Pathophysiology, Investigations, and Management of Atrial Septal Defect

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Abstract

Background: ASD (atrial septal defect) is one of the most prevalent types of congenital heart disorders, affecting around one-quarter of all children. When the link between the right and left atria is not closed, an atrial septal defect arises. It includes both real septal membrane abnormalities and additional anomalies that allow communication between the two atria. From most common to least common, there are five types of atrial septal defects: patent foramen ovale, ostium secundum defect, ostium primum defect, sinus venosus defect, and coronary sinus defect. In most cases, small atrial septal abnormalities close on their own throughout development. Large lesions that do not seal on their own may require percutaneous or surgical intervention to avoid consequences like stroke, dysrhythmias, and pulmonary hypertension. This exercise covers the assessment, diagnosis, and treatment of atrial septal defect, as well as the importance of team-based interprofessional care for individuals with the condition.

Conclusion: The goal of this review article is to identify the etiology of the atrial septal defect, describe the presentation of a patient with atrial septal defect, describe the treatment and management options available for atrial septal defect, and explain interprofessional team strategies for improving care coordination and communication for patients with the atrial septal defect.

Keywords*: Atrial septal defect; Congenital heart disease; Ostium primum defect; Ostium secundum defect; Pulmonary arterial hypertension; Sinus venosus defect*

Introduction

An atrial septal defect is one of the most common congenital heart disorders that appear in adulthood (ASD). ASD is caused by a defect in the interatrial septum, which allows pulmonary venous return from the left atrium to flow directly into the right atrium. Depending on the extent of the defect, the size of the shunt, and other defects, this might result in anything from no noticeable cardiac repercussions to right-sided volume overload, pulmonary arterial hypertension, and even atrial arrhythmias. In comparison to previous incidence studies that used catheterization, surgery, or autopsy for diagnosis, the routine use of echocardiography has enhanced the identification and consequently the incidence of ASD. The lack of evident physical examination findings and often modest symptoms in the first 2-3 decades of life contribute to a delay in diagnosis, with the majority of cases (more than 70%) detected by the fifth decade (1).

Pathophysiology

The size of the atrial septal defect (ASD), relative compliance of the ventricles, and relative resistance in the pulmonary and systemic circulations all influence the amount of the left-to-right shunt. Left atrial pressure can be several millimeters greater than right atrial pressure with a minor ASD, but mean atrial pressures are nearly equivalent with a large ASD. Late ventricular systole and early diastole are the most common times for shunting over the interatrial septum, which is usually left-to-right. There is presumably some amplification during atrial contraction. A transient and modest right-to-left shunt can occur even in the absence of pulmonary arterial hypertension, especially during respiratory periods of decreasing intrathoracic pressure (2).

A chronic left-to-right shunt causes increased pulmonary blood flow and diastolic overload of the right ventricle. Even though pulmonary blood flow may be more than twice that of systemic blood flow, resistance in the pulmonary vascular bed in children with ASD is usually normal, and the volume load is usually well tolerated. Ventricular compliance can vary as people get older, resulting in an increased left-to-right shunt that might produce symptoms. Pneumatic arterial hypertension, shunt reversal, and Eisenmenger syndrome can all occur from a significant chronic left-to-right shunt. Due to an increase in plasma volume during pregnancy, shunt volume may increase, causing symptoms. In most cases, the pressure in the pulmonary arteries is normal (3).

Causes and Risk Factors

An atrial septal defect is a congenital heart abnormality caused by a spontaneous deformation of the interatrial septum (ASD). The various types of ASD are as follows: ASD second ostium This type of ASD is caused by an incomplete adhesion between the flap valve associated with the foramen ovale and the septum secundum after birth. The abnormal septum primum resorption during the formation of the foramen secundum causes the patent foramen ovale.

Resorption in unusual locations causes a fenestrated or netlike septum primum. Excessive resorption of the septum primum results in a short septum primum that does not close the foramen ovale. An overly large foramen ovale could occur from a defect in the septum secundum's development. This form of aberrant foramen ovale is not closed by the usual septum primum at birth (4).

Autism Spectrum Disorder (ASD) is a hereditary disorder that can run in families. Holt-Oram syndrome is characterized by an autosomal dominant inheritance pattern and upper-limb deformities and has been attributed to a single gene defect in TBX5 (most commonly, missing or hypoplastic radii). The penetrance rate of HoltOram syndrome is about 100 percent. New mutations are responsible for around 40 percent of Holt-Oram cases. Ellis van Creveld syndrome is a 60 percent autosomal recessive skeletal dysplasia characterized by short limbs, short ribs, postaxial polydactyly, dysplastic nails and teeth, and a common atrium. Mutations in the cardiac transcription factor NKX2.5 have been associated with the syndrome familial ASD with progressive atrioventricular block. This is an autosomal dominant trait with high penetrance but no associated skeletal abnormalities. Variations in the GATA4 gene have also been associated with ASD. A novel methylation site mutation in GATA4 (c.A899C, p.K300T) has recently been related to ASD (figure 1) (5).

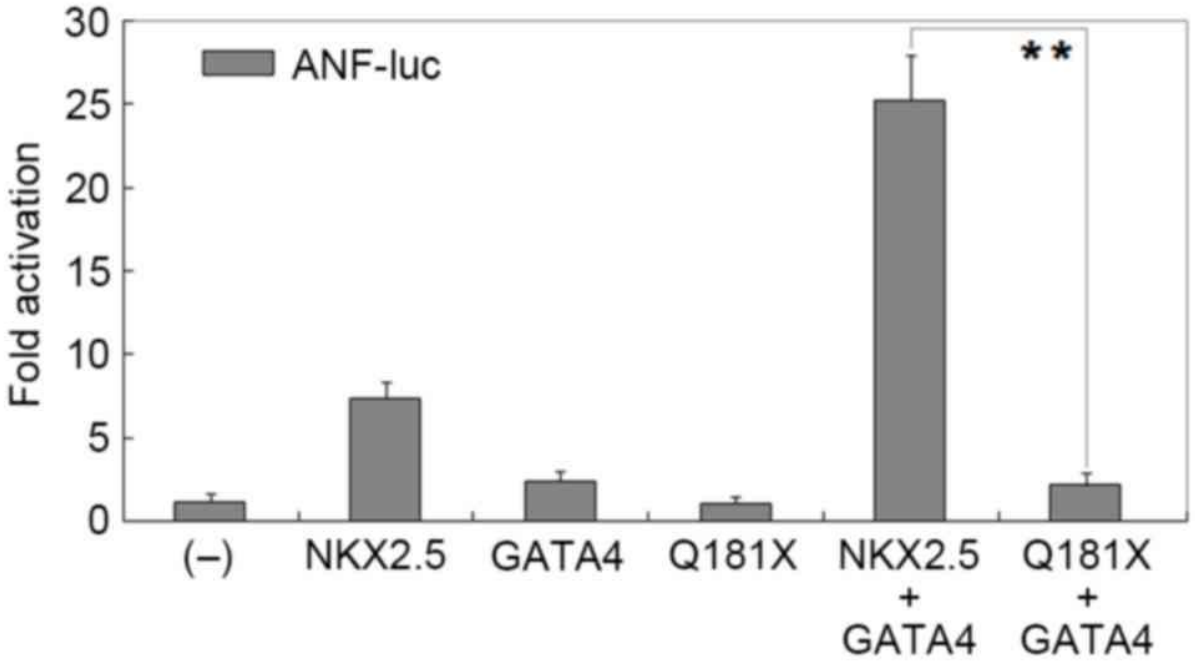

Figure 1 Abrogated synergistic activation between NKX2.5 and GATA4 caused by mutation. Activation of the ANF promoter driven luciferase in COS-7 cells by wild-type NKX2.5 or Q181X-mutant NKX2.5 (Q181X), in the presence of GATA4, revealed disrupted synergistic activation by the mutant protein. Experiments were performed in triplicate and data are expressed as the mean ± standard deviation. **P<0.001, compared with its wild-type counterpart. NKX2.5, NK2 homeobox 5; ANF, atrial natriuretic factor; GATA4, GATA binding protein 4 (5).

According to a doctor, downregulation of the following genes in ASD may influence the formation of the atrial septum, cardiomyocyte proliferation, and cardiac muscle development: Extracellular signal molecules VEGFA and BMP10, cardiac sarcomeric proteins MYL2, MYL3, MYH7, TNNT1, and TNNT3, According to the researchers, dysregulation of these genes during heart septum morphogenesis may lead to cell cycle as the dominant pathway among downregulated genes, with reduced expression of cell cycle proteins potentially disrupting cardiomyocyte growth and differentiation during atrial septum formation (5).

Types of ASD

The following are the five primary forms of ASD: An atrial septal defect (ASD) is a hole in the interatrial septum that allows blood to flow freely between the atria. ASD is divided into four categories based on the location of the septal defect. The most prevalent type of ASD is the Ostium secundum, which affects the fossa ovalis in the mid-septal region. Mitral valve prolapse is connected with 20% of these instances (MVP). The Ostium primum kind of ASD is uncommon, and the defect is located around the AV valves. The AV valves may be distorted as well. The sinus venosus form of ASD is likewise uncommon; the defect is visible high in the atrial septum near the superior vena cava (SVC) entry, and the coronary sinus type of ASD is also uncommon (figure 2) (6).

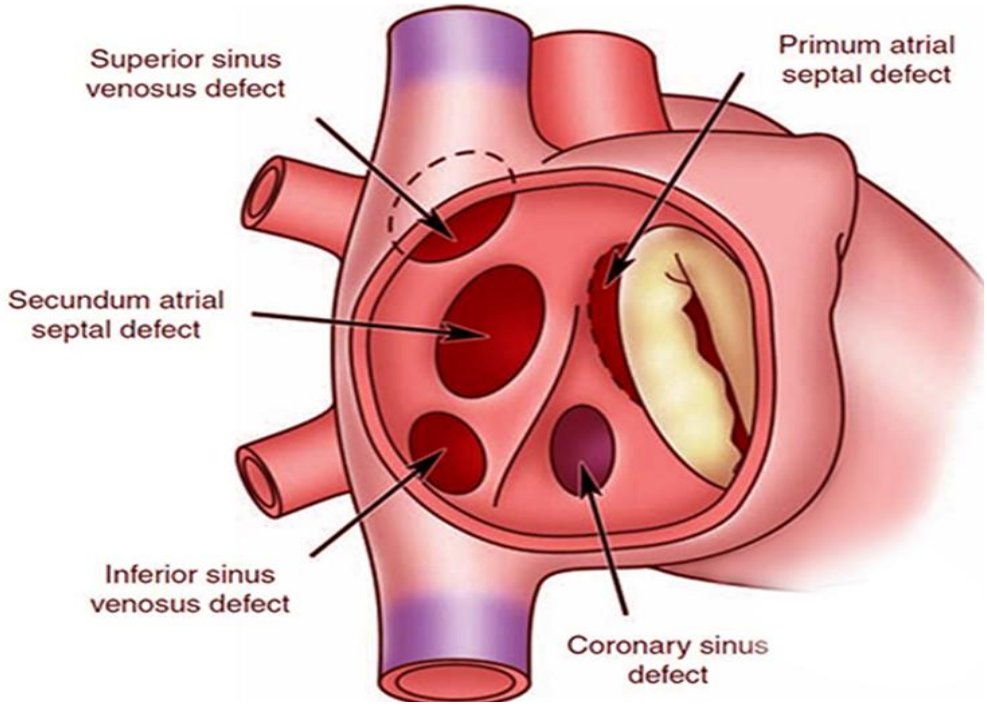

Figure 2 the five types of ASD (6)

Symptoms and Presentation

When a baby is born with an atrial septal defect, many of them have no symptoms or signs. Signs and symptoms of adulthood can appear. Some of the signs and symptoms of an atrial septal defect include: Shortness of breath is a common ailment, especially when exercising. Fatigue, Swelling in the legs, feet, or abdomen Heart palpitations, also known as skipped

beats, is a type of heart palpitation that occurs when the heart produces a whooshing sound that can be heard with a stethoscope. Heart murmur (a whooshing sound that can be heard with a stethoscope) and stroke (7).

The atrial septal defect (ASD) anomaly can be misdiagnosed for decades due to minor physical screening results and a lack of symptoms. Even isolated anomalies of moderate to large magnitude may not cause symptoms in youngsters. Symptoms such as tiredness, recurring respiratory infections, or exertional dyspnea may occur in certain persons. If a heart murmur is identified during a routine physical examination or after an abnormal result on chest radiographs or an electrocardiogram, the diagnosis is routinely examined in youngsters (ECG). If untreated in childhood, symptoms may appear gradually over decades and are mostly the result of decreased compliance with age, pulmonary arterial hypertension, atrial arrhythmias, and, in some cases, those associated with mitral valve disease in a primum ASD. ASD patients who live into their sixth decade are almost all symptomatic (8).

Several variables contribute to the clinical decline in elderly persons, including the following: An age-related decrease in left ventricular compliance contributes to the left-to-right shunt. Second, atrial arrhythmias become more common during the fourth decade, especially atrial fibrillation, but also atrial flutter or paroxysmal atrial tachycardia, which can lead to right ventricular failure. Third, most symptomatic people over 40 have mild-to-moderate pulmonary arterial hypertension due to a persistent substantial left-to-right shunt, which puts pressure and volume overload on the right ventricle, which is aging. Clinically significant mitral regurgitation is another cause of symptoms, particularly those connected to primum ASD. The frequency, scope, and severity of dysfunction increase as people get older. A rise in left atrial pressure and a larger degree of left-to-right shunt result from mitral valve insufficiency. The most common presenting symptoms are dyspnea, easy fatigability, palpitations, persistent atrial arrhythmia, syncope, stroke, and/or heart failure. One of the most common symptoms in adults is palpitations caused by atrial arrhythmias (8).

Examination

The size of the defect, the diastolic properties of both ventricles, and the relative resistance of the pulmonary and systemic circulations determine the degree of left-to-right shunt and its hemodynamic consequences, which are determined by the size of the defect, the diastolic properties of both ventricles, and the relative resistance of the pulmonary and systemic circulations. Keep in mind the following: The patient may have a hyperdynamic right ventricular impulse due to high diastolic filling and large stroke volume. A detectable pulmonary artery pulse and an ejection click are signs of a dilated pulmonary artery. S1's second component is frequently divided, and the second component's strength may be increased, indicating robust right ventricular contraction and delayed tricuspid valve closure. S2 is typically widely separated and fixed due to lesser respiratory variation due to delayed pulmonic valve closure (seen only if pulmonary artery pressure is normal and pulmonary vascular resistance is low). Almost all patients with big left-to-right shunts have this distinguishing anomaly (9).

Blood flow over the ASD does not induce a murmur at the shunt site because there is no considerable pressure differential between the atria. Due to increased right ventricular stroke volume over the pulmonary outflow pathway, ASD with moderate-to-large left-to-right shunts creates a crescendo-decrescendo systolic ejection murmur. This murmur can be heard in the second intercostal zone of the upper left sternal border. Patients with significant left-to-right shunts usually have a rumbling mid-diastolic murmur near the lower left sternal boundary due to increased flow across the tricuspid valve. The auscultatory indications of ASD can be mistaken for minor valvular or infundibular pulmonic stenosis, as well as idiopathic pulmonary artery dilatation. There is a systolic ejection murmur in all of these disorders, but unlike ASD, the S2 shifts with breathing, there is no pulmonary ejection click, and there is no tricuspid flow murmur (9).

Patients with an ostium primum defect and a cleft of the mitral valve may hear an apical systolic regurgitant murmur of mitral regurgitation. People who develop pulmonary arterial hypertension and right ventricular hypertrophy may have a right ventricular S4. In these circumstances, the midsystolic pulmonic murmur is softer and shorter, the tricuspid flow murmur is missing, the splitting of S2 is smaller with an emphasized pulmonic component, and pulmonic regurgitation murmur is possible. ASD is a cyanotic condition. As a result, the patient's saturation level should be normal. In the rare case of severe pulmonary arterial hypertension, atrial shunt reversal (Eisenmenger syndrome) might occur, resulting in cyanosis and clubbing (10).

Complications

A little atrial septal defect might not create any problems. Small atrial septal defects often close during childhood. Larger faults can lead to serious consequences like right-sided heart failure, abnormal heart rhythms (arrhythmias), a higher risk of stroke, and a shorter life span. If a large atrial septal defect is left untreated, increased blood flow to the lungs raises blood pressure in the lung arteries, which is a serious issue (pulmonary hypertension). Pulmonary hypertension, also known as Eisenmenger syndrome, can harm the lungs for the rest of one's life. Eisenmenger syndrome is a chronic illness that affects only a tiny fraction of people who have substantial atrial septal defects. Treatment can help you avoid or control a lot of these problems (11).

Pregnancy and atrial septal defect: Most women with an atrial septal defect can carry a child without any difficulties. Having a larger defect or consequences such as heart failure, arrhythmias, or pulmonary hypertension can increase pregnancy difficulties. Doctors strongly advise women with Eisenmenger syndrome not to become pregnant since it could jeopardize their lives. Congenital heart disease is more likely to develop in children whose parents have the condition. If you're thinking about creating a family and you have a congenital heart defect, whether it's corrected or not, you should see a doctor. The doctor may advise repair before conception (11).

Prevention

Abnormalities of the atrioventricular septum are virtually invariably irreversible. If the patient intends to become pregnant, make an appointment with their health care practitioner. The following items should be included in this visit: Getting a rubella immunity test: If the patient is not immune, ask the doctor about being vaccinated.Examining the current state of the patient's health and medications: The doctor will need to keep an eye on specific health issues during the pregnancy. Your doctor may urge you to change or stop using certain drugs before becoming pregnant.Examining the family's medical history: If the patient has a family history of cardiac defects or other genetic disorders, consult a genetic counselor to determine the risk. (12).

Investigations

A doctor may suspect an atrial septal defect or similar cardiac problem in a child after hearing a heart murmur during a checkup. If a heart defect is suspected, the doctor may prescribe one or more of the following tests (12).

Laboratory Studies

No particular laboratory blood testing is suggested in the case of atrial septal anomalies (ASDs). In patients undergoing ASD intervention, routine laboratory tests such as complete blood cell (CBC) count, Type, and screen, Metabolic profile or chemical panel, and coagulation investigations should be performed (prothrombin time [PT] and activated partial thromboplastin time [aPTT]) (13).

Echocardiogram

Transthoracic 2-dimensional (2-D) echocardiography clarifies an ambiguous diagnosis by providing noninvasive direct visualization of most types of atrial septal defects (ASDs), including evaluation of the right atrium, right ventricle, and pulmonary arteries, as well as other associated abnormalities. Often, the subcostal viewpoint is the most beneficial. An exception is the identification of a sinus venosus defect, for which transesophageal echocardiography (TEE) may be necessary to image the defect although TEE may still be unable to see the pulmonary venous return. Any patient with an ASD, especially those with a sinus venosus defect, should be checked for abnormalities of the systemic venous connection. These can be easily identified using two-dimensional imaging. A TEE should be performed if there is an enlargement of the right atrial and right ventricular chambers without a known cause. Doppler echocardiography can help illustrate flow through the atrial septum. It has a biphasic (systolic and diastolic) rhythm with a little right-to-left shunt at the beginning of the systole. Real-time (RT) 3-dimensional (3D) Doppler TEE can also provide detailed and precise information on the appropriate occluder device, as well as facilitate transcatheter occlusion by guiding the catheter through the frequently challenging anatomy of the patient (figure 3) (14).

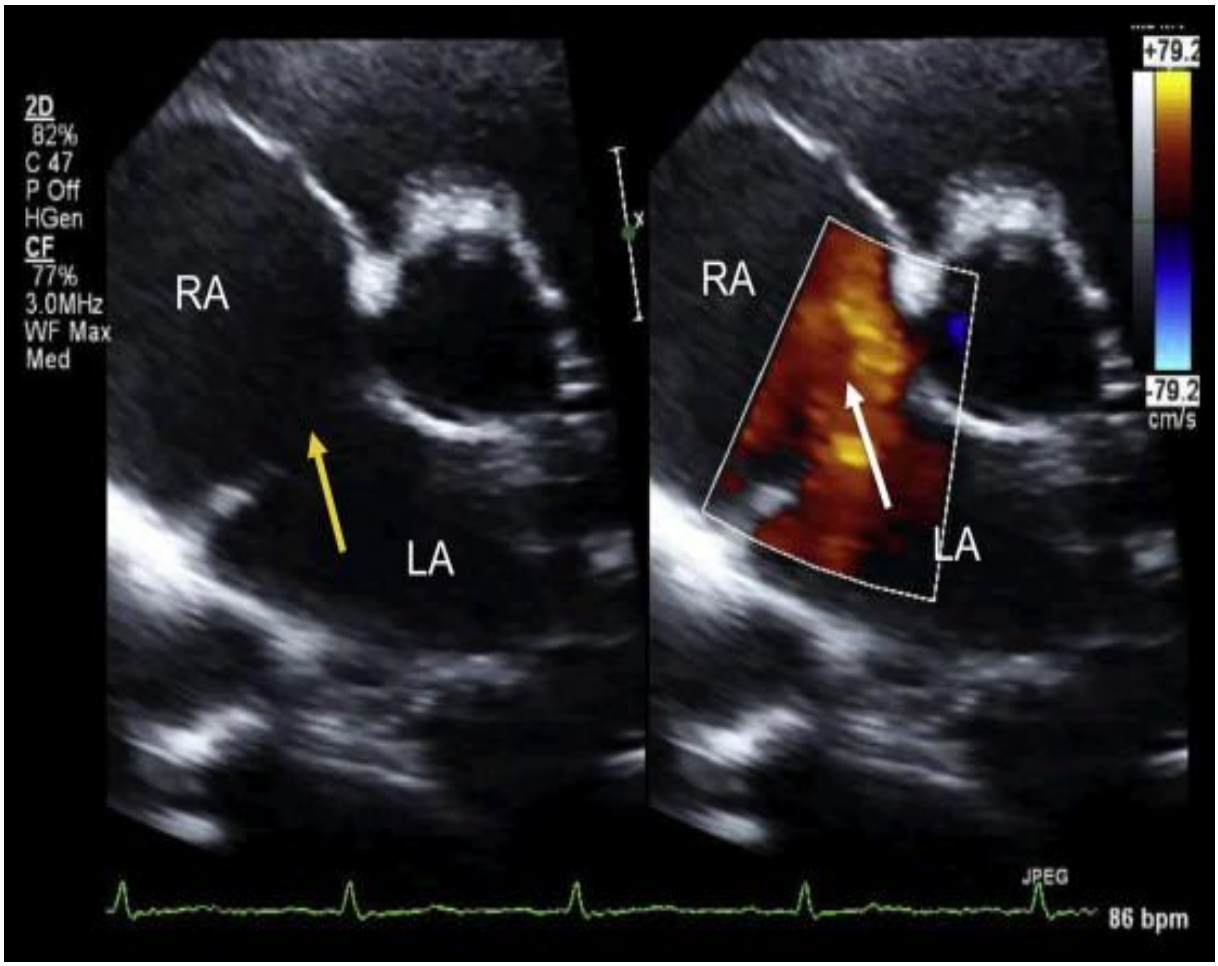

Figure 3 echocardiography shows Atrial Septal Defect (14)

Transthoracic echocardiography (TTE) may be inadequate in some patients with narrow echocardiographic windows. In such instances, TEE can offer excellent atrial septum delineation. TEE can also be utilized to guide device placement and offer instant intraoperative confirmation that the defect has been closed during catheter-assisted ASD occlusion procedures. Continuous-wave Doppler echocardiography is useful for measuring right ventricular (and pulmonary arterial when there is no associated right ventricular outflow tract obstruction) systolic pressure when a tricuspid regurgitant jet is present. This technique can also be used to check for pulmonary venous return obstruction in patients. To confirm the diagnosis, contrast echocardiography can be employed. A right-to-left shunt can be detected by micro cavitation bubbles in the left atrium and left ventricle. A left-to-right shunt can be identified in the right atrium as a negative contrast washout effect (15).

Chest X-ray

Cardiomegaly is most commonly seen on chest radiographs when there is a clinically significant left-to-right shunt due to dilatation of the right atrium and right ventricular chamber. The pulmonary artery is prominent in the lung fields, and pulmonary vascular markings are expanded. Left atrial enlargement is only caused by clinically severe mitral regurgitation. Proximal dilation of the superior vena cava may be noticed in the case of a sinus venosus defect (figure 4) (16).

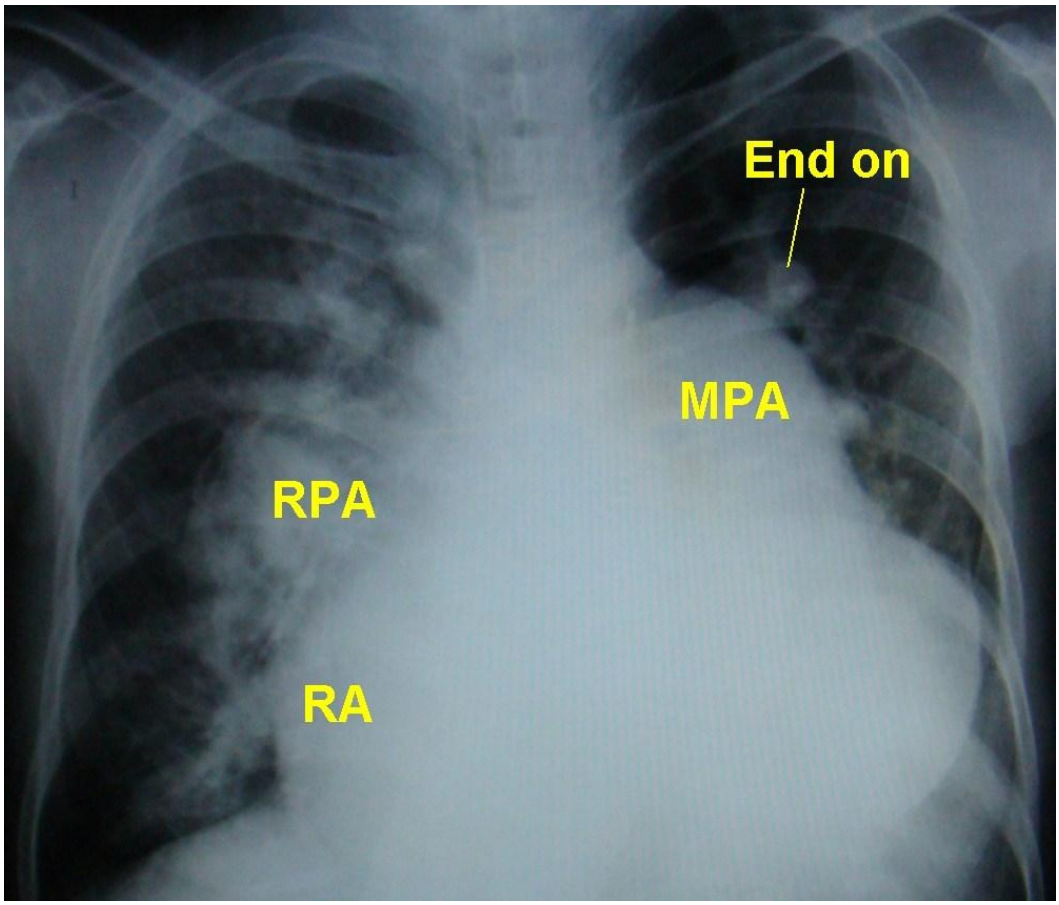

Figure 4 this chest X-ray in ASD shows grossly dilated main pulmonary artery (16)

Electrocardiogram (ECG)

In patients with Secundum atrial septal defect (ASD), a normal sinus rhythm, right-axis deviation, and an rSR' pattern in V1, as well as an interventricular conduction delay or right bundle branch block, are all common findings (which represents delayed posterobasal activation of the ventricular septum and enlargement of the right ventricular outflow tract). Left-axis deviation and an rSR' pattern in V1, as well as an interventricular conduction delay or right bundle branch block, are all signs of an ostium primum defect. Left-axis deviation with a negative P wave in lead III indicates a sinus venous abnormality. When pulmonary hypertension rises, the rSR' pattern in V1 can be lost, and a tall monophasic R wave with a deeply inverted T wave can appear. In familial ASD or ostium primum, a prolonged P-R interval might be observed due to left atrial enlargement and a larger distance for internodal conduction induced by the defect itself. In some cases, the AV node has been discovered to be shifted in a posteroinferior direction, as well as an expanded right atrium (figure 5) (17).

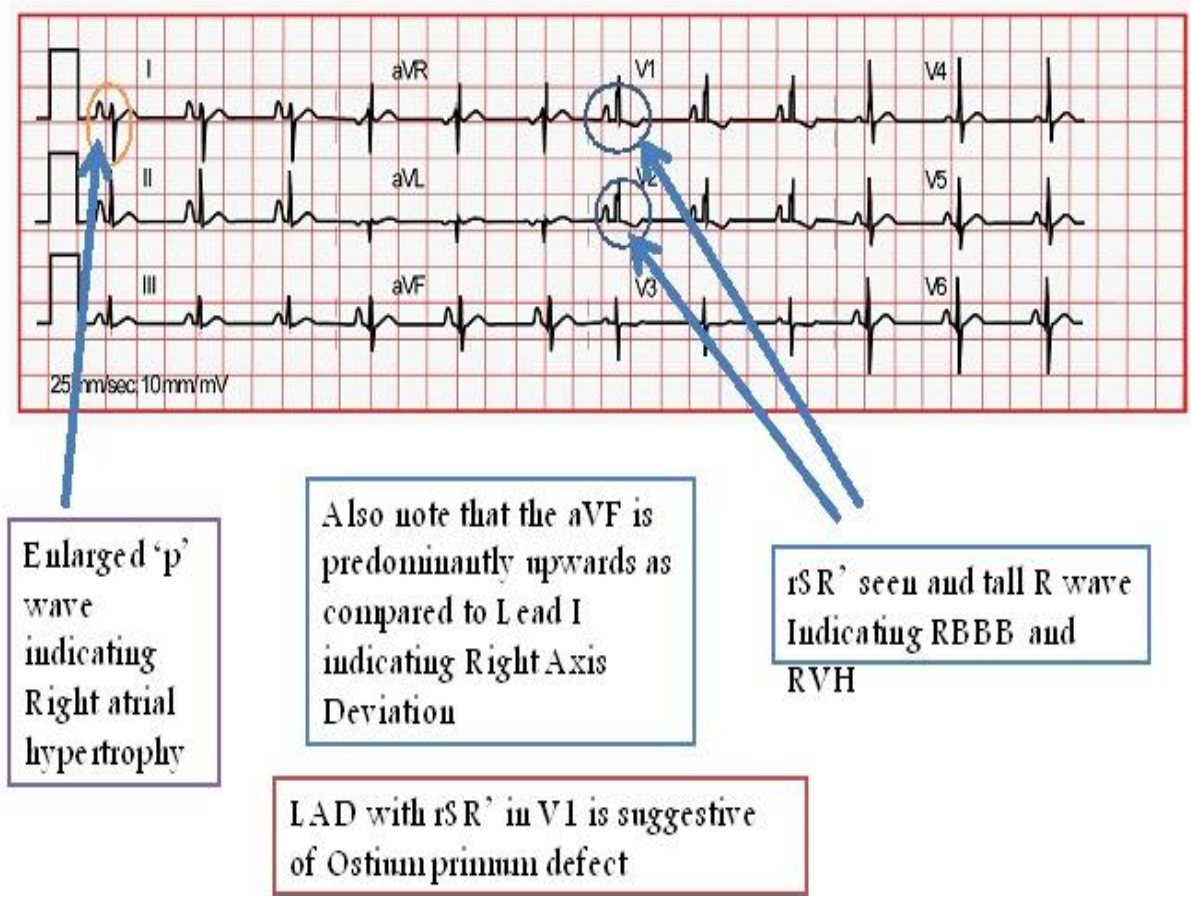

Figure 5 electrocardiogram of Atrial Septal Defect (17)

Cardiac catheterization

A catheter is a thin, flexible tube that is guided to the heart through a blood artery in the groyne or arm. Catheterization allows doctors to evaluate congenital heart disease, test how well the heart pumps, monitor heart valve function, and measure blood pressure in the lungs (figure 6) (18).

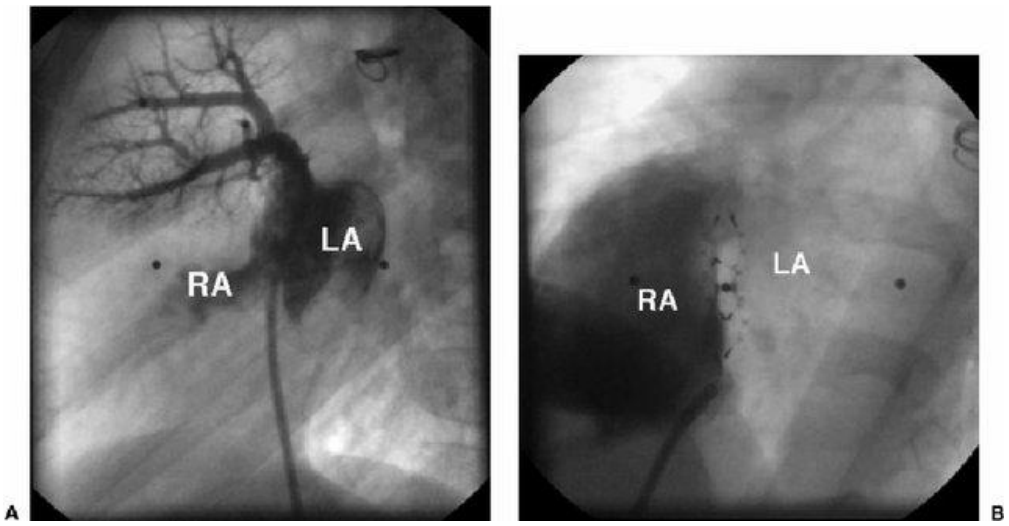

Figure 6 repair of an atrial septal defect. (A) A contrast shunting left-to-right through a moderate-sized atrial septal defect into the right atrium is shown on an angiography done in the left atrium (LA) (RA). The catheter is positioned across the LA defect. In addition, contrast is injected into the left upper pulmonary vein. (B) An angiography conducted in the RA after the implantation of a CardioSEAL ASD occlusion device (NMT Medical Inc., Boston, MA) shows that the double umbrella device is well positioned, resulting in an unbroken atrial septum (18).

MRI

MRI has been used to successfully identify the extent and location of ASD. On the other hand, minor errors are of minimal utility. A primary advantage of MRI is the capacity to quantify right ventricular size, volume, and function, as well as identify systemic and pulmonary venous return (figure 7) (19).

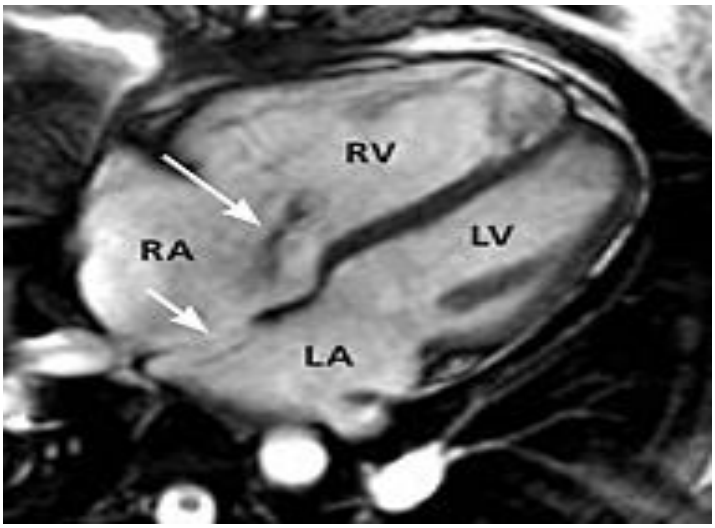

Figure 7 one of the atrial septal flaws (short arrow) is seen on magnetic resonance imaging, as well as enlargement of the right atrium and right ventricle. A systolic regurgitant flow jet (long arrow) can also be seen coming from the tricuspid valve in this image (19).

CT scan

Using a series of X-rays, this method provides comprehensive images of the heart. It can be used to diagnose an atrial septal defect and concomitant congenital heart defects if they have not been definitively established by echocardiography (figure 8)(20).

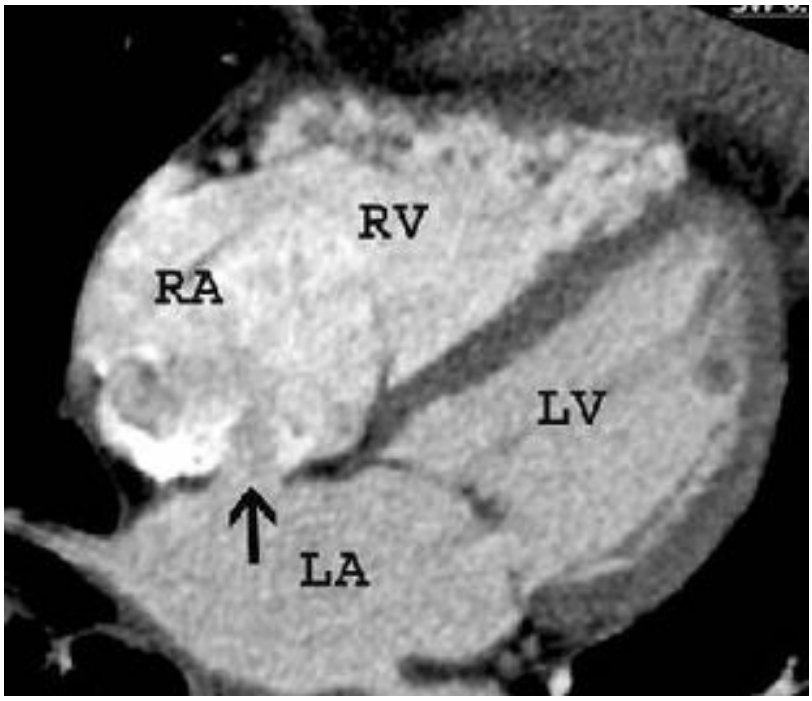

Figure 8 the contrast jet extends from the left atrium to the right atrium in this CT picture showing a defect in the fossa ovalis (arrow) (20).

Management

Many atrial septal defects close on their own during childhood. If a tiny atrial septal abnormality does not close, it may not require treatment. However, many persistent atrial septal defects will require surgery in the future. The cardiologist may recommend that if the child has an atrial septal defect, it be watched for some time to determine if it closes on its own. The doctor will evaluate when the child needs therapy based on the issue and whether the infant has other congenital cardiac problems. Medications: Although drugs will not close the hole, they may help to relieve some of the symptoms of an atrial septal defect. Drugs may also be administered to reduce the risk of complications following surgery. Medications to keep the heartbeat regular (beta-blockers) or to lessen the danger of blood clots may be prescribed (anticoagulants) (21).

Surgery

Many specialists recommend repairing a medium to large atrial septal defect diagnosed in childhood or later in life to minimize future complications. Surgery is not recommended if the patient has severe pulmonary hypertension since it may worsen the condition. In both adults and children, the abnormal opening between the atria is stitched closed or patched. Doctors will evaluate the problem and suggest one of two treatments (22):

Minimally invasive approaches

In recent years, minimally invasive approaches for the treatment of ASD have aroused a lot of attention. Alternate methods of cardiopulmonary bypass usually include reducing the size of the incision. Examples include partial or complete submammary skin incisions,

hemisternotomy, and restricted thoracotomy. The goal of these procedures is to improve cosmetic effects because they are not associated with lower morbidity or death. Completely endoscopic minimally invasive surgery may be a viable alternative to catheter-based ASD therapy in persons with unfavorable anatomy or clinical contraindications. In a retrospective study, the results of fully endoscopic closure using a glutaraldehyde-treated autologous pericardial patch in 37 Japanese patients with ASD who had been postponed from transcatheter surgery were favorable (2011-2015). According to the researchers, there were no surgical deaths, post-procedure ASD reinterventions, or heart failure readmissions, and follow-up echocardiography revealed no recurrent shunt or calcification of the autologous pericardial patch (figure 9) (23).

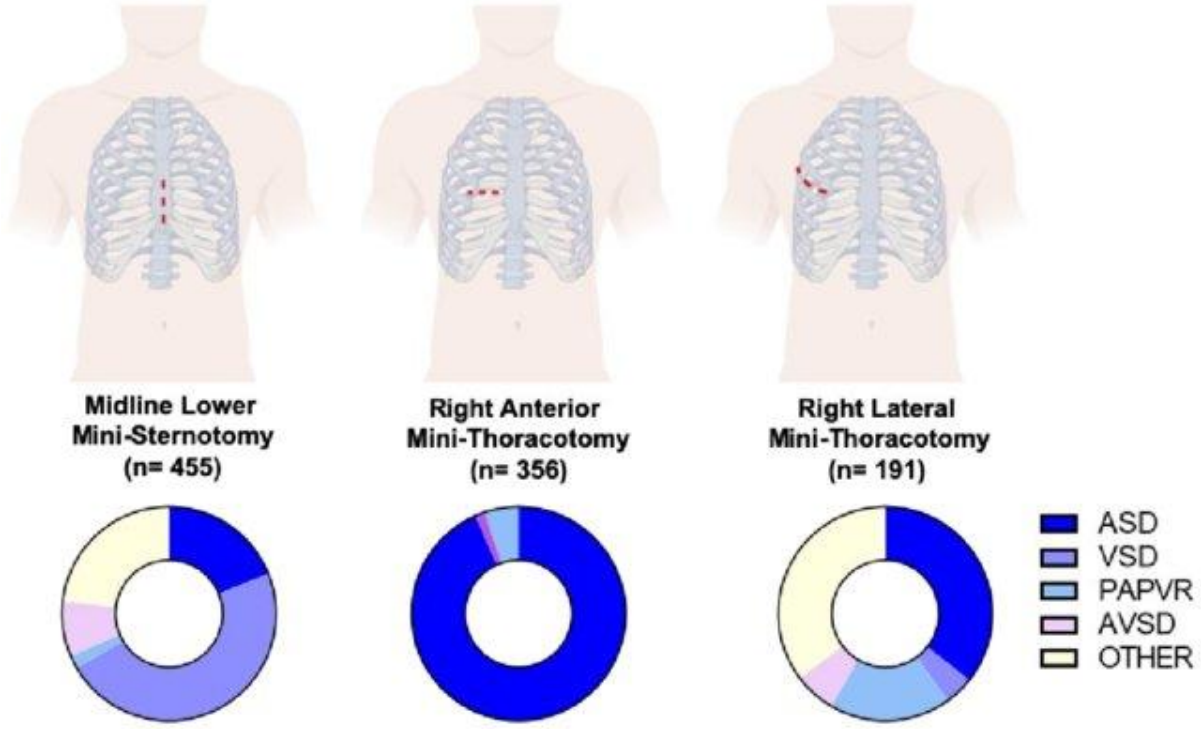

Figure 9 access for minimally invasive cardiac surgery and diagnoses. (Graphical representation of the three different type of access for minimally invasive cardiac surgery used. On the bottom, pie charts illustrate the distribution of diagnoses according to surgical access. ASD: Atrial Septal Defect; VSD: Ventricular Septal Defect; PAPAVR: Partial Anomalous Pulmonary Venous Return; pAVSD: Partial Atrioventricular Septal Defect) (23)

Percutaneous transcatheter closure

Secundum ASDs have recently successfully closed using a variety of catheter-implanted occlusion devices rather than direct surgery with cardiac bypass. These umbrella-like devices are implanted through the femoral vein and utilized to close the septal defect. These devices are especially useful for centrally located Secundum faults. Even though surgical closure has a low rate of morbidity and death and excellent long-term outcomes, sternotomy and cardiopulmonary bypass are necessary. Drs. King and Mills performed the first transcatheter closure of a secundum ASD in the mid-1970s. William Rashkind was the first to design a

percutaneous ASD closure method in the late 1970s. Jim Lock devised the clamshell technique in 1989. At the same time, Sideris commenced clinical trials with a buttoned device (24).

Even though a variety of devices have been investigated, four major devices have recently become available: CardioSEAL, Amplatzer septal occluder (ASO), HELEX septal occluder, and Sideris patch. The ASO is presently the most widely used device because it is straightforward to implant and facilitates the closure of large orifices with high success rates in most scenarios. It was used in humans for the first time in 1995. Choosing a specific device is difficult because no randomized research has been conducted. Furthermore, present devices do not allow for percutaneous closure of ostium primum and sinus venous abnormalities (figure 10) (25).

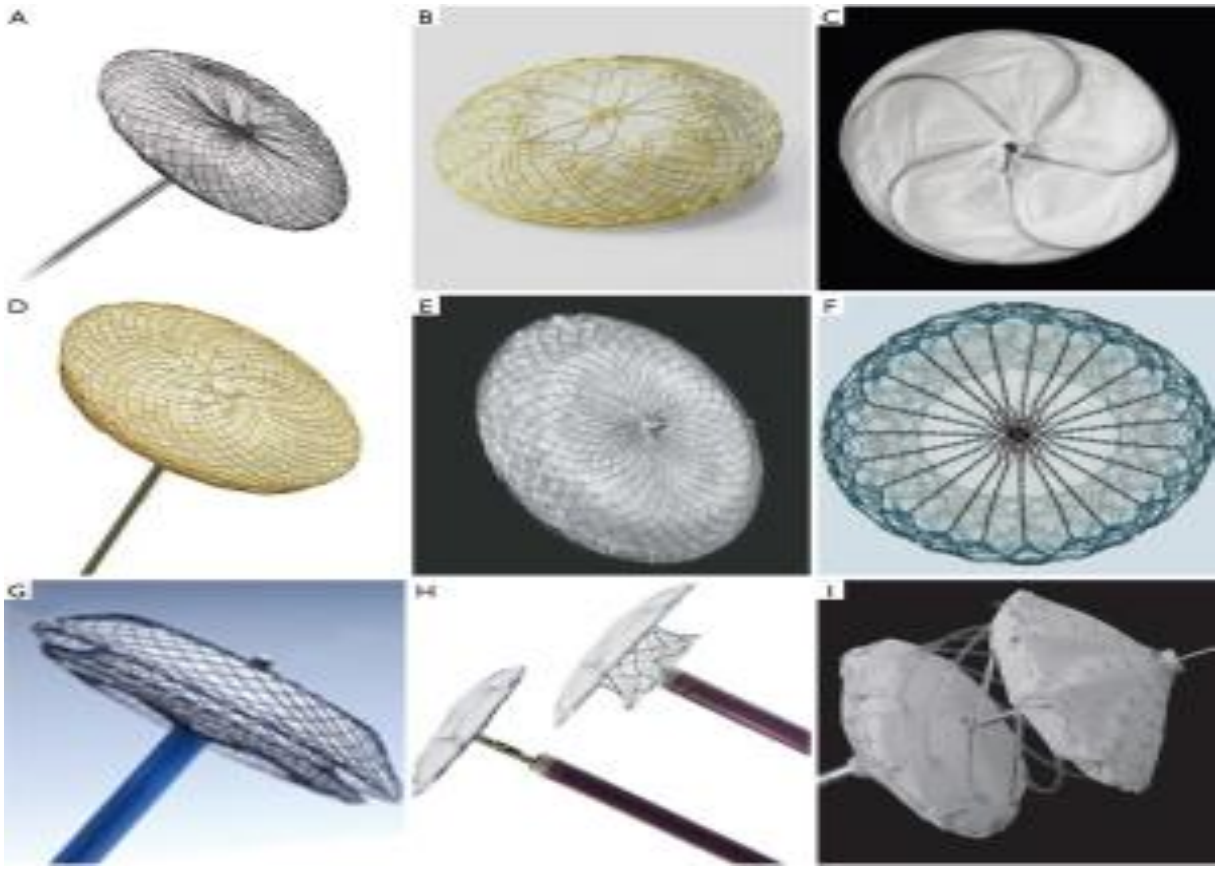

Figure 10 currently available devices for atrial septal defect closure. (A) Amplatzer Septal Occluder; (B) Occlutech Figulla Flex II device; (C) Gore Cardioform Septal Occluder; (D) Cocoon Septal Occluder; (E) CeraFlex ASD device; (F) Nit Occlud ASD-R device; (G) Cardi-O-Fix Septal Occluder; (H) Ultracept II ASD Occluder and (I) Carag Bioresorbable Septal occlude (25).

In this process, transesophageal echocardiography is utilized to assess the static diameter of the defect. The diameter is then measured with a sizing balloon, and the device's proper diameter is determined using the "stop flow" method. TEE is used in this treatment to inflate the sizing balloon until no flow is visible through the defect. The orifice's margins must be large enough (5 mm) to allow the edges of the closing device. Although it can be painful, TEE

has long been the primary procedure for measuring, inserting, and deploying medical devices. Airway protection and general anesthesia are also required. Intracardiac echocardiography has been used in the same way. Transcatheter closure of ASDs is now standard therapy in most cardiac clinics. With over 96 percent successful implantation rates, it is safe in skilled hands, cost-effective, and compares favorably to surgical closure. Transcatheter closure has been linked to fewer problems, shorter hospital stays, and less blood product usage (26).

At any age, ASD repair results in symptomatic improvement, regression of positive airway pressure (PAP), and right ventricular enlargement; however, persons with less functional impairment and lower PAP have the best prognosis. Because symptoms, right ventricle remodeling, and PAP all rise with age, ASD closure should be recommended as soon as possible following diagnosis, regardless of symptoms, especially in the elderly. Furthermore, when compared to surgery, transcatheter closure appears to provide additional hemodynamic benefits. According to one study, after transcatheter closure with ASO, the left atrial volume index left ventricular myocardial performance index, and right ventricular myocardial performance index all improved. The last was disappointing after surgery, likely because of cardiopulmonary bypass (27).

Another study compared atrial function in 45 people with an average age of 9 years following surgery and after percutaneous closure using strain-rate imaging. They discovered that both atrial functions were intact after transcatheter closure, whereas this was not the case after surgery. An atriotomy scar could have harmed right atrial function, while perioperative hypoxia or intraoperative myocardial damage could have altered the deformation properties of the left atrium. A study of mid-to-long-term follow-up findings of successful transcatheter ASD closures in 179 patients older than 40 years found improvements in New York Heart Association (NYHA) functional class, pulmonary artery pressure, and cardiac rhythm. The study took 8.8 years to complete, with a median follow-up of 3.82.1 years (28).

The type of defect, the treatment recommended, and the existence of concomitant defects all influence follow-up care. When the child is discharged from the hospital, one year later, and as needed by the child's doctor, echocardiograms are conducted. Simple atrial septal defects that were closed during childhood normally only require follow-up treatment on a sporadic basis. Adults who have had an atrial septal defect fixed should be monitored throughout their lives for complications such as pulmonary hypertension, arrhythmias, heart failure, or valve anomalies. Once a year, follow-up exams are routinely performed (29).

Discussion

The three most common types of the atrial septal defect (ASD) account for 10% of all congenital heart disease and 20-40% of adult-onset congenital heart disease. The most common types of ASD are as follows: Second-generation ostium (second-generation ostium): The most common type of ASD, accounting for 75 percent of all ASD cases and roughly 7% of all congenital cardiac abnormalities and 30-40 percent of all congenital heart disease in those over the age of 40. Ostium primum: The second most common ASD, accounting for 15-20% of all ASDs, is Ostium primum. Primum ASD, or atrioventricular septal defect, is a form of atrioventricular septal defect that is usually linked to mitral valve issues. The SV ASD is

the least common of the three, accounting for just around 5% to 10% of all ASDs. On the superior side, the hole runs the length of the atrial septum. It is usual and anticipated for the right-sided pulmonary veins to join abnormally. In most cases, other imaging is required (30).

Women are more likely than males to have ASD, with a female-to-male ratio of roughly 2:1. Although the onset of clinical symptoms is depending on the degree of the left-to-right shunt, patients with ASD might remain asymptomatic throughout childhood. People's symptoms become more common as they get older. By the age of 40, 90% of untreated people have symptoms such as exertional dyspnea, fatigue, palpitation, chronic arrhythmia, or even signs of heart failure (31).

Summary and Conclusion

Atrial septal defects are a form of congenital heart defect in which tissue at the interatrial septum is either insufficient or missing. Uncorrected defects can cause right heart volume overload, atrial arrhythmia, and pulmonary arterial hypertension. The three most prevalent types of atrial septal defect are ostium secundum defect, ostium primum defect, and sinus venosus defect. Physical findings include a midsystolic pulmonary flow or ejection murmur, followed by a fixed split-second heart sound. Larger faults, on the other hand, may persist and create hemodynamic and clinical issues, necessitating percutaneous or surgical surgery. If you have severe pulmonary arterial hypertension, you should avoid closure.

Conflict of Interest

There is nothing to appear

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