Review Article

Pathophysiology, Investigations, and Management of Atrial Septal Defect

Abstract

Background: An atrial septal defect (ASD) is one of the most common congenital heart defects, 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 inter professional care for individuals with the condition.

Conclusion: Small defects may close on their own; larger abnormalities may persist and cause hemodynamic and clinical complications that necessitate percutaneous or surgical intervention.

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

Introduction

One of the most frequent congenital heart defects that manifests in adulthood is atrial septal defect (ASD). A deficiency in the interatrial septum causes pulmonary venous return from the left atrium to enter straight into the right atrium, causing ASD. This can result in a spectrum of disease ranging from no notable cardiac consequences to right-sided volume overload, pulmonary arterial hypertension, and even atrial arrhythmias, depending on the extent of the defect, size of the shunt, and related anomalies. In comparison to prior incidence studies that used catheterization, surgery, or autopsy for diagnosis, the identification and, thus, the incidence of ASD has increased with the routine use of echocardiography. The lack of obvious physical examination findings and often minor symptoms in the first 2-3 decades of life contribute to a delay in diagnosis into maturity, with the majority of cases (more than 70%) being diagnosed by the fifth decade (1).

Pathophysiology

The extent of the left-to-right shunt across an atrial septal defect (ASD) is determined by the defect size, relative compliance of the ventricles, and relative resistance in the pulmonary and systemic circulations. With a small ASD, left atrial pressure can be several millimetres higher than right atrial pressure, however with a big ASD, mean atrial pressures are practically comparable. Shunting over the interatrial septum happens most frequently in late ventricular systole and early diastole and is usually left-to-right. During atrial contraction, there is likely some amplification. Even in the absence of pulmonary arterial hypertension, a temporary and minor right-to-left shunt can occur, especially during respiratory periods of decreasing intrathoracic pressure (2).

Increased pulmonary blood flow and diastolic overload of the right ventricle ensue from a chronic left-to-right shunt. Even though pulmonary blood flow may be more than twice that of systemic blood flow, resistance in the pulmonary vascular bed is usually normal in children with ASD, and the volume load is usually well tolerated. With ageing, ventricular compliance can change, resulting in an increased left-to-right shunt, which can cause symptoms. A substantial chronic left-to-right shunt can modify pulmonary vascular resistance, resulting in pulmonary arterial hypertension, shunt reversal, and Eisenmenger syndrome. Shunt volume might increase due to an increase in plasma volume during pregnancy, causing symptoms. The pressure in the pulmonary arteries is usually normal (3).

Causes and Risk Factors

A congenital cardiac defect produced by a spontaneous deformation of the interatrial septum is known as an atrial septal defect (ASD). The following are the different forms of ASD: ASD ostium secundum After birth, there is an imperfect adhesion between the flap valve associated with the foramen ovale and the septum secundum, resulting in this kind of ASD. The patent foramen ovale is caused by aberrant septum primum resorption during the development of the foramen secundum. A fenestrated or netlike septum primum is caused by resorption in atypical areas. The septum primum resorbs excessively, resulting in a short septum primum that does not shut the foramen ovale. Defective development of the septum secundum might result in an excessively large foramen ovale. This form of aberrant foramen ovale is not closed by the usual septum primum at birth (4).

ASD is a genetic condition that can run in families. A single gene deficiency in TBX5 has been linked to Holt-Oram syndrome, which is characterised by an autosomal dominant inheritance pattern and upper-limb abnormalities (most commonly, missing or hypoplastic radii). HoltOram syndrome has a nearly 100% penetrance rate. New mutations are responsible for about 40% of Holt-Oram cases. Ellis van Creveld syndrome is an autosomal recessive skeletal dysplasia characterised by short limbs, short ribs, postaxial polydactyly, dysplastic nails and teeth, and a common atrium, which affects 60% of those affected. The syndrome familial ASD accompanied with progressive atrioventricular block has been linked to mutations in the cardiac transcription factor NKX2.5. This is an autosomal dominant trait with a high degree of penetrance but no skeletal deformities linked with it. ASD has also been linked to variations in the GATA4 gene. Recently, a new mutation in GATA4's methylation site (c.A899C, p.K300T) has been linked to ASD (figure 1) (5).

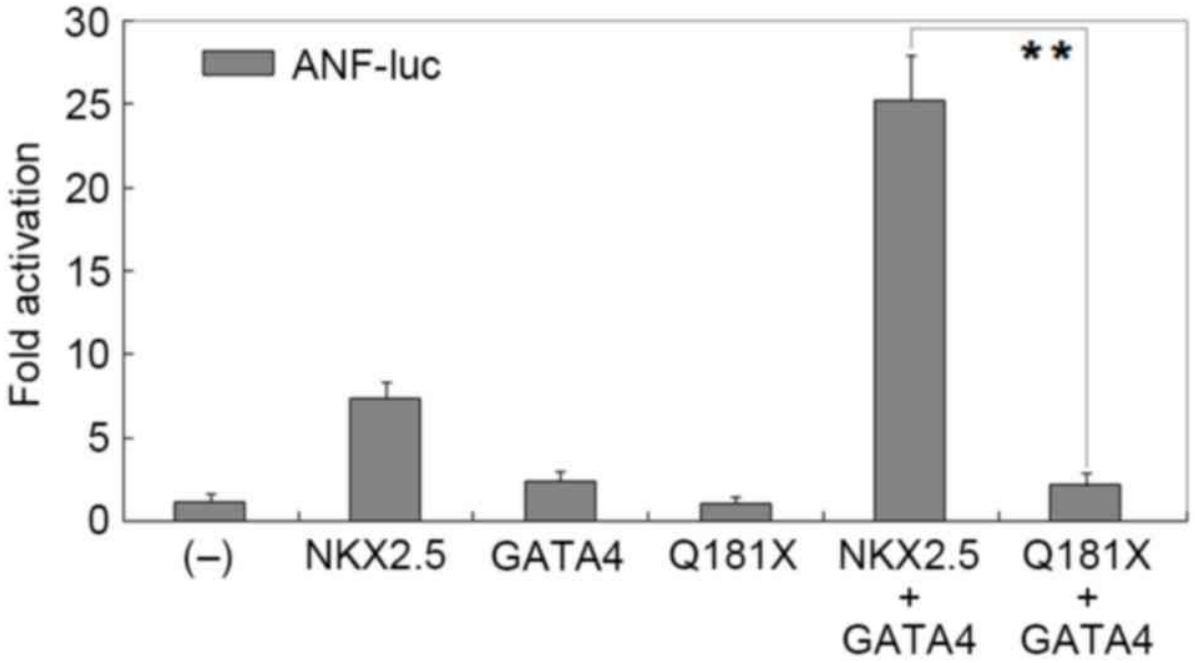

Figure 1 Abrogated synergistic activation between NKX2.5 and GATA4 caused by mutation. Activation of the ANF promoter driven luciferase in COS-7 cells by wild-type NKX2.5 or Q181X-mutant NKX2.5 (Q181X), in the presence of GATA4, revealed disrupted synergistic activation by the mutant protein. Experiments 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).

Downregulation of the following genes in ASD may influence the establishment of the heart's atrial septum, cardiomyocyte proliferation, and cardiac muscle development, according to a doctor: GATA4 and NKX2-5, extracellular signal molecules VEGFA and BMP10, cardiac sarcomeric proteins MYL2, MYL3, MYH7, TNNT1, and TNNT3, and cardiac sarcomeric proteins MYL2, MYL3, MYH7, TNNT1, and TNNT3, Dysregulation of these genes during heart septum morphogenesis, according to the researchers, may lead to cell cycle as the dominant pathway among downregulated genes, with the potential for decreased expression of cell cycle proteins disrupting cardiomyocyte growth and differentiation during atrial septum formation (5).

Types of ASD

The main five types of ASD as following: atrial septal defect (ASD) is a defect in the interatrial septum permitting free communication of blood between the atria. Based on the location of the septal defect ASD can be classified into four types. Ostium secundum ASD is the commonest type of ASD which involves the fossa ovalis, in the mid-septal region. 20% of these cases are associated with mitral valve prolapse (MVP). Ostium primum type of ASD is rare, defect is near AV valves. The AV valves may also be deformed. Sinus venosus type is also rare, defect is seen high in the atrial septum near the entry of superior venacava (SVC), and Coronary sinus type of ASD (figure 2) (6).

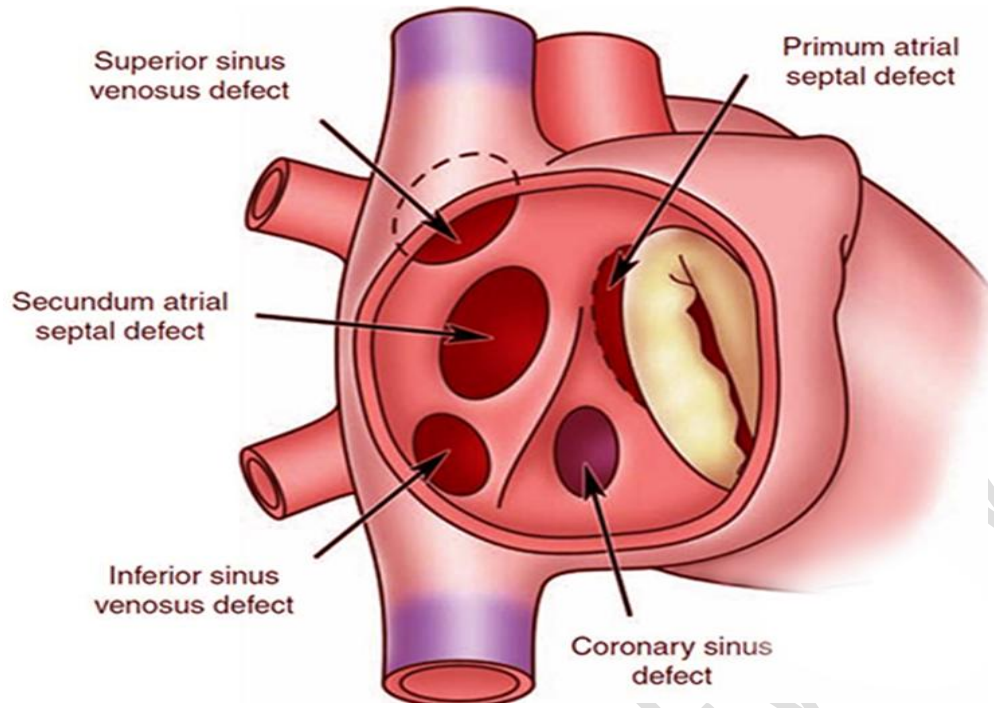

Figure 2 the five types of ASD (6)

Symptoms and Presentation

Many newborns with atrial septal abnormalities have no symptoms or indicators when they are born. Adulthood can bring on signs or symptoms. The following are some of the signs and symptoms of an atrial septal defect: Shortness of breath, especially when exercising, is a common complaint. Fatigue, Legs, foot, or abdomen swelling Heart palpitations, also known as skipped beats, are a type of heart palpitation that occurs when the heart Heart murmur (a whooshing sound that can be heard with a stethoscope) and stroke (7).

Due to minor physical examination results and a lack of symptoms, the atrial septal defect (ASD) abnormality can lie misdiagnosed for decades. In children, even isolated abnormalities of moderate to large magnitude may not elicit symptoms. Some people, however, may experience symptoms such as fatigue, recurrent respiratory infections, or exertional dyspnea. The diagnosis is frequently evaluated in children if a cardiac murmur is discovered during a routine physical examination or after an aberrant result on chest radiographs or an electrocardiogram (ECG). Symptoms might emerge gradually over decades if undiagnosed in childhood, and are mostly the result of altered compliance with age, pulmonary arterial hypertension, atrial arrhythmias, and, in certain cases, those linked with mitral valve disease in a primum ASD. Almost of ASD patients who live into their sixth decade are symptomatic (8).

Clinical decline in older people is caused by a number of factors, including the following: The left-to-right shunt is augmented by an age-related decrease in left ventricular compliance. Second, after the fourth decade, atrial arrhythmias, particularly atrial fibrillation, but also atrial flutter or paroxysmal atrial tachycardia, become more common and can lead to right ventricular failure. Third, most symptomatic persons over the age of 40 have mild-to-

moderate pulmonary arterial hypertension in the presence of a chronic significant left-to-right shunt, putting pressure and volume overload on the ageing right ventricle. Another cause of symptoms, especially those linked to primum ASD, is clinically severe mitral regurgitation. With age, the incidence, scope, and degree of dysfunction rises. Insufficiency of the mitral valve causes a rise in left atrial pressure and a greater degree of left-to-right shunt. Dyspnea, easy fatigability, palpitations, prolonged atrial arrhythmia, syncope, stroke, and/or heart failure are the most prevalent presenting symptoms. Palpitations associated with atrial arrhythmias are one of the most prevalent symptoms in adults (8).

Examination

The degree of left-to-right shunt and its hemodynamic consequences are determined by the size of the defect, the diastolic properties of both ventricles, and the relative resistance of the pulmonary and systemic circulations, 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 the following in mind: Due to excessive diastolic filling and big stroke volume, the patient may have a hyperdynamic right ventricular impulse. A dilated pulmonary artery can be recognised by palpable pulse of the pulmonary artery and an ejection click. The second component of S1 is usually divided, and the strength of the second component may be enhanced, indicating vigorous right ventricular contraction and delayed closure of the tricuspid leaflets. Because of lower respiratory variation due to delayed pulmonic valve closure, S2 is frequently widely split and fixed (seen only if pulmonary artery pressure is normal and pulmonary vascular resistance is low). This characteristic abnormality is found in almost all patients with large left-to-right shunts (9).

Because there is no significant pressure differential between the atria, blood flow over the ASD does not create a murmur at the site of the shunt. ASD with moderate-to-large left-to-right shunts, on the other hand, causes a crescendo-decrescendo systolic ejection murmur due to increased right ventricular stroke volume over the pulmonary outflow tract. This murmur can be detected in the upper left sternal border's second intercostal region. Because of increased flow across the tricuspid valve, patients with extensive left-to-right shunts frequently have a rumbling middiastolic murmur at the lower left sternal boundary. The ASD's auscultatory signs may resemble mild valvular or infundibular pulmonic stenosis and idiopathic pulmonary artery dilatation. These illnesses all have a systolic ejection murmur, but they differ from ASD in that the S2 moves with breathing, there is no pulmonary ejection click, and there is no tricuspid flow murmur (9).

An apical systolic regurgitant murmur of mitral regurgitation may be seen in patients with an ostium primum defect and an accompanying cleft of the mitral valve. A right ventricular S4 may be present in people who develop pulmonary arterial hypertension and right ventricular hypertrophy. The midsystolic pulmonic murmur is softer and shorter in these situations, the tricuspid flow murmur is absent, the splitting of S2 is narrower with an emphasised pulmonic component, and pulmonic regurgitation murmur may be audible. ASD is a cyanotic disorder. As a result, the patient should have a normal saturation level. Atrial shunt reversal

(Eisenmenger syndrome) can develop in the uncommon situation of severe pulmonary arterial hypertension, resulting in cyanosis and clubbing (10).

Complications

A little atrial septal defect may never cause any issues. During childhood, small atrial septal abnormalities commonly close. Larger flaws can lead to significant complications, such as right-sided heart failure, irregular heart rhythms (arrhythmias), an increased risk of stroke, and a shorter life span. Serious issues that are less prevalent include: Increased blood flow to the lungs increases the blood pressure in the lung arteries if a big atrial septal defect is left untreated (pulmonary hypertension). Pulmonary hypertension, also known as Eisenmenger syndrome, can result in lifelong lung damage. Eisenmenger syndrome is a long-term condition that affects only a small percentage of patients with significant atrial septal abnormalities. Many of these issues can be avoided or managed with treatment (11).

Pregnancy and atrial septal defect: Most women with an atrial septal defect are able to carry a child without experiencing any complications as a result of the defect. Complications during pregnancy can be increased by having a bigger defect or complications such as heart failure, arrhythmias, or pulmonary hypertension. Doctors strongly warn women with Eisenmenger syndrome not to get pregnant since it could put their lives in jeopardy. Children of parents with congenital heart disease have an increased risk of developing congenital heart disease. Anyone considering starting a family and has a congenital heart problem, whether fixed or not, should consult a doctor. Repair may be suggested by the doctor prior to conception (11).

Prevention

Atrioventricular septal abnormalities are almost always irreversible. Schedule an appointment with the patient's health care provider if the patient is planning to become pregnant. This visit should contain the following: Getting tested for rubella immunity: Inquire with the doctor about being vaccinated if the patient is not immune. Examining the patient's current health and medications: During pregnancy, the doctor will need to keep an eye on specific health issues. Before becoming pregnant, your doctor may advise you to alter or stop taking certain medications. Examining the medical history of the family: Consider speaking with a genetic counsellor if the patient has a family history of heart abnormalities or other genetic problems to identify the risk (12).

Investigations

Hearing a heart murmur during a checkup may lead a doctor to suspect an atrial septal defect or another cardiac abnormality in the child. The doctor may order one or more of the following tests if a heart defect is suspected (12).

Laboratory Studies

In the case of atrial septal abnormalities, no special laboratory blood testing are recommended (ASDs). Routine laboratory tests, such as complete blood cell (CBC) count, Type and screen, Metabolic profile or chemical panel, and coagulation investigations, should be conducted in

patients undergoing ASD intervention (prothrombin time [PT] and activated partial thromboplastin time [aPTT]) (13).

Echocardiogram

Transthoracic 2-dimensional (2-D) echocardiography clarifies an ambiguous diagnosis by providing direct noninvasive visualisation 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. The subcostal perspective is frequently the most effective. The diagnosis of a sinus venosus defect, for which transesophageal echocardiography (TEE) may be required to image the defect, but TEE may still be unable to view the pulmonary venous return, is an exception. Anomalies of systemic venous connection should be sought in any patient with an ASD, particularly those with a sinus venosus defect. Two-dimensional imaging can easily identify these. The presence of right atrial and right ventricular enlargement without a known reason should trigger a TEE. When it comes to demonstrating flow through the atrial septum, Doppler echocardiography can be useful. It features a biphasic rhythm (systolic and diastolic) with a slight right-to-left shunt at the start of systole. Real-time (RT) 3-dimensional (3D) Doppler TEE can also provide detailed and precise information on the suitable occluder device, as well as aid transcatheter occlusion by guiding the catheter through the sometimes difficult patient anatomy (figure 3) (14).

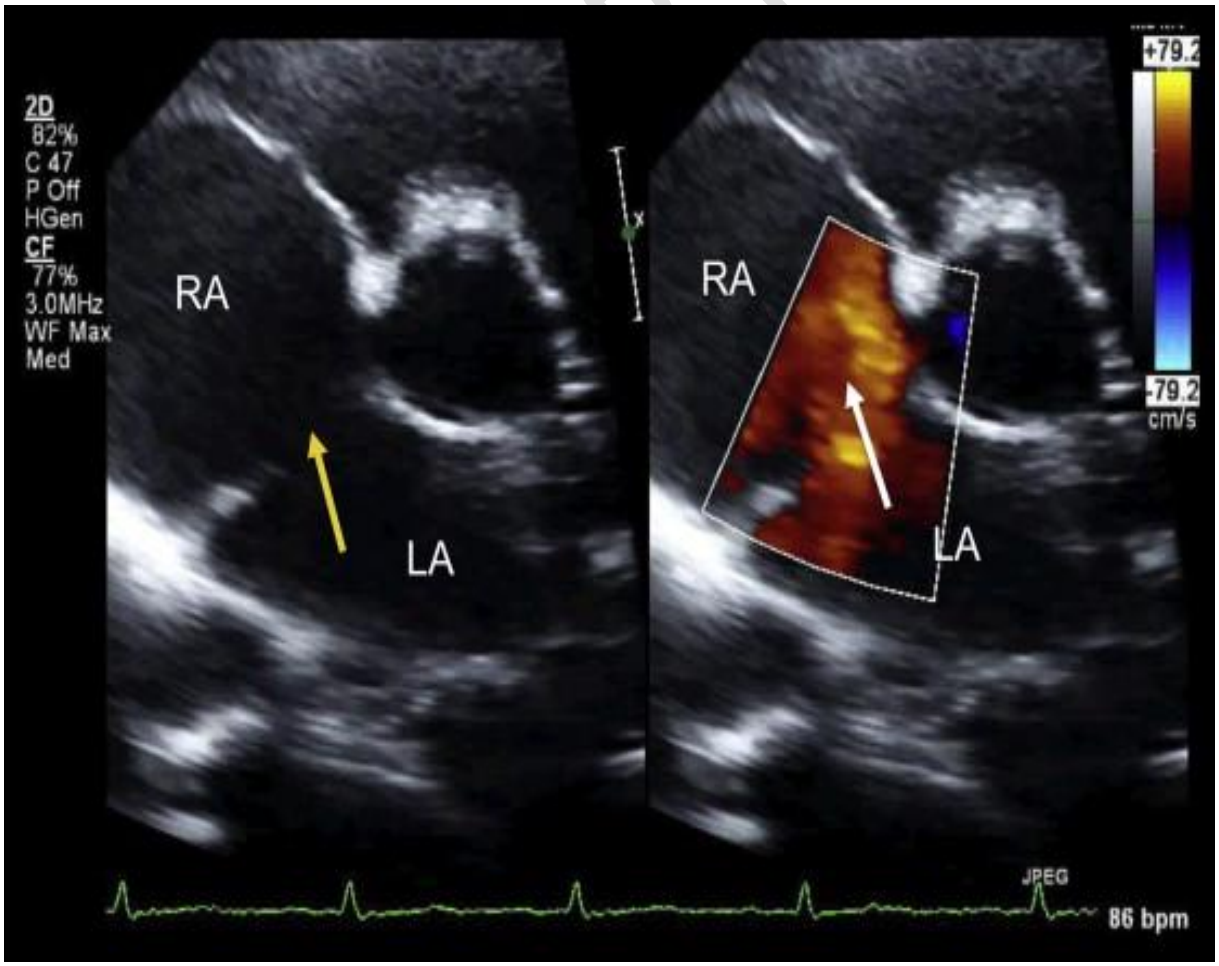

Figure 3 echocardiography shows Atrial Septal Defect (14)

In some patients with weak echocardiographic windows, transthoracic echocardiography (TTE) may be unsatisfactory. TEE can provide excellent delineation of the atrial septum in such cases. TEE can also be used to guide device placement during catheter-assisted ASD occlusion procedures and to provide immediate intraoperative confirmation that the defect has been closed. When a tricuspid regurgitant jet is present, continuous-wave Doppler echocardiography is useful for assessing right ventricular (and pulmonary arterial when there is no accompanying right ventricular outflow tract obstruction) systolic pressure. This method can also be used to assess patients for pulmonary venous return blockage. Contrast echocardiography can be used to confirm the diagnosis. Microcavitation bubbles in the left atrium and left ventricle can be used to diagnose a right-to-left shunt. A left-to-right shunt can be identified in the right atrium as a negative contrast washout effect (15).

Chest X-ray

Chest radiographs most typically show cardiomegaly in the context of a clinically substantial left-to-right shunt due to dilation of the right atrium and right ventricular chamber. In the lung fields, the pulmonary artery is conspicuous, and pulmonary vascular markings are enlarged. Only clinically severe mitral regurgitation causes left atrial enlargement. In the case of a sinus venosus defect, proximal dilatation of the superior vena cava might be detected (figure 4) (16).

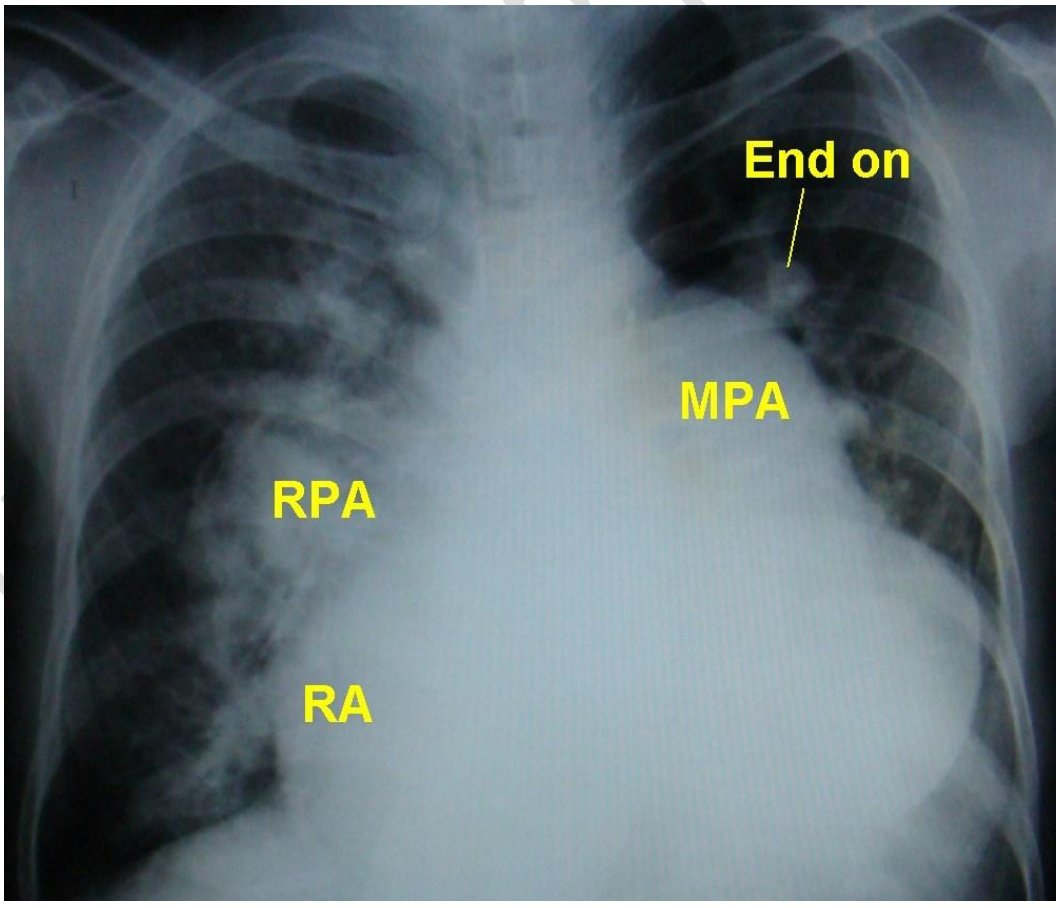

Figure 4 this chest X-ray in ASD shows grossly dilated main pulmonary artery (16)

Electrocardiogram (ECG)

A normal sinus rhythm, right-axis deviation, and a rSR' pattern in V1, as well as an interventricular conduction delay or right bundle branch block, are all common findings in patients with secundum atrial septal defect (ASD) (which represents delayed posterobasal activation of the ventricular septum and enlargement of the right ventricular outflow tract). An ostium primum defect is indicated by left-axis deviation and a rSR' pattern in V1, as well as an interventricular conduction delay or right bundle branch block. Sinus venosus defect is indicated by left-axis deviation and a negative P wave in lead III. Loss of the rSR' pattern in V1 and a tall monophasic R wave with a deeply inverted T wave can occur when pulmonary hypertension rises. Due to left atrial enlargement and a greater distance for internodal conduction caused by the defect itself, a prolonged P-R interval can be noted in familial ASD or ostium primum. The AV node has been found to be displaced in a posteroinferior orientation in some cases, as well as an enlarged right atrium (figure 5) (17).

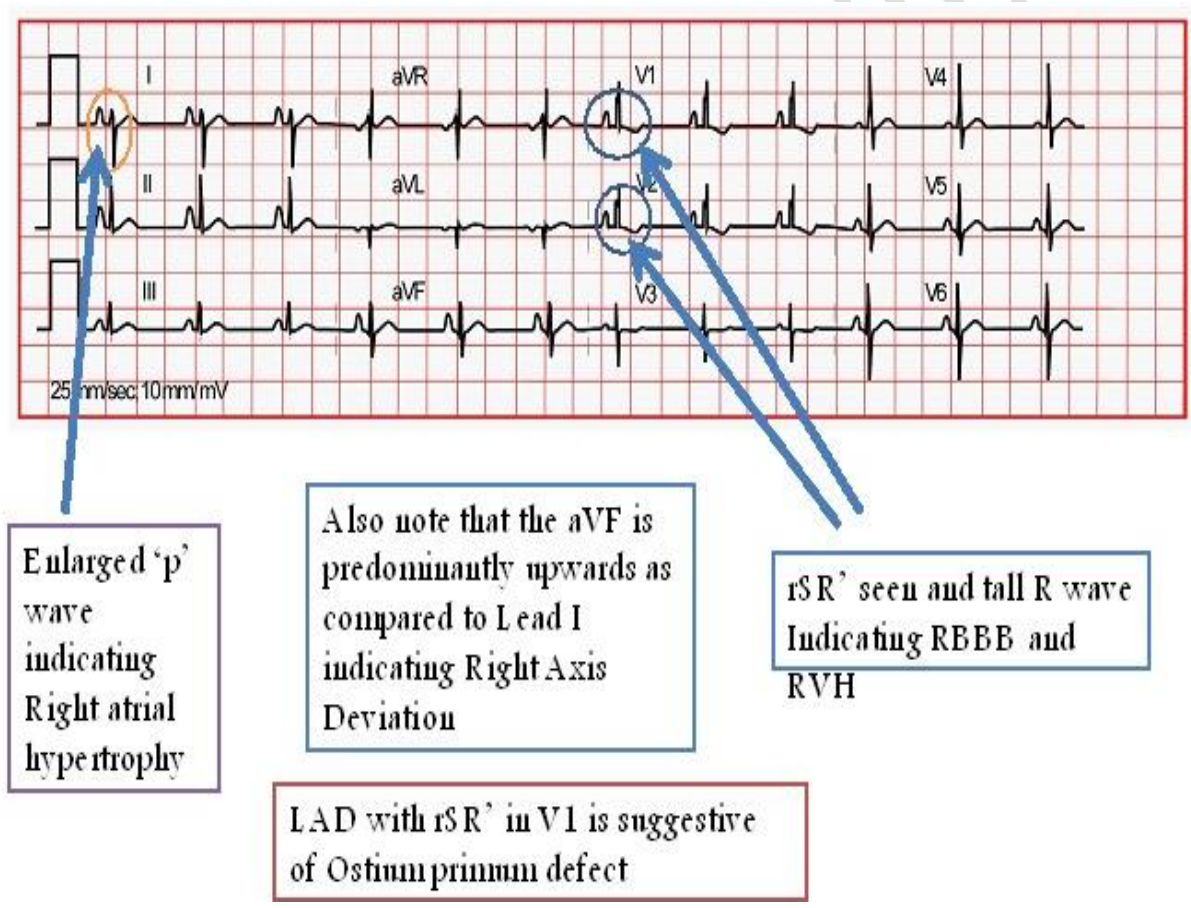

Figure 5 electrocardiogram of Atrial Septal Defect (17)

Cardiac catheterization

A catheter is a thin, flexible tube placed into a blood artery in the groyne or arm and guided to the heart. Doctors can diagnose congenital heart problems, assess how well the heart pumps, examine heart valve function, and measure blood pressure in the lungs using catheterization (figure 6) (18).

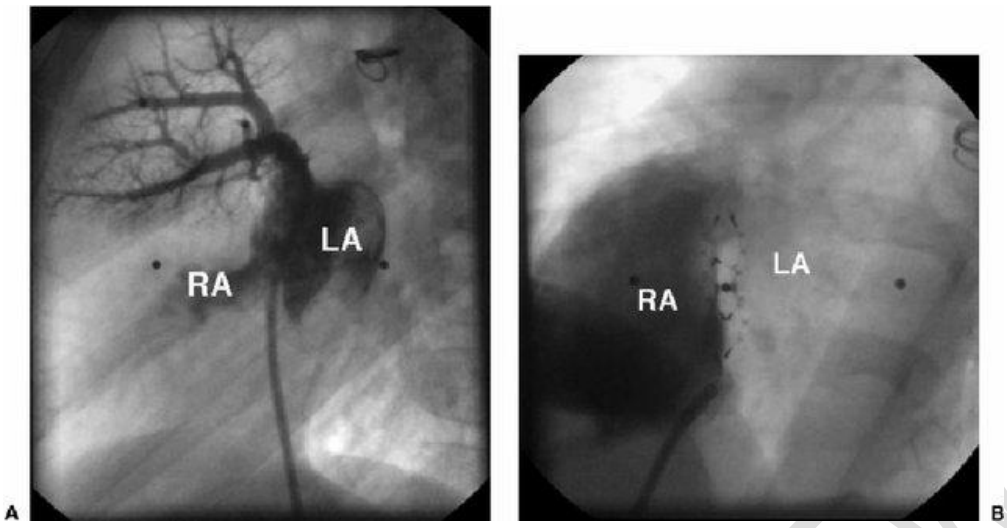

Figure 6 repair of an atrial septal defect. (A) A contrast shunting left-to-right through a moderate-sized atrial septal defect into the right atrium is shown on an angiography done in the left atrium (LA) (RA). The catheter is positioned across the LA defect. In addition, contrast is injected into the 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

The size and location of ASD have been successfully identified using MRI. Small flaws, on the other hand, have limited utility. The ability to quantify right ventricular size, volume, and function, as well as identify systemic and pulmonary venous return, is a major benefit of MRI (figure 7) (19).

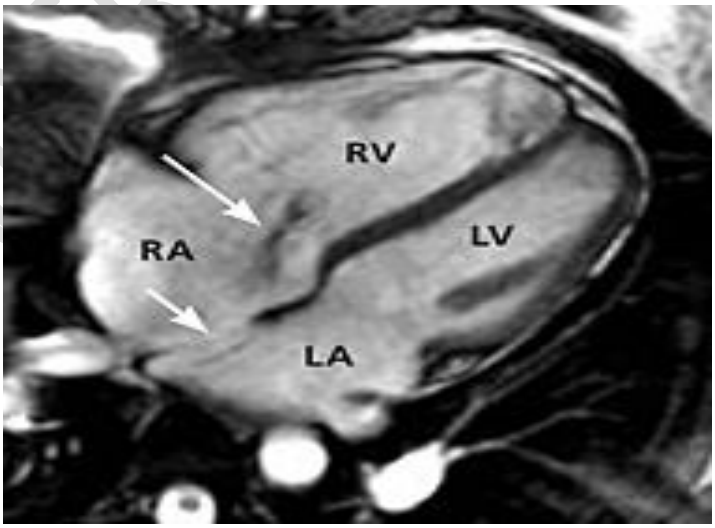

Figure 7 one of the atrial septal flaws (short arrow) is seen on magnetic resonance imaging, as well as enlargement of the right atrium and right ventricle. A systolic regurgitant flow jet (long arrow) can also be seen coming from the tricuspid valve in this image (19).

CT scan

This procedure creates detailed pictures of the heart using a succession of X-rays. If an atrial septal defect and accompanying congenital cardiac defects have not been definitively confirmed by echocardiography, it can be utilized to diagnose them (figure 8)(20).

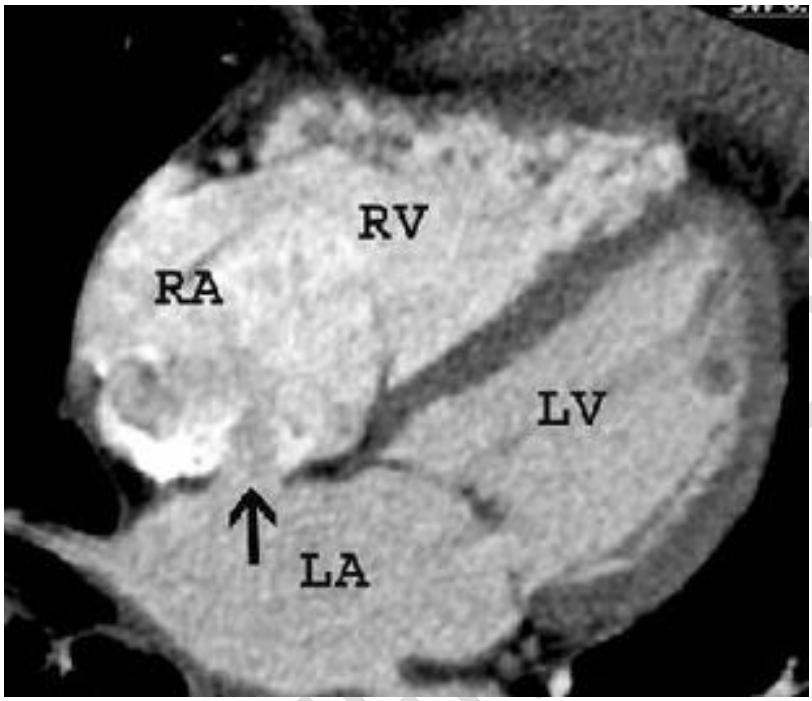

Figure 8 the contrast jet extends from the left atrium to the right atrium in this CT picture showing a defect in the fossa ovalis (arrow) (20).

Management

During childhood, many atrial septal abnormalities close on their own. Some tiny atrial septal abnormalities may not require treatment if they do not close. Many persistent atrial septal abnormalities, however, will eventually necessitate surgery. If the child has an atrial septal defect, the cardiologist may urge that it be monitored for a period of time to see if it closes on its own. Depending on the condition and whether the infant has other congenital heart problems, the doctor will determine when the child need therapy. Medications: Although medications will not close the hole, they may help to alleviate some of the signs and symptoms that come with an atrial septal defect. Drugs may also be used to lower the chances of problems following surgery. Medications to keep the heartbeat regular (beta blockers) or to lessen the danger of blood clots may be prescribed (anticoagulants) (21).

Surgery

To avoid future difficulties, many clinicians advocate fixing a medium to large atrial septal defect discovered in childhood or age. However, if the patient has significant pulmonary hypertension, surgery is not indicated because it may worsen the disease. The anomalous opening between the atria is sewn closed or patched in both adults and children. Doctors will assess the situation and recommend one of two procedures (22):

Minimally invasive approaches

Minimally invasive techniques to the treatment of ASD have sparked a lot of interest in recent years. In most cases, alternate ways to cardiopulmonary bypass simply reduce the size of the incision. Partial or full submammary skin incisions, hemisternotomy, and restricted thoracotomy are examples. Because these techniques are not linked to lower morbidity or death, the purpose is to improve cosmetic effects. In individuals with adverse anatomy or clinical contraindications, completely endoscopic minimally invasive surgery may be a viable option to catheter-based management for ASD. The outcomes of completely endoscopic closure using a glutaraldehyde-treated autologous pericardial patch in 37 Japanese patients with ASD who were postponed from transcatheter repair were favourable in a retrospective research (2011-2015). There were no operative deaths, post-procedure ASD reinterventions, or readmissions for heart failure, according to the researchers, and follow-up echocardiography revealed no recurring shunt or calcification of the autologous pericardial patch (figure 9) (23).

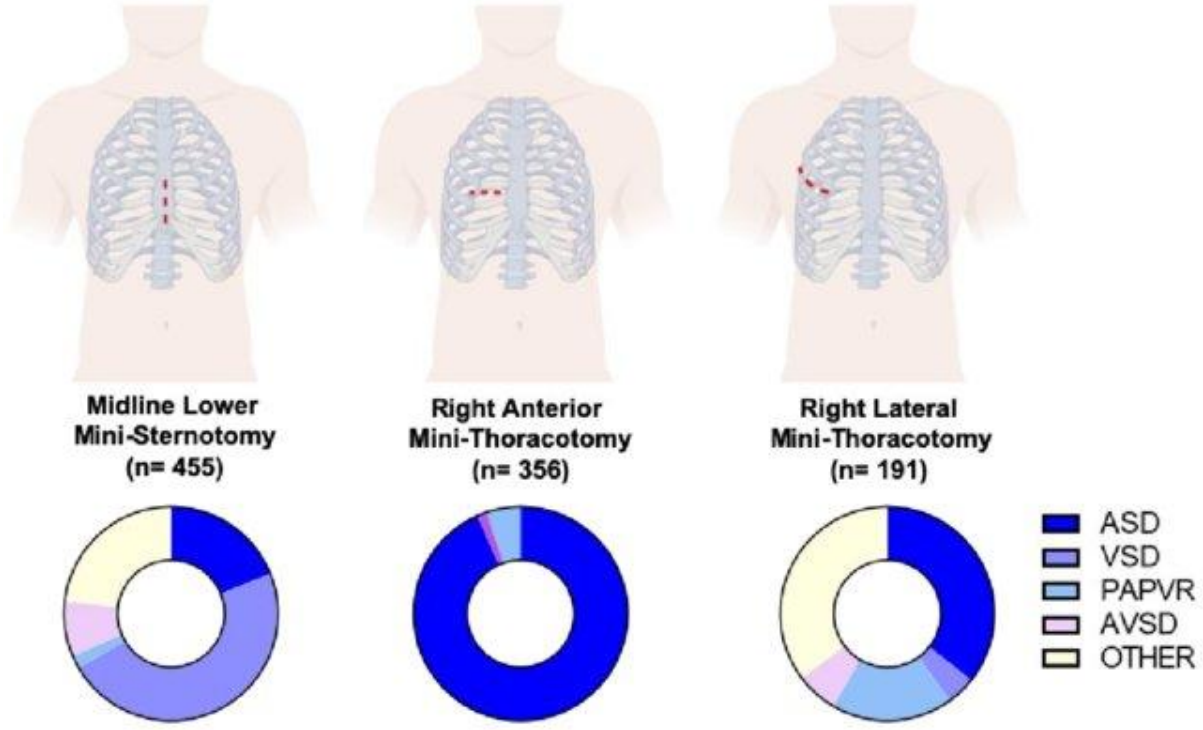

Figure 9 access for minimally invasive cardiac surgery and diagnoses. (Graphical representation of the three different type of access for minimally invasive cardiac surgery used. On the bottom, pie charts illustrate the distribution of diagnoses according to surgical access. ASD: Atrial Septal Defect; 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 devices are implanted

via a femoral venous route and are used to close the septal defect like an umbrella. These devices are most effective for secundum faults that are centrally positioned. Despite the fact that surgical closure has a low morbidity and mortality rate and great long-term results, sternotomy and cardiopulmonary bypass are required. In the mid-1970s, Drs. King and Mills conducted the first transcatheter closure of a secundum ASD. In the late 1970s, William Rashkind pioneered the development of a percutaneous ASD closure procedure. In 1989, Jim Lock invented the clamshell approach. Sideris began clinical testing with a buttoned device at the same time (24).

Despite the fact that various devices have been explored, four important devices have become accessible in the recent few years: CardioSEAL, Amplatzer septal occluder (ASO), HELEX septal occluder, and Sideris patch. Because it is simple to implant and allows closure of large orifices with great success rates in most situations, the ASO is now the most extensively utilised device. In 1995, it was utilised for the first time in humans. Because no randomised studies have been undertaken, choosing a specific device is problematic. Furthermore, percutaneous closure of ostium primum and sinus venosus defects is currently not possible with current devices (figure 10) (25).

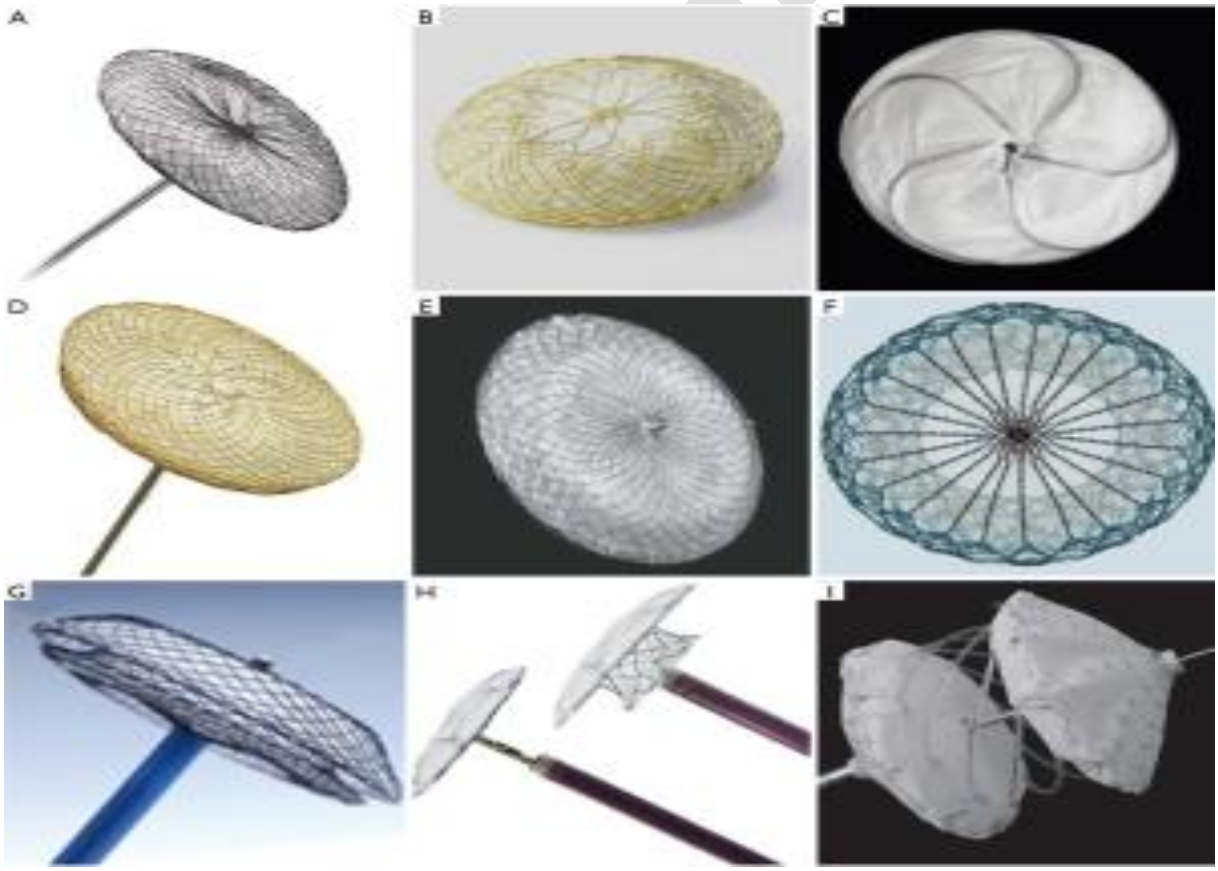

Figure 10 currently available devices for atrial septal defect closure. (A) Amplatzer Septal Occluder; (B) Occlutech Figulla Flex II device; (C) Gore Cardioform Septal Occluder; (D) Cocoon Septal Occluder; (E) CeraFlex ASD device; (F) Nit Occlud ASD-R device; (G) Cardi-O-Fix Septal Occluder; (H) Ultracept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

Transesophageal echocardiography is used to determine the static diameter of the defect in this procedure. The diameter is then measured with a size balloon and the "stopflow" approach is used to determine the device's proper diameter. The sizing balloon is inflated until no flow is evident through the defect utilising TEE in this procedure. The orifice's margins must be broad enough (5 mm) to accommodate the closing device's edges. TEE has long been the standard method for sizing, placing, and deployment of medical devices, although it can be uncomfortable. There is also the need for airway protection and general anaesthesia. The same thing has been done with intracardiac echocardiography. Most cardiac clinics currently use transcatheter closure of ASDs as standard therapy. It has been shown to be safe in expert hands, is cost-effective, and compares favourably to surgical closure, with more than 96 percent successful implantation rates. Transcatheter closure has been associated with fewer complications, shortened hospitalization, and reduced need for blood products (26).

Symptomatic improvement and regression of positive airway pressure (PAP) and right ventricle enlargement follow ASD repair at any age; however, individuals with less functional impairment and lower PAP have the greatest prognosis. Because symptoms, right ventricle remodelling, and PAP all increase with age, ASD closure should be advocated early after diagnosis, regardless of symptoms, especially in persons of advanced age. Furthermore, compared to surgery, transcatheter closure appears to have extra benefits in terms of hemodynamic improvement. The left atrial volume index, the left ventricular myocardial performance index, and the right ventricular myocardial performance index all improved after transcatheter closure with ASO, according to one study. After surgery, the last was disappointing, presumably because to cardiopulmonary bypass (27).

Another group used strain-rate imaging to compare atrial function in 45 individuals with an average age of 9 years after surgery and after percutaneous closure. They discovered that after transcatheter closure, both atrial functions were intact, but this was not the case after surgery. An atriotomy scar could have had a negative impact on right atrial function, whereas perioperative hypoxia or intraoperative myocardial injury could have changed the left atrium's deformation properties. Investigators observed improvements in New York Heart Association (NYHA) functional class, pulmonary artery pressure, and cardiac rhythm in a study of mid- to long-term follow-up findings of successful transcatheter ASD closures in 179 patients older than 40 years. The research lasted 8.8 years, with a median follow-up of 3.82.1 years (28).

Follow-up care is determined by the type of defect, the treatment recommended, and the presence of other defects. Echocardiograms are performed when the child is discharged from the hospital, one year later, and as needed by the child's doctor. Simple atrial septal abnormalities that were closed during childhood usually only require occasional follow-up therapy. Adults who have had an atrial septal defect repaired should be watched for consequences such as pulmonary hypertension, arrhythmias, heart failure, or valve abnormalities throughout their lives. Follow-up exams are usually done once a year (29).

Discussion

The three basic kinds of atrial septal defect (ASD) account for 10% of all congenital heart disease and 20-40% of congenital heart disease that manifests in adulthood. The following are

the most known kinds of ASD: Ostium secundum: The most prevalent kind of ASD, accounting for 75 percent of all ASD instances and accounting for about 7% of all congenital cardiac abnormalities and 30-40% of all congenital heart illness in those over the age of 40. Ostium primum: This is the second most frequent ASD, accounting for 15-20% of all ASDs. Atrioventricular septal defect, or primum ASD, is a type of atrioventricular septal defect that is frequently associated with mitral valve problems. Sinus venosus (SV) ASD is the least prevalent of the three, accounting for about 5-10% of all ASDs. The hole runs the length of the atrial septum on the superior side. An abnormal connection of the right-sided pulmonary veins is common and expected. Alternate imaging is generally required (30).

ASD is more common in women than in men, with a female-to-male ratio of about 2:1. Patients with ASD can be asymptomatic throughout childhood, albeit the onset of clinical symptoms is dependent on the degree of left-to-right shunt. As people get older, their symptoms become increasingly common. 90% of untreated people exhibit symptoms such as exertional dyspnea, tiredness, palpitation, persistent arrhythmia, or even signs of heart failure by the age of 40 (31).

Summary and Conclusion

Atrial septal defects are a type of congenital cardiac defect in which tissue at the interatrial septum is inadequate or absent. Right heart volume overload, atrial arrhythmia, and pulmonary arterial hypertension can all be symptoms of an unrepaired defect. The ostium secundum defect, ostium primum defect, and sinus venosus are the three most common kinds of atrial septal defect. A midsystolic pulmonary flow or ejection murmur, followed by a fixed split-second heart sound, are common physical findings. Small flaws may resolve on their own; bigger defects, on the other hand, may persist and cause hemodynamic and clinical complications that necessitate percutaneous or surgical intervention. Closure is not recommended if you have severe pulmonary arterial hypertension.

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