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Atrial Septal Defect

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Abstract

Atrial Septal Defect (ASD) is a congenital heart disease that can be asymptomatic in childhood but has the potential to cause long-term hemodynamic complications. This paper aims to comprehensively discuss the definition, epidemiology, etiology, risk factors, pathogenesis, clinical manifestations, diagnosis, management, indications for closure, complications, and prognosis of ASD. This study uses a descriptive study with a literature review method. The study population consists of scientific sources on ASD and related congenital heart diseases, while the sample consists of relevant literature sources used in the study. The research instruments, consisting of literature sources and data, were analyzed descriptively through identification, grouping, and synthesis of information based on the discussion topic. The results of the study indicate that a significant left-to-right shunt can cause right ventricular volume overload, right heart enlargement, exercise intolerance, atrial arrhythmia, pulmonary hypertension, and Eisenmenger syndrome. Echocardiography is the main examination, while hemodynamically significant ASD may require transcatheter device closure or surgical closure. In conclusion, early evaluation and appropriate closure of ASD can support a good prognosis and reduce the risk of long-term complications.

Keywords: ***Atrial Septal Defect, Congenital Heart Disease, Echocardiography, Pulmonary Hypertension, Transcatheter Closure***

INTRODUCTION

Atrial Septal Defect (ASD) is a congenital heart disease characterized by a defect in the septum separating the right and left atria due to developmental disorders during embryogenesis. ASDs primarily consist of the ostium secundum, ostium primum, and sinus venosus, with secundum ASD being the most common type. In most cases, higher left atrial pressure causes a left-to-right shunt, increasing blood flow to the right side of the heart and pulmonary circulation (Yates & Gewillig, 2018).

ASD has a significant prevalence in congenital heart disease. A meta-analysis of 260 studies involving over 130 million births showed a prevalence of 1,441 per 1,000 live births, accounting for approximately 15.4% of all congenital heart disease subtypes. In Indonesia, epidemiological data on ASD is still limited. Research at Dr. Sardjito General Hospital in Yogyakarta indicates that ASD is the second most common congenital heart disease lesion in children, accounting for 17% of cases, and the most common congenital heart disease lesion in adults, accounting for approximately 60% (Liu et al., 2019).

The problem with ASD is that most children don't show obvious symptoms during infancy and childhood. This condition often results in it being discovered incidentally during a routine physical examination. However, persistent left-to-right shunt can increase blood volume in the right heart and cause dilation of the right atrium and ventricle (Yates & Gewillig, 2018; Kligman, 2024).

Large, untreated ASDs can lead to long-term complications due to increased pulmonary blood flow. This condition can lead to increased pulmonary vascular resistance, pulmonary hypertension, and, in advanced cases, a right-to-left shunt, which can lead to Eisenmenger syndrome. Furthermore, long-standing ASDs can be associated with atrial arrhythmia, right-sided heart failure, tricuspid regurgitation, growth retardation, and exercise intolerance (Yates & Gewillig, 2018; Kligman, 2024).

Another issue relates to the diagnosis and determination of the need for ASD closure. Echocardiography is the primary examination to confirm the diagnosis, determine the size and location of the defect, the direction of the shunt, and the hemodynamic impact on the right heart. Management depends on the size of the defect, the extent of the shunt, symptoms, and right ventricular volume

overload. Small ASDs can be monitored because they can shrink or close spontaneously, while ASDs with significant hemodynamic impact may require closure (Yates & Gewillig, 2018).

This paper aims to discuss ASD, including its definition, epidemiology, etiology, risk factors, pathogenesis, clinical manifestations, diagnosis, management, indications for closure, complications, and prognosis. This discussion is important because ASD can be asymptomatic in childhood but has the potential to cause long-term complications. The novelty of this paper lies in the comprehensive presentation of ASD, linking hemodynamic mechanisms, clinical manifestations, diagnosis, management, complications, and prognosis (Yates & Gewillig, 2018; Kligman, 2024).

RESEARCH METHODS

This study uses a descriptive literature review method, discussing Atrial Septal Defect (ASD) based on relevant scientific sources. The study covers the definition, epidemiology, etiology, risk factors, pathogenesis, clinical manifestations, diagnosis, management, complications, and prognosis of ASD, with reference to the literature used.

The research instrument consisted of literature sources related to Atrial Septal Defect (ASD). Data were analyzed descriptively by identifying, categorizing, and describing information according to the main topics of ASD.

The study population consisted of literature sources discussing Atrial Septal Defect (ASD) and related congenital heart disease. The sample consisted of literature sources used in the paper, namely Kligman (2024), Yates and Gewillig (2018), Liu et al. (2019), Germany et al. (2021), Gomersall et al. (2024), Davis et al. (2026), and Tuan et al. (nd).

The research was conducted by determining the topic of ASD, collecting relevant literature, identifying information according to the topic, then categorizing and analyzing it descriptively. The study results were then systematically compiled into a discussion on Atrial Septal Defect (ASD).

RESULTS AND DISCUSSION

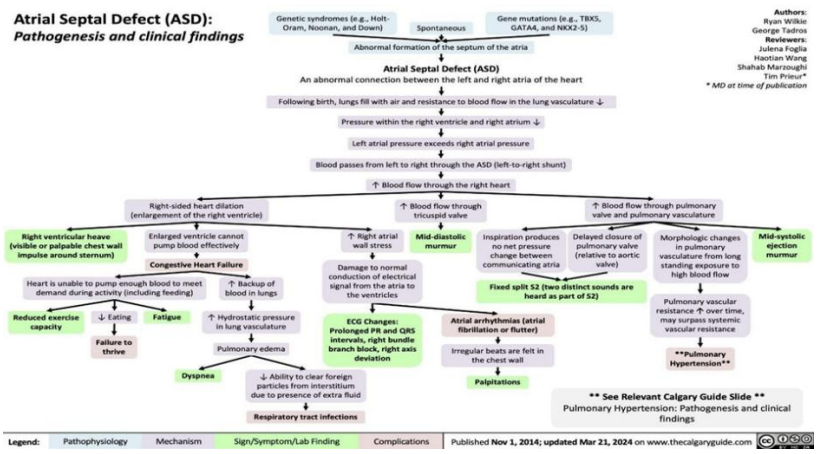

Figure 1. Pathogenesis of Atrial Septal Defect (ASD) (Kligman, 2024).

Clinical Symptoms

Asymptomatic

Most children with ASD, especially secundum ASD, are asymptomatic. The abnormality is often discovered incidentally during a routine physical examination, for example, when a doctor detects an abnormal heart murmur. This condition is related to the body's tolerance to left-to-right shunts in childhood and the absence of severe hemodynamic complications (Yates & Gewillig, 2018).

Growth disorders (failure to thrive)

In younger children, especially those with ASD with a significant left-to-right shunt, growth retardation

may be present. This may manifest as difficulty gaining weight or slower growth compared to peers. However, these manifestations are usually mild and not always present. Nelson emphasized that failure to thrive in children with ASD can be subtle and often not a primary symptom.

Easily tired and decreased activity tolerance (exercise intolerance)

In older children and adolescents, more common manifestations include fatigue or decreased tolerance for physical activity. Children may be able to perform normal daily activities but have lower athletic or physical activity abilities than their peers. This condition often goes unnoticed by families because it develops gradually. Nelson explains that activity limitations may become more apparent after ASD is corrected, as the child experiences increased activity and growth.

Shortness of breath (dyspnea)

Shortness of breath is not a primary manifestation in most children with simple ASD. However, it can occur in patients with a large left-to-right shunt or in patients whose disease has progressed to pulmonary circulatory failure. Long-term increased pulmonary blood flow can lead to pulmonary vascular changes, and in some patients, pulmonary hypertension can develop, which can then lead to shortness of breath.

Palpitations

Palpitations can occur primarily in patients with ASD who have had long-term right atrial dilation. Atrial dilation and structural changes in atrial tissue can increase the likelihood of atrial arrhythmias, such as atrial fibrillation or atrial flutter. Palpitations are more common in patients with ASDs that remain unclosed until later in life than in children who undergo early correction.

Cyanosis

Cyanosis is not a typical manifestation of isolated ASD with a left-to-right shunt. In uncomplicated ASD, blood continues to flow from the left atrium to the right atrium, thus not causing cyanosis. Cyanosis can occur in later stages if there is a severe increase in pulmonary vascular resistance, causing the shunt to change direction to right-to-left. This condition is part of Eisenmenger syndrome (Kligman, 2024).

Manifestations in ASD with Large Shunt and Complications

In patients with ASD who have a large and long-standing left-to-right shunt, increased blood flow to the right side of the heart can cause progressive structural and functional changes. Manifestations may include:

Dilation of the right atrium and right ventricle

Chronically increased blood volume passing through the right atrium and right ventricle causes right-sided volume overload. This condition can lead to enlargement of the right atrium, right ventricle, and pulmonary artery.

Atrial arrhythmia

Long-standing right atrial dilation can cause structural and electrophysiological changes in the atrial myocardium, increasing the risk of atrial arrhythmias, particularly atrial fibrillation and atrial flutter. These complications are more common in patients whose ASDs are not repaired until adulthood.

Pulmonary hypertension

*Left-to-right shunt*Large volumes cause chronic increases in pulmonary blood flow. Over the long term, exposure of the pulmonary vasculature to high flow can lead to vascular remodeling and increased pulmonary vascular resistance. In a small proportion of patients, this process can progress to pulmonary hypertension.

Eisenmenger syndrome

In advanced disease with a severe increase in PVR, pressure on the right side of the heart can increase, causing the left-to-right shunt to diminish and eventually revert to a right-to-left shunt. Deoxygenated venous blood then enters the systemic circulation, causing cyanosis. This condition is called Eisenmenger syndrome. In ASD, this complication is relatively rare in childhood and is more of a long-term complication (Yates & Gewillig, 2018).

Diagnosis

Anamnesis

A history taking in patients with suspected ASD is performed to identify symptoms resulting from the left-to-right shunt and increased pulmonary blood flow. Most children with secundum ASD are asymptomatic, but growth retardation, fatigue, exercise intolerance, shortness of breath, palpitations, or cyanosis may be present. A family history should also be explored, as ASD may be associated with genetic factors such as Holt-Oram syndrome and the TBX5, NKX2.5, and GATA4 genes.

Physical examination

Most children with ASD have a good general condition. A physical examination of the heart may reveal a right ventricular systolic lift or heave at the left sternal border due to right ventricular volume overload and a

mild left precordial bulge due to right heart enlargement (Yates & Gewillig, 2018).

Cardiac Auscultation

A typical finding of ASD is a wide and fixed splitting of S2 due to increased right ventricular volume. Additionally, a systolic ejection murmur may be heard at the mid-to-upper left sternal border due to increased blood flow to the pulmonary artery. With a large left-to-right shunt, a mid-diastolic rumble may be heard due to increased flow through the tricuspid valve, which may indicate a significant shunt with a Qp:Qs ratio of approximately ≥2:1 (Kligman, 2024).

Electrocardiography (ECG)

An ECG cannot confirm the diagnosis of ASD, but it can indicate its impact on the right side of the heart. In secundum ASD, right axis deviation and incomplete right bundle branch block with an rsR' pattern, particularly in V1, can be found. In older patients with chronic right atrial dilation, atrial arrhythmia can be found.

Thoracic X-ray

A chest x-ray can demonstrate the hemodynamic consequences of an ASD. A significant left-to-right shunt may reveal right atrial and ventricular enlargement, pulmonary artery dilation, and increased pulmonary vascularity. However, a chest x-ray may remain normal in small defects, so a normal result does not rule out an ASD.

Echocardiography

Echocardiography is the primary test for confirming an ASD. This test can determine the location, size, and number of defects, the direction of the shunt, and hemodynamic impacts such as right ventricular volume overload. Evaluation of the pulmonary veins is also important to detect the possibility of partial anomalous pulmonary venous return (PAPVR) in secundum ASD.

Transesophageal Echocardiography (TEE)

TEE is not always necessary in children because most secundum ASDs can be assessed with TTE. TEE is considered if TTE imaging is inadequate, particularly in sinus venosus ASDs or prior to transcatheter device closure. This examination helps assess the size, shape, and septal rims of the defect (Germany et al., 2021).

Cardiac Magnetic Resonance (CMR)

CMR is not a routine examination for ASD in children. It may be used if echocardiography results are inadequate or if a more accurate assessment of right ventricular volume and function, Qp:Qs, and pulmonary venous anatomy is needed in complex cases.

Cardiac Computed Tomography (CT)

Cardiac CT is not a routine examination for pediatric ASD. It may be used in certain circumstances to obtain more detailed anatomical images, particularly if an anomalous pulmonary venous connection or complex abnormalities that cannot be optimally assessed with echocardiography are suspected. Its use should be selective due to radiation exposure.

Cardiac Catheterization

Cardiac catheterization is not routinely required for isolated secundum ASD diagnosed by echocardiography. This examination is primarily considered if pulmonary hypertension or increased pulmonary vascular resistance is suspected and can be used to more accurately measure heart pressure, PVR, and Qp:Qs (Kligman, 2024). If performed, a step-up in oxygen saturation in the right atrium may be observed, although this finding is not specific for ASD and can be seen in other conditions such as PAPVR (Germany et al., 2021).

Governance

Atrial Septal Defect (ASD) management is determined by the type and size of the defect, the extent of the left-to-right shunt, right ventricular volume overload, symptoms, age, and the presence of PVR and pulmonary hypertension. Small ASDs with minimal shunt and without right ventricular enlargement can be monitored because they can shrink or close spontaneously. Closure is considered in symptomatic or asymptomatic patients with Qp ≥2:1 or right ventricular enlargement (Yates & Gewillig, 2018).

Pharmacological Management

There is no medication that can anatomically close an ASD. Pharmacological therapy is generally unnecessary for uncomplicated ASD. Medication is only given supportively to manage underlying conditions such as heart failure, arrhythmia, or pulmonary hypertension, not to close the ASD defect.

Diuretic

Can be given if there is heart failure or signs of congestion, for example due to a large left-to-right shunt. For example furosemide. The goal is to reduce excess fluid and symptoms of congestion, but not to improve or close the ASD.

Heart failure therapy if necessary

If a large ASD causes heart failure, therapy may be given according to the clinical condition, such as diuretics and other heart failure therapies. This therapy is only temporarily supportive, especially while waiting for definitive closure.

Pulmonary hypertension therapy

In patients with established pulmonary arterial hypertension, specific pulmonary hypertension medications such as endothelin receptor antagonists, PDE-5 inhibitors, or prostacyclin pathway agents may be considered by a pediatric cardiologist/congenital heart disease specialist based on hemodynamic evaluation. This is not routine therapy for ASD without pulmonary hypertension.

Antiarrhythmic

If a patient experiences atrial arrhythmia, such as atrial flutter or atrial fibrillation, antiarrhythmic therapy or arrhythmia management can be given depending on the type and condition of the patient. Arrhythmia is more common in long-standing ASD.

Anticoagulant/antiplatelet

Not routinely prescribed for ASD. Anticoagulants are considered if there are specific indications, such as certain atrial arrhythmias or thromboembolism. After device closure, antiplatelets such as aspirin are generally given for a specific period, depending on the center's protocol and the type of device (Yates & Gewillig, 2018).

Non-Pharmacological Management

Non-pharmacological management of atrial septal defects (ASDs) aims to monitor the development of the defect, assess the hemodynamic impact, prevent complications, and perform defect closure if indicated. Not all patients with ASDs require immediate intervention. For small ASDs with minimal left-to-right shunts and without right ventricular enlargement, regular observation and monitoring are recommended. Infants with small to moderate ASDs can be monitored because the defect can shrink during the first year of life.

Observation and Follow-up

In small ASDs with minimal shunt and no right ventricular enlargement, closure is generally not necessary. Patients can be monitored closely.

Periodic monitoring by a pediatrician/pediatric cardiologist with clinical examination and echocardiography is needed to monitor the development of the defect size, shunt direction, right atrial and ventricular size, and pulmonary pressure. In infants and young children with small to moderate ASDs, observation is important because the defect can shrink spontaneously during the first year of life. Nelson stated that small to moderate ASDs in infants can be closely monitored because the defect often becomes smaller in the first year of life.

The American Heart Association also states that small ASDs often do not require therapy, and some ASDs found in infants can shrink or close spontaneously. There is no medication that can accelerate anatomical closure of an ASD. However, if right ventricular enlargement, increased left-to-right shunt, the appearance of symptoms, or the development of pulmonary hypertension are detected during follow-up, the patient should be re-evaluated to determine the need for ASD closure.

Physical Activity

In children with small, uncomplicated ASDs, physical activity generally does not need to be restricted. Children can engage in activities according to their physical ability and tolerance. In larger ASDs with right ventricular volume overload, pulmonary hypertension, arrhythmias, or other complications, the type and intensity of activity need to be adjusted based on the patient's clinical condition and the results of the cardiology evaluation. Once the ASD is successfully closed and there are no residual complications, most children can gradually return to normal physical activity.

Nutrition and Growth

Monitoring nutritional and growth status is an integral part of the care of children with ASD, especially those with large defects and significant shunts. Weight and height should be monitored regularly using age- and sex-appropriate growth charts. In children with failure to thrive or increased energy needs due to heart disease, nutritional intake should be optimized. The goal is to ensure optimal growth and development. However, it should be emphasized that nutritional therapy cannot anatomically close an ASD.

plays a major role in supporting the growth and general condition of patients (Davis et al., 2026).

Education for Parents

Parents need to be educated about the nature of the disease, the potential natural history of ASD, and the signs to watch for. In small ASDs, parents need to understand that the condition can be monitored without immediate intervention, as some defects can shrink spontaneously.

Parents also need to be informed about symptoms that require further evaluation, such as:
· easily tired during activities;
· hard to breathe;
· decreased activity tolerance;
· growth disorders;
· palpitations;
· cyanosis;
· episodes of fainting or decreased consciousness.
Education also includes the importance of regular check-ups and echocardiography examinations as scheduled by the doctor, as children can still appear healthy despite having a significant left-to-right shunt.

ASD Closure as a Definitive Nonpharmacological Measure

In hemodynamically significant ASDs, definitive therapy is not medication, but defect closure. According to Nelson, closure can be performed with transcatheter device closure or surgical closure in symptomatic patients as well as asymptomatic patients with Qp:Qs.
≥2:1 or right ventricular enlargement. For secundum ASDs with suitable anatomy, transcatheter device closure is generally the preferred option because it is performed through a catheter without open surgery. Conversely, ASDs that are not suitable for the device or certain types of ASDs, such as primum ASDs and sinus venosus ASDs, generally require surgical closure.

Indications for ASD Closure

ASD closure is considered if the defect causes significant hemodynamic impact. According to Nelson 22nd edition, transcatheter device closure or surgical closure is recommended for:

- Patients with symptoms due to ASD.
- Asymptomatic patients with Qp:Qs ≥2:1.
- Patients with right ventricular enlargement due to volume overload.

The decision to close an ASD is based not only on the defect diameter but also on right ventricular enlargement due to increased stroke volume, an important parameter of hemodynamic significance. In children requiring closure, elective surgery is generally performed in childhood, usually after the first year of life and before school age, to correct the left-to-right shunt and prevent long-term complications. After closure, symptoms may improve, right heart size may approach normal, and signs of right ventricular volume overload on the ECG may diminish (Germany et al., 2021).

Transcatheter Device Closure

Understanding

Transcatheter device closure is a method of ASD closure that involves inserting an occlusion device through a catheter, usually inserted through the femoral vein and then directed toward the heart. This method is primarily used for secundum ASDs with suitable anatomy. In most patients with secundum ASDs requiring closure, percutaneous catheter device closure is the procedure of choice. The device is placed through a transvenous venous access in a cardiac catheterization laboratory..

Main requirements

Not all secundum ASDs can be closed using a device. Before the procedure, the following should be evaluated:

- Size and shape of ASD.
- ASD Location.
- Adequate septal rims.
- Relationship of defects to AV valves.
- Relationship with the vena cava.
- Relationship with the coronary sinus.
- Relationship to the aorta.
- Pulmonary venous drainage.
- Whether or not there are additional defects.

Device closure is the primary option for secundum ASD when anatomically feasible, taking into consideration the diameter of the defect and the adequacy of the rim for device anchorage.

Profit

The advantages of device closure over surgery include:

- No sternotomy required.

- Does not require open-heart surgery.
- The length of stay is usually shorter.
- Faster recovery period.
- Closure results are excellent in suitable patients.

Nelson stated that after device closure, patients can generally be discharged the following day and the results of the procedure are excellent (Kligman, 2024).

Surgical Closure

Surgical closure is performedif the ASD is not suitable for closure using a device or there are other heart defects that require surgery.

Indications for surgery include:

- ASD with anatomy unsuitable for device closure.
- **Secundum ASD**with inadequate rims.
- **Primum ASD**.
- **Sinus venosus ASD**.
- ASD accompanied by other heart defects requiring surgical correction.
- ASD with PAPVR requiring concomitant correction.

During surgery, the defect can be closed directly with sutures or a patch, depending on the size and location of the defect. Surgical closure in childhood has excellent outcomes with an operative mortality of <1%, especially when performed in patients without pulmonary hypertension and severe comorbidities.

Complications

Complications of ASDs are primarily related to the size and duration of the left-to-right shunt, increased pulmonary blood flow, right heart enlargement, and the development of pulmonary vascular changes. In children, ASDs are generally well tolerated and often do not cause severe symptoms. However, large, untreated ASDs can lead to long-term complications.

Right Atrial and Right Ventricular Enlargement

The presence of a left-to-right shunt increases the volume of blood entering the right atrium and subsequently the right ventricle. If prolonged, this condition leads to dilation of the right atrium and right ventricle (right heart volume overload). Right ventricular enlargement is an important indicator that an ASD is having significant hemodynamic impact. This condition is also a primary consideration for ASD closure (Kligman, 2024).

Pulmonary Hypertension

A large ASD causes chronically increased blood flow to the lungs. Over the long term, increased pulmonary blood flow can cause changes in the pulmonary vessels, increasing pulmonary vascular resistance (PVR) and ultimately leading to pulmonary hypertension.

In most children with ASD, PVR remains low throughout childhood. However, PVR can increase in adulthood if significant ASD is not treated.

Eisenmenger Syndrome

In advanced stages of the disease, a severe increase in PVR can cause the pressure on the right side of the heart to become so high that the direction of the shunt can be reversed from:

Left → right → Right → left

As a result, oxygen-poor venous blood enters the systemic circulation, causing cyanosis. This condition is called Eisenmenger syndrome. Eisenmenger syndrome is a late complication, and when severe pulmonary vascular disease has already developed, the ASD should not be routinely closed, as the defect can serve as a decompression pathway for the right heart.

Atrial Arrhythmia

Chronic right atrial dilation can cause changes in atrial structure and electrophysiology, increasing the risk of atrial dysrhythmia, especially in patients who are not treated until adulthood.

Arrhythmias that can be found include:

· atrial fibrillation;
· atrial flutter;
· supraventricular tachycardia;
· atrioventricular conduction disorders in certain conditions.

The risk of arrhythmia increases if the ASD has been present for a long time and the patient undergoes closure at an older age. Nelson stated that late right-sided heart failure and arrhythmias are more common in patients who undergo repair after age 20 than in those who undergo repair earlier.

Right-Sided Heart Failure

*Chronic right ventricular volume overload*can cause dilation and ultimately right ventricular dysfunction. This condition can progress to right-sided heart failure, especially in patients with large, uncorrected ASDs and pre-existing pulmonary hypertension. Manifestations can include fatigue, peripheral edema, hepatomegaly, increased jugular venous pressure, and activity intolerance.

Tricuspid Regurgitation

Enlargement of the right atrium and ventricle due to volume overload can cause changes in the tricuspid annulus and ultimately lead to tricuspid regurgitation. Tricuspid regurgitation can further increase the volume load on the right heart and, if severe, can contribute to the development of right heart failure. Nelson lists tricuspid insufficiency as a late complication of ASD (Yates & Gewillig, 2018).

Mitral Regurgitation

Mitral regurgitation can be found as a complication or accompanying abnormality of ASD, although it is not a primary complication of secundum ASD. In certain ASDs, particularly primum ASD/atrioventricular septal defect, atrioventricular valve abnormalities may be more prominent.

Paradoxical Embolism and Stroke

In an ASD, blood normally flows from the left atrium to the right atrium, preventing direct systemic embolism. However, if right atrial pressure increases or a right-to-left shunt occurs, thrombus from the venous system can pass through the ASD into the systemic circulation. This phenomenon is called paradoxical embolism and can lead to stroke or systemic embolism. This risk is more relevant in adults or patients with conditions that increase right atrial pressure. A similar concept is also important in patent foramen ovale (PFO), although PFOs are anatomically different from ASDs.

Growth Disorders and Exercise Intolerance

In children, milder clinical complications can include failure to thrive and exercise intolerance. Children with large ASDs can experience increased pulmonary blood flow and cardiac workload, which can impair growth and activity tolerance. Nelson noted that younger children may show subtle signs of failure to thrive, while older children may experience varying degrees of exercise intolerance.

Pregnancy Complications in Adult Patients

In women with uncorrected ASD, the increase in circulating volume during pregnancy can increase the cardiac volume load. In significant ASD, this condition can worsen right heart volume overload, pulmonary hypertension, or arrhythmias. Symptoms of ASD complications can begin to appear in adulthood and can be triggered by the increased volume load during pregnancy (Kligman, 2024).

Prognosis

In general, the prognosis for atrial septal defects (ASDs), particularly secundum ASDs, in children is quite good. Prognosis is influenced by the size of the defect, the extent of the left-to-right shunt, the presence of right ventricular volume overload, the age at diagnosis and closure, and the presence or absence of pulmonary hypertension and other complications. Most children with secundum ASDs tolerate the defect well during childhood, with many remaining asymptomatic into adulthood. The 22nd edition of the Nelson Textbook of Pediatrics states that small to moderate ASDs found in full-term infants can shrink or close spontaneously. Secundum ASDs are also generally well tolerated during childhood, with significant symptoms typically appearing in the third decade or later (Kligman, 2024).

***Quo ad vitam:*Dubia ad bonam**

In general, the prognosis for life is good, especially in children with small secundum ASDs or in patients with significant ASDs who undergo closure before pulmonary hypertension and right heart complications develop. Nelson stated that secundum ASDs are generally well tolerated during childhood, and outcomes after surgical and device closure in children with medium-to-large shunts are excellent.

***Quo ad functionam:*Dubia ad bonam**

Function and activity capacity are generally good, especially if ASD is treated early. After closure, symptoms may resolve, growth may improve, heart size may return to normal, and right ventricular volume load may decrease. However, the functional prognosis worsens if pulmonary hypertension, arrhythmia, or heart failure develop.

***Quo ad sanationam:*Dubia ad bonam**

The prognosis for recovery is good because significant ASDs can be definitively corrected through transcatheter device closure or surgical closure. In small ASDs, the defect can even shrink or close spontaneously. However, the outcome depends on the size and type of ASD, hemodynamic

status, and the presence or absence of complications such as pulmonary hypertension and Eisenmenger syndrome.

CONCLUSION

Atrial Septal DefectASD, especially the secundum type, is a congenital heart disease that is generally well tolerated in childhood, but can cause complications if the left-to-right shunt is long-standing. Key findings indicate that ASD can cause right ventricular volume overload, right atrial and ventricular dilation, exercise intolerance, atrial arrhythmia, pulmonary hypertension, and even Eisenmenger syndrome in advanced stages. Echocardiography is the primary examination to confirm the diagnosis and assess the hemodynamic impact. Treatment is determined by the type and size of the defect, the extent of the shunt, symptoms, and right ventricular enlargement. Small ASDs can be monitored for their potential to shrink or close spontaneously, while ASDs with significant hemodynamic impact may require transcatheter device closure or surgical closure. The prognosis is generally good if ASD is treated early before it develops into severe complications.

The limitations of this discussion lie in the use of a literature review, which did not yield primary data and could not directly describe the condition of ASD patients. Furthermore, the epidemiological data on ASD in Indonesia used is still limited and comes partly from hospital-based studies. Future research is recommended to utilize population-based studies and a more comprehensive congenital heart disease registry system to obtain a more accurate picture of the epidemiology of ASD. Practically, the results of this discussion emphasize the importance of detection, echocardiographic evaluation, regular monitoring, and determining the appropriate timing and method of closure based on hemodynamic impact. Parental education regarding symptoms, growth, physical activity, and the importance of regular check-ups is also needed to support management and prevent long-term complications.

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