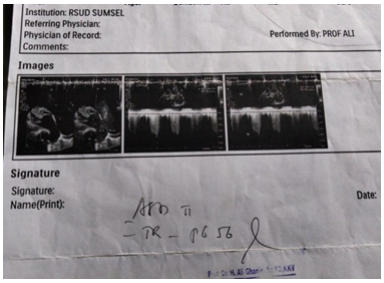
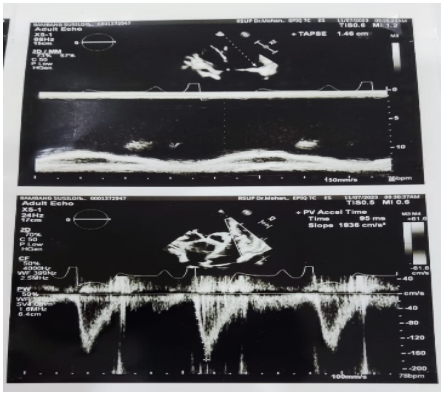


Figure 6. Patient echo at Siti Fatimah Hospital

Impression: ASD II, TR (PG 56)

d. Echocardiography RSMH



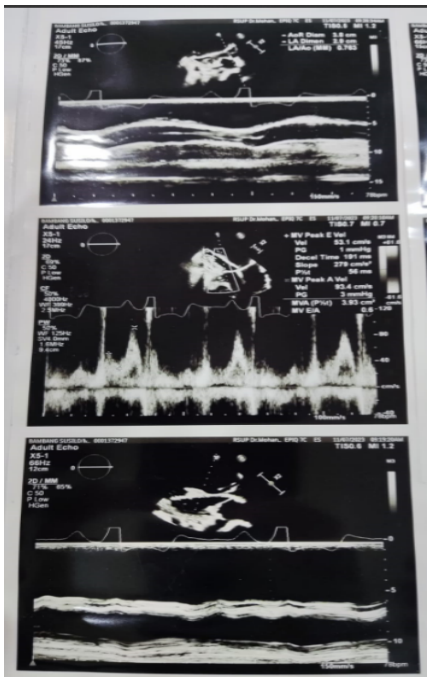


Figure 7. Echocardiography RSMH November 7, 2023

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Mild LA dilation, severe RA dilation, large secundum type ASD, severe RV dilation, mild to moderate reduced systolic function, EF 71.8%

Impression: Large ASD, left to right shunt

e. Echocardiography RSMH 7/12/23

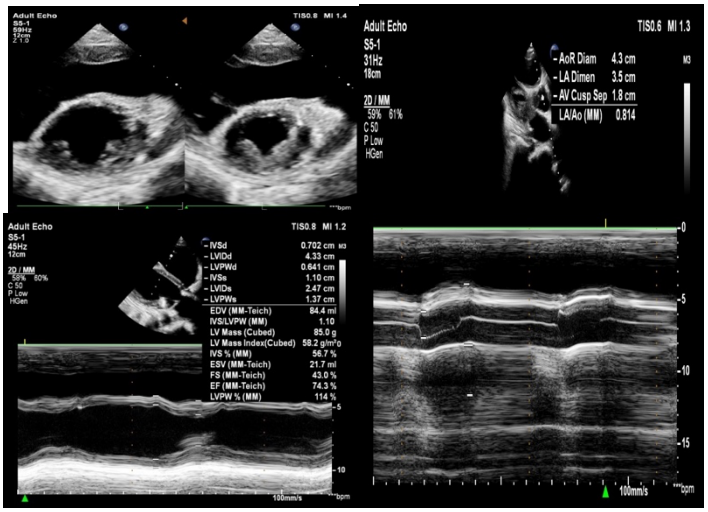


Figure 8. Echocardiography RSMH December 7, 2023

Impression: Moderate RA dilatation, moderate RV dilatation, moderate LA dilatation, normal LV function

Impression: post ASD closure, normal LV/RV function, EF 74%

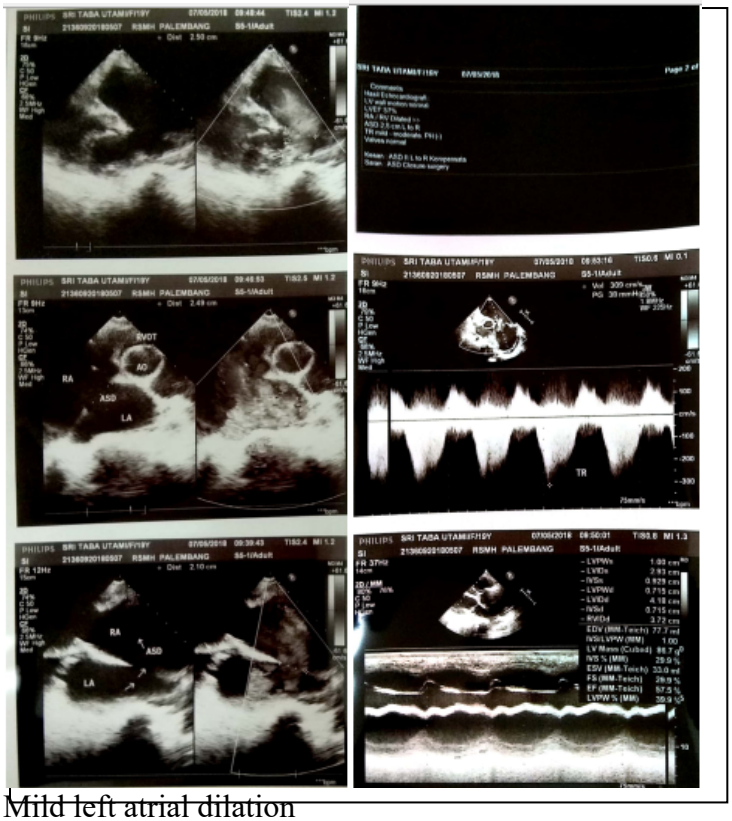
2.4 Developments During Treatment

Date	**November 4–20, 2023**
Subjective	Shortness of breath during activity, fatigue
Objective General condition Sensorium TD (mmHg) Pulse (x/minute) RR (x/minute) Temperature (OC) VAS Saturation	 Moderate pain Compos mentis 110/70 50x, adequate filling and tension 22x, regular, thoracoabdominal breathing pattern 36.6 0 97% room air

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Specific Circumstances	Head : Pale palpebral conjunctiva (-), icteric sclera (-) Neck : JVP 5-2 cmH20, enlarged KGB (-) Thorax : Barrel chest (–), intercostal spaces not widened Heart : HR 50/min regular, systolic murmur (+) maximum; point at ICS II parasternal left, radiating laterally,grade 3/6, lifting (–), gallop (–) Lungs :Vesicular breath sounds (+) normal, rales (-)/-), wheezing (-) Abdomen : Flat, soft, liver and spleen not palpable, tenderness (-), bowel sounds (+) normal Extremities: Warm extremities, pale palms, pretibial edema (-)
Supporting examination	**Laboratory** **RSMH on October 19, 2023** Hb 13,7 Eritrosit 4.56 Ht 39 Leukosit 5.740 Trombosit 168.000 MCV/MCH/MCHC 85,1/30/35 Diff Count 0/2/65/27/6 PT K/P 13.20/15.2 APTT K/P 28,7/31.3 Fibrinogen K/P 249/357 INR 1.11 D-dimer 0,27 SGOT/SGPT 17/11 Albumin 4,5 Ur/Cr 18/1.07 Ca 9 Natrium/Kalium 143/4,1 HBSAG NR, Anti HCV NR, Anti HIV NR **Echocardiography at Siti Fatimah Hospital on October 11, 2023** ASD II, TR (PG 56) **Ro thorax PA (19/10/23)** Cardiomegaly with pulmonary congestion
Assessment	- Right Heart Failure due to Secundum ASD, Tricuspid Regurgitation
Differential diagnosis	-
Planning Non farmakologi	Rest Rice Diet NB TKTP O2 3 L/minute if short of breath R/ ASD closure repair surgery (awaiting scheduling) Education Consultation with thoracic surgeon

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Farmakologi	IVFD Venflon Furosemide 1x20mg IV Spironolakton 1x12.5mg PO Ramipril 1x1.25mg PO
Plan Inspection	Echocardiografi
Supporting examination	***Echocardiography* 7/11/2023**  Mild left atrial dilation Severe right atrial dilation Large secundym type ASD Severe right ventricular dilation Mild to moderate systolic function reduction EF 71.8% Impression: Large ASD, left to right shunt

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Planning Nonfarmakologi	Rest Rice diet NB TKTP O2 3 L/minute if short of breath R/ ASD closure repair surgery (scheduled for Thursday, 11/16/2023) Education Response from Dr. Bermansyah SpBTKV: ASD closure surgery will be planned. Recommendation: preoperative coronary catheterization
Plan	Dr. Ria Nova, Pediatrician (K)

Date	**November 21, 2023**
Subjective	No congestion
Objective General condition Sensorium TD (mmHg) Pulse (x/minute) RR (x/minute) Temperature (OC) VAS	 Moderate pain Compos mentis 90/70 68x, adequate volume and tension 20x, regular, thoracoabdominal breathing pattern 36.6 0
Specific Circumstances	Head : Pale conjunctiva (-), icteric sclera (-) Neck : JVP 5-2 cmH20, enlarged KGB (-) Thorax : *Barrel chest (-), intercostal spaces not widened* Cor : Regular HR 70x/m, systolic murmur (+) maximum punctum at ICS II parasternal left, lateral radiation, grade 3/6, lifting (-), gallop (-) Pulmo : Vesikuler (+) normal, ronkhi (-/-), wheezing (-) Abdomen : Flat, limp, liver and spleen not palpable, tenderness (–), bowel sounds (+) normal Ekstremitas : Warm extremities, pale palms, pretibial edema (-)
Assessment	- Right Heart Failure with Secundum ASD, Tricuspid Regurgitation
Differential Diagnosis	-

e-ISSN : 2622-4968, p-ISSN : 2622-1349

Planning Nonfarmakologi Farmakologi	- Rest - NB TKTP rice diet - O2 3 L/minute if short of breath - R/ ASD closure repair surgery today - IVFD Venflon - Furosemide 1x20mg IV - Spironolakton 1x12.5mg PO - Ramipril 1x1.25mg PO
Operations report	- Anesthesia induction went smoothly, with arterial line and CVC monitors installed. - Skin preparation with povidone iodine was followed by draping. - Median sternotomy incision. - The pericardium was opened, appearing as in the findings. - The pericardium was freed and preserved. - Heparin was administered, and after the ACT value was reached, cannulation of the aorta, SVC, and IVC was performed. - An antegrade cardioplegia cannula was inserted. - The heart machine was started, and the temperature was lowered. - A cross-clamp was placed on the aorta, cardioplegia fluid was administered antegrade, and the heart immediately went into asystole. - The RA is opened. - Evaluation reveals a 30 mm secundum ASD, all pulmonary veins to the left atrium. - ASD closure is performed using a pericardial patch and PFO creation. - Tricuspid evaluation is as found, tricuspid valve repair (TVr) is performed with a no. 30 ring annuloplasty, deairing the left heart. - The pulmonary artery (PA) was opened, and no patent ductus arteriosus (PDA) was identified. - The PA was closed again. - The RA was closed. - The temperature was raised again. - The aortic cross-clamp was removed, and the rhythm was sinus rhythm. - After the body temperature returned to normal, the heart machine was weaned off until it stopped. - TEE evaluation showed: PFO creation (+), residual TR (-). IVC and SVC were decannulated, protamine was administered, and bleeding was treated. - The aorta was decannulated after the protamine was depleted.

e-ISSN : 2622-4968, p-ISSN : 2622-1349

	- One 32 Fr substernal drain is placed. - The pericardium is partially closed, the sternum is closed with sternal wire, and the surgical wound is closed layer by layer with synthetic absorbable sutures. - The operation is complete. - The patient is transferred to the ICU with hemodynamics: - ABP 85/49 (63) mmHg - HR 100 bpm SR - CVP 5 mmHg - SpO2 96% with dobutamine support 5 mcg/kgBW/minute - milrinone 0.375 mcg/kgBW/minute. Amount of blood loss: 400cc, transfusion of 2 bags of PRC and 2 bags of FFP performed

CONCLUSION

The case of a 41-year-old man with complaints of shortness of breath, especially during physical activity, which he had been experiencing for 3 months prior to admission, was discussed. Based on the results of the medical history, physical examination, and supporting tests, the patient was diagnosed with right heart failure due to secundum atrial septal defect and tricuspid regurgitation.

REFERENCES

1. Atmakusuma D, Setyaningsih I. Dasar-dasar talasemia: Salah satu jenis hemoglobinopati. Dalam: Setiati S, Alwi I, Sudoyo AW, Sumadibrata M, Setyohadi B, Syam AF, editors. Buku Ajar Ilmu Penyakit Dalam. 6th ed. Jakarta: Internal Publishing. 2014. hal 2625-32
2. P2PTM Kemenkes RI. Putuskan Mata Rantai Talasemia, Indonesia Lebih Sehat pada**.** 2019. http://p2ptm.kemkes.go.id (diakses 15 September 2023)
3. L. Edward. Disorder of Hemoglobin. Harrison Internal Medicine chapter 127.Mc Graw Hill. 2015. hal 420-97.
4. Cappellini MD, Motta I. New therapeutic targets in transfusion-dependent and-independent thalassemia. Hematology 2014, the American Society ofHematology Education Program Book. 2017 Dec 8;2017(1):278-83.
5. F. Assadi. Diagnosis of hypokalemia: A problem solving approach to clinical cases. Iran: Iran Jurnal Kidney Disease, 2008.
6. Widjajanti A, Agustini SM. Hipokalemia periodik paralisis. Ind J Clin Pathol Med Lab. 2005; 12(1) : 19-22
7. Siregar P. Gangguan Keseimbangan Air dan Elektrolit. In : Setiati S, Alwi I, Sudoyo AW, Simadibrata

e-ISSN : 2622-4968, p-ISSN : 2622-1349

M, Setiyohadi B, Syam AF. Buku Ajar Ilmu Penyakit Dalam Jilid II. Edisi VI. Jakarta: Interna Publishing; 2014: 2241-2257

8. Lines LS. Disorder of potassium metabolism: fluid and electrolyte disorder. the journal of the association of physicians of india, 2000:101-1010
9. Palmer F. Biff. Normal acid-base balance and metabolic acidosis: fluid andelectrolyte disorder.NEJM, 2000:1212-14.
10. Harvey TC. Addison's disease and the regulation of potassium: the role of insulinand aldosterone. Med Hypotheses. 2007;69(5):1120-6
11. Rujito L, Talasemia: Genetik Dasar dan Pengelolaan Terkini. Dalam: Siswandari W, Lestari DWD, editors. Universitas Jenderal Soedirman. 2019. hal 39-47.
12. Bakta IM. Pendekatan terhadap pasien anemia. Dalam: Setiati S, Alwi S, Sudoyo AW, Simadibrata M, Setiohadi B, Syam AF, Editors. Buku Ajar Ilmu Penyakit Dalam. Jilid III. Edisi 6. Jakarta: Internal Publishing. 2014. hal 2575-81.
13. Pedoman Nasional Pelayanan Kedokteran Tatalaksana Thalasemia. Keputusan Menteri Kesehatan Republik Indonesia. Nomor HK.01.07/MENKES/1/2018. Jakarta. 2018.
14. Jameson LJ, Kasper DL, Longo DL, Fauci AS, Hauser SI, Loscalzo J. Harrison's Principles of Internal Medicine, 21th edition. New York: McGraw-Hill Education 2022.
15. Vasquez-Rios G, Westrich Jr DJ, Philip I, Edwards JC, Shieh S. Distal renal tubular acidosis and severe hypokalemia: a case report and review of the literature. Journal of Medical Case Reports. 2019; 13:103-10.
16. Khositseth Sookkasem, Urawithya Panapat, Soomparn Poorichaya, Charngkaew Komgrid. Autophagic degradation of Aquaporin-2 is an early event in hypokalemia-induced Nephrogenic diabetes Insipidus. 2019. Scientific Reports:5:183111
17. Both T, Zietse R, Hoorn EJ, van Hagen PM, Dalm VA, van Laar JA, et al. Everything you need to know about distal renal tubular acidosis in autoimmune disease. Rheumatol Int. 2014; 34(8):1037-45.

e-ISSN : 2622-4968, p-ISSN : 2622-1349