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PATHOPHYSIOLOGY, INVESTIGATIONS, AND MANAGEMENT OF VENTRICULAR SEPTAL DEFECT

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AUTHORS' CONTRIBUTIONS

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ABSTRACT

Background: Ventricular septal defects (VSDs) are still one of the most prevalent surgical indications in newborns and children with congenital heart disease. With advances in echocardiography, cardiac catheterization is no longer necessary in the treatment of these individuals. Although perioperative mortality and morbidity for isolated defects are still low, unique scenarios such as surgical care of numerous VSDs and decision-making in patients with pulmonary hypertension remain difficult. This chapter examines both classic and recent evidence that has shaped the management of this condition, as well as the facts underlying developing interventional methods utilized in both the catheterization lab and the operating room.

Conclusion: VSD is the most common congenital abnormality at birth. Small flaws should close on their own within the first year of life; however, larger faults can cause serious difficulties. The major interventions for big problems are surgical VSD closure and device closure.

Keywords: Ventricular septal defect; echocardiography; surgical closure; percutaneous treatment.

1. INTRODUCTION

A ventricular septal defect (VSD) is a hole or defect in the septum that separates the heart's two bottom chambers and allows communication between them. A VSD can occur as a single anomaly or in combination with other significant heart abnormalities. It can also be a single component of a number of intracardiac anomalies, such as tetralogy of Fallot (TOF), full atrioventricular (AV) canal defects, transposition of the major arteries, and repaired transpositions. The term ventricular septal defect is used in this article to denote an isolated VSD or a defect in a heart with AV concordance. That is, the atria are connected to the correct ventricle and the normally linked arteries (great arteries issuing from the appropriate ventricle [ie, a heart that is otherwise normal]), with no other serious defects. Isolated VSDs affect about 2-6 out of every 1000 live births, accounting for more than 20% of all congenital heart disorders. VSDs are the second most common congenital cardiac abnormality after bicuspid aortic valves [1].

Roger was the first to describe VSDs in clinical terms in 1879, and the name "Maladie de Roger" is still used

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to refer to a minor asymptomatic VSD. Eisenmenger documented a patient suffering from VSD, cyanosis, and pulmonary hypertension in 1898. The Eisenmenger complex has been named after this combination. The Eisenmenger syndrome is defined as pulmonary vascular disease and cyanosis in the presence of any other systemic-to pulmonary link. In 1958, Heath and Edwards defined the morphologic alterations associated with pulmonary vascular disease, and their six vascular change categories have remained the gold standard of comparison to this day. The size of the defect and the severity of the left-to-right shunt, which is dependent on the relative resistances of the systemic and pulmonary circulations, determine the symptoms and physical abnormalities associated with ventricular septal defects (VSDs) [2].

In the workup of a VSD, chest radiography, magnetic resonance imaging (MRI), and electrocardiography (ECG) may all be beneficial. While cardiac catheterization used to be a common aspect of the examination, thorough echocardiography is now the preferred diagnostic imaging technique. Small VSDs in children are asymptomatic and have a good long-term prognosis. Neither medical nor surgical treatment is recommended. Medical therapy is recommended to manage symptomatic congestive heart failure (CHF) in children with moderate or large VSDs since some VSDs may shrink over time, while uncontrolled CHF symptoms with growth failure are a reason for surgical repair. Neither the patient's age nor his or her stature is a barrier to surgery. The Heart Health Center, as well as Tetralogy of Fallot and Ventricular Septal Defect, have patient education tools [3].

2. PATHOPHYSIOLOGY

Because of the pressure difference between the ventricular chambers, the interventricular septum is an asymmetric curving structure. It is divided into five sections: membranous, muscular (also known as trabecular), infundibular, atrioventricular, and inlet. A VSD in the corresponding component occurs when one of the above components fails to develop or fuse during embryonic heart morphogenesis. VSDs have a variety of categories and nomenclature schemes due to their anatomical sites and histologic differences [4].

Shunt formation between the right and left ventricles are the primary pathophysiologic mechanism of VSD. The hemodynamic significance of the VSD is determined by the amount of blood shunted and the direction in which it is shunted. The size, location, and pulmonary vascular resistance all influence these characteristics. While VSDs are categorized by location, they can also be categorized by size. The diameter of the aortic annulus is used to characterize the size. They are classified as small if they are less than or equal to 25% of the aortic annulus diameter, medium if they are more than 25% but less than 75% of the annulus diameter, and large if they are larger than 75% of the annulus diameter. The pulmonary vascular endothelium develops irreversible alterations in the presence of long-standing significant left-to-right shunts, resulting in persistent PAH. When the pulmonary circulation pressure exceeds the systemic circulation pressure, the shunt direction reverses and becomes a right-to-left shunt. This is known as Eisenmenger syndrome, and it occurs in 10% to 15% of patients with VSD [4].

3. CLASSIFICATION OF VSD

1st type: (infundibular, outlet) This VSD is found in the right ventricle's outflow septum, above the crista supraventricularis, below the semilunar valves (aortic and pulmonary). It's also known as supracristal VSD. It is the least frequent form, accounting for only 6% of all VSDs, with the exception of the Asian population, where it accounts for roughly 30%. The lack of support of the right and/or noncoronary cusps of the aortic valve causes aortic valve prolapse and regurgitation. It's uncommon for these faults to close on their own. 2nd type: (membranous) this form of VSD is by far the most common, accounting for 80% of all defects. It's found beneath the crista supraventricularis in the membranous septum. When it's called perimembranous, it usually affects the muscular septum. The tricuspid valve's septal leaflet can sometimes develop a "pouch" that lowers the shunt and leads to spontaneous closure. 3rd type: (inlet or atrioventricular canal) Within the inlet region of the right ventricular septum, this VSD is positioned just inferior to the inlet valves (tricuspid and mitral). It barely accounts for 8% of all malformations. It is seen in Down syndrome patients. 4th type: (muscular, trabecular) This VSD is found in the muscular septum, usually in the apical, middle, and outlet sections of the interventricular septum, and is bordered by muscle. They can come in a variety of sizes and shapes, with a "Swiss cheese" appearance. They account for up to 20% of VSDs in babies. Adults, on the other hand, have a decreased incidence due to the tendency for spontaneous closure (Fig. 1) [5].

4. CAUSES AND RISK FACTORS

Clustering the types stated before according to putative pathogenic processes is beneficial for etiologic study. The following pathologic classification allows similar problems to be compared:

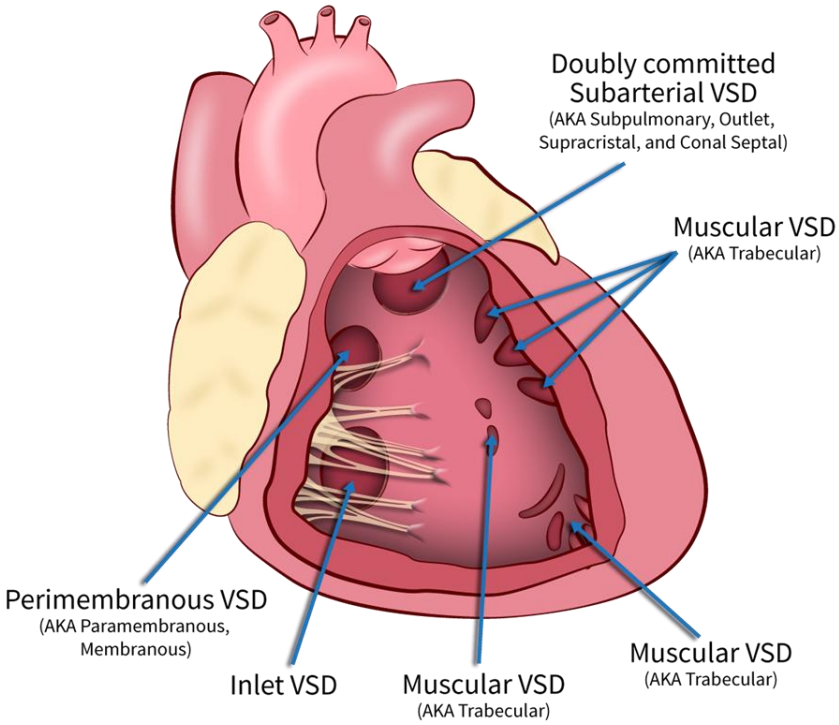

Fig. 1. Types of VSD [5]

Subarterial VSDs are abnormalities of ectomesenchymal tissue migration, pericardiac VSDs are abnormalities of intracardiac blood flow, muscular VSDs are abnormalities in cell death, and type III inflow VSDs are abnormalities of the extracellular matrix and faults in the endocardial cushion. The problems are currently thought to be caused by a complex etiology including an interaction between inherited predisposition and environmental effects [6].

Maternal factors: It has long been known that maternal diabetes is a risk factor for congenital cardiovascular malformations (CCVMs). For newborns of women with poorly controlled increased phenylalanine levels, the risk of CCVMs remains substantial. There are no population-based statistics on the range of risks that alcohol intake poses to the developing cardiovascular system. Only muscle ventricular septal defect was linked to maternal alcohol consumption, according to researchers from the Baltimore-Washington Infant Study (BWIS) [6].

The presence of a genetic risk factor, defined as a past occurrence of a congenital cardiovascular abnormality in the family, is the single greatest determinant in the BWIS data set. A major risk factor is a family history of a cardiac or noncardiac abnormality in either a parent or a previous sibling. VSD is three times more common in siblings of individuals with the same defect than in the general population. VSDs have been documented in identical twins, however, even in identical twins, discordance is common. By phenotype and developmental mechanism, familial congenital heart abnormalities are frequently concordant. Previous transposition, tetralogy of Fallot (TOF), and truncus arteriosus are all more common than predicted in VSD cases [7].

Genotype-phenotype correlation: The next generation of pediatric cardiologists will face difficulty in collaborating with geneticists to define genotype-phenotype correlations. In terms of genetic counseling and prevention, the single most significant shift in CCVM recurrence risk counseling is the detection of familial and chromosomally based abnormalities (see Table below). The following items are included in a thorough evaluation: A clinical diagnostic of the cardiovascular defect(s) that is correct and arranged in a hierarchy (this is necessary to specify the type of VSD) Noncardiac abnormalities are meticulously noted. A rigorous examination of pregnancy loss, racial origin, and consanguinity, as well as a search for risk factors such as gestational diabetes mellitus, is performed on first- and second-degree relatives [7].

5. SIGNS AND SYMPTOMS

The first few days, weeks, or months of a child's existence are commonly marked by signs and symptoms of significant cardiac abnormalities. Poor eating, failure to thrive, fast breathing or dyspnea, and easy fatigue are all indications of a ventricular septal defect (VSD) in a baby. A ventricular septal defect may go undetected by you and your doctor at birth. Symptoms may not develop until later in childhood, if at all if the abnormality is minor. The signs and symptoms vary depending on the size of the hole and any other cardiac abnormalities that may be present. If your doctor hears a murmur while listening to your baby's heart with a stethoscope during a routine visit, he or she may suspect a heart abnormality. Ultrasound can sometimes detect a VSD before the baby is born. A VSD may not be discovered until a person reaches maturity. Symptoms and signs can include shortness of breath or a heart murmur your doctor hears when listening to your heart with a stethoscope [8].

6. COMPLICATIONS

The most serious complication of a big VSD is the Eisenmenger complex. The left-to-right shunt is reversed to a right-to-left shunt as a result of fixed and irreversible pulmonary hypertension. Aortic valve leaflet prolapse is linked to secondary aortic insufficiency. It is uncommon in children under the age of two. Only about 5% of people with VSD experience this problem. Supracristal VSDs have a higher incidence than perimembranous VSDs. The occurrence of aortic regurgitation in the presence of doubly committed subarterial VSD is well-known. Aortic regurgitation is caused by a cusp prolapse caused by a weakly supported right coronary cusp mixed with the Venturi effect created by the VSD jet. In a large cohort of VSD patients in France, RV outflow tract blockage was found in 7% of the cases. The occlusion was infundibular, according to the investigators. A later angiocardiographic study showed that the obstruction was most often secondary to anomalous muscle bundles and only rarely infundibular [9].

VSD is sometimes linked with discrete fibrous subaortic stenosis. Perimembranous VSDs are the most common cause of this problem, which can occur following either spontaneous or surgical closure. Zielinsky et colleagues determined that all patients with a VSD who develop distinct subaortic stenosis had anterior or posterior malalignment of the outlet or conal septum. Infectious endocarditis is uncommon in children under the age of two. In the presence of infective endocarditis in the pulmonary circulation, it is critical to properly record the patient's history and use echocardiography to analyze the left-to-right shunt. VSDs can influence both the systemic and pulmonary circulation, resulting in vegetation on both sides [9].

Regardless of the vegetation's morphology, embolization is expected. Even if there are no symptoms, vegetation larger than 10 mm, especially if pedunculated, should be considered a cause for surgical surgery. The infection is generally found on the tricuspid or pulmonary valve leaflets, or on the ridge of the VSD itself. Following biventricular correction of conotruncal abnormalities, intramural VSD, in which interventricular contacts occur through right ventricular free wall trabeculations, may arise. These VSDs are associated with higher surgical morbidity, death, and longer hospital stays [9].

7. PREVENTION

In most circumstances, there is little you can do to avoid having a child with a ventricular septal defect. However, it is critical to take every precaution to ensure a healthy pregnancy. Here are the fundamentals: Get prenatal treatment as soon as you find out you're expecting. Before you get pregnant, talk to your doctor about your health and any lifestyle adjustments that your doctor may suggest for a safe pregnancy. Also, make sure to discuss any medications you're taking with your doctor. Consume a well-balanced diet. Take a folic acid-containing vitamin supplement. Exercise on a regular basis. Consult your doctor to come up with an exercise regimen that is good for you. Risks should be avoided. Harmful substances such as alcohol, cigarettes, and illegal drugs are among them. Infections should be avoided. Before you get pregnant, make sure you're up to date on all of your vaccines. A developing fetus can be harmed by certain types of illnesses. Maintain diabetes control. If you have diabetes, check with your doctor to make sure it's under control before you start trying to conceive. Consider consulting a genetic counselor before becoming pregnant if you have a family history of heart abnormalities or other genetic diseases [10].

8. INVESTIGATIONS

Chest radiography, magnetic resonance imaging (MRI), and electrocardiography (ECG) can all help with a ventricular septal defect diagnosis (VSD). Although cardiac catheterization used to be a common aspect of a VSD evaluation, thorough echocardiography is now the preferred method. The information needed for surgical closure is provided by echocardiography. Cardiac catheterization is commonly performed in the following two situations:

Unknown response pulmonary hypertension A small-to-moderate defect with only slight left ventricular (LV) enlargement; cardiac catheterization is important in this context for definitively evaluating the pulmonary-to-systemic flow ratio (Qp: Qs), which can help with decision-making about the necessity for surgery (though MRI can provide this information noninvasively) [11].

With a sensitivity of 96 percent and a specificity of 95 percent, an experienced pediatric cardiologist can accurately diagnose newly-referred kids with murmurs on clinical examination. Cardiac biomarkers could be useful in assessing the clinical status of children with congenital heart disease and congestive heart failure. Troponin I and amino-terminal procollagen type III peptide (PIIIP) levels are higher in children with ASDs and VSDs, according to Sugimoto et al. Patients with pulmonary stenosis and tetralogy of Fallot have higher PIIIP levels. Furthermore, B-type natriuretic peptide (BNP)/N-terminal proBNP levels were linked to pediatric heart failure scores [11].

9. ECHOCARDIOGRAPHY

Because of its high sensitivity, color Doppler transthoracic echocardiography (TTE) is the most useful tool for diagnosis. Color Doppler TTE can detect up to 95% of VSDs, especially non-apical lesions bigger than 5 mm, and can offer morphologic information like size, position, and the number of defects, as well as hemodynamic information like jet size, severity, and pulmonary artery pressure assessment. TTE is helpful in diagnosing aortic insufficiency and other congenital cardiac abnormalities that may be present. Finally, TTE can be used to assess the size and function of the right and left ventricular chambers. Operator reliance and a poor acoustic window are two drawbacks. A trans-esophageal echo (TEE) is advised when conventional TTE is ambiguous (Fig. 2) [12].

10. ELECTROCARDIOGRAPHY

In 50% of VSD patients, electrocardiography (ECG) is completely normal. In people with big shunts, an aberrant ECG may identify LV hypertrophy. The ECG may demonstrate right bundle branch block, right axis deviation, and right ventricular (RV) hypertrophy and strain in patients with PAH (Fig. 3) [13].

11. CHEST X-RAY

In people with minor problems, chest radiography (CXR) is frequently normal. In patients with greater defects and larger LVs, the cardiac profile can be seen to be enlarged. Patients with PAH have an enlarged RV and a larger pulmonary diameter (Fig. 4) [14].

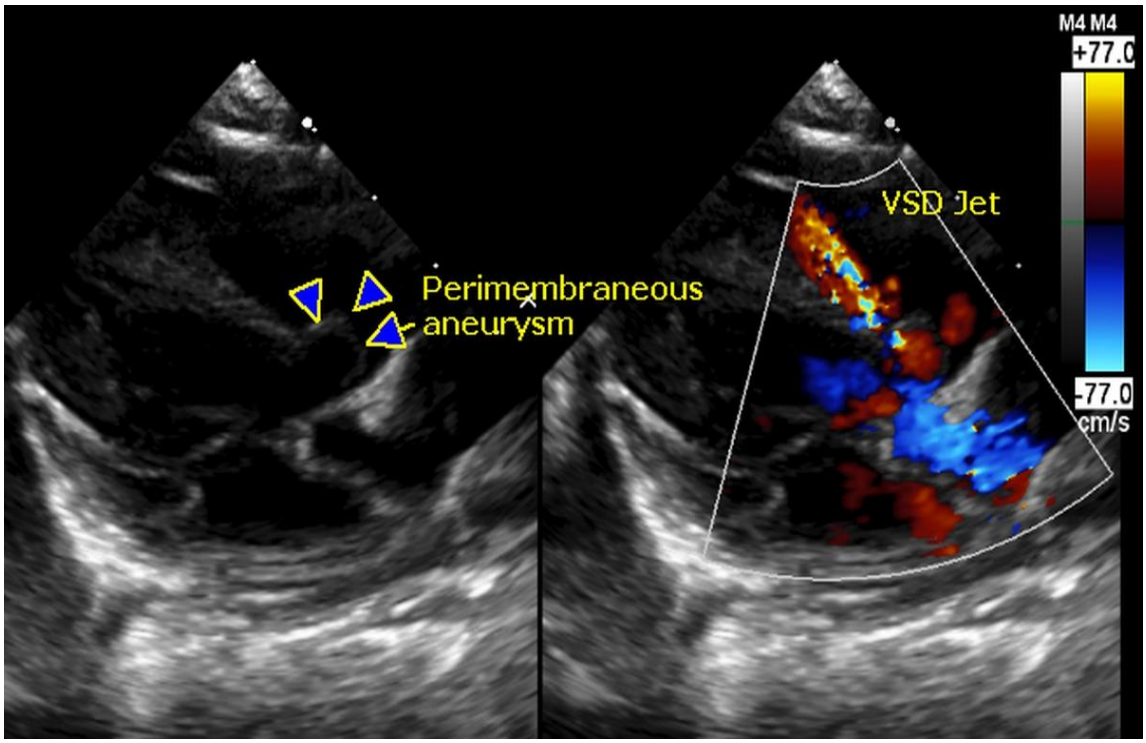

Fig. 2. Echocardiogram shows ventricular septal defect [12]

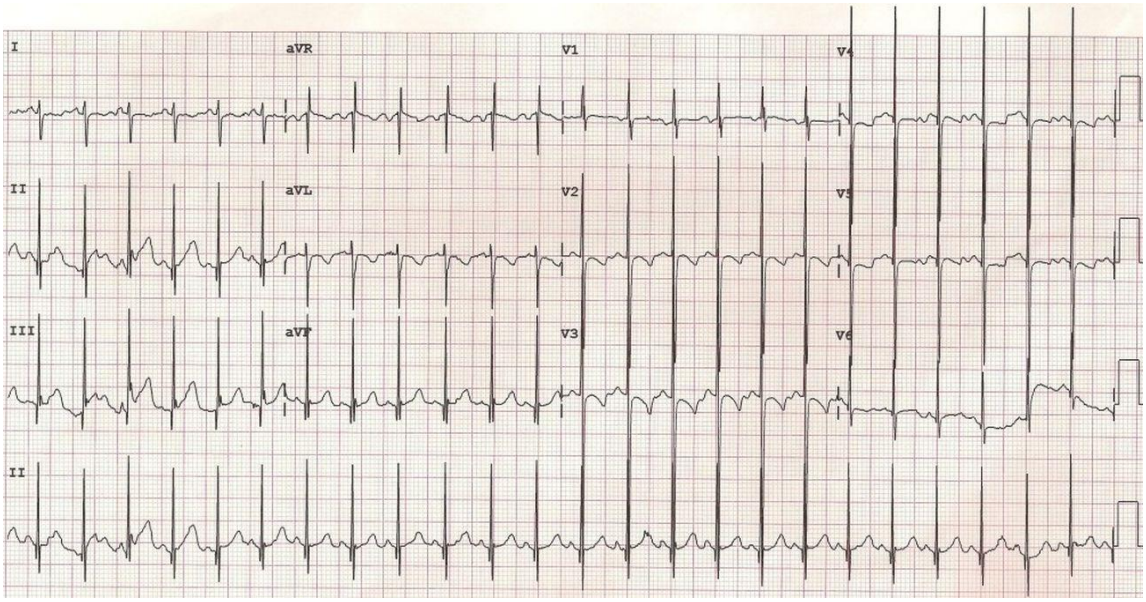

Fig. 3. In a VSD with a substantial left-to-right shunt, the Katz-Wachtel phenomena occurs. In a VSD with a significant left to right shunt, tall biphasic QRS complexes in the mid precordial leads suggest the Katz-Wachtel phenomenon [13]

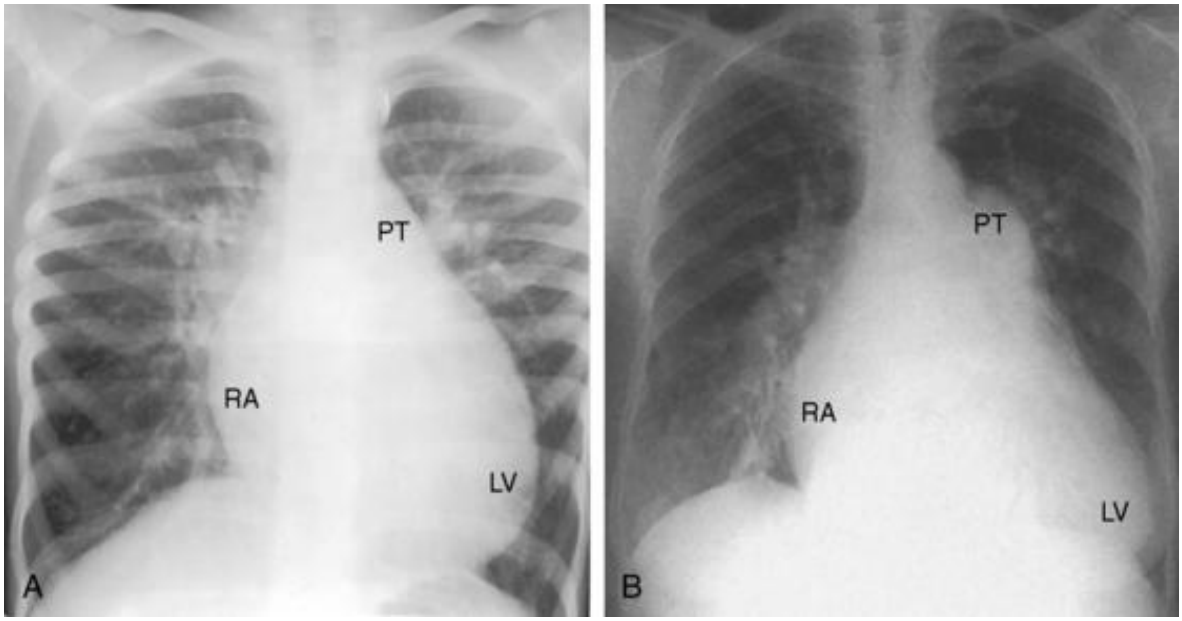

Fig. 4. X-rays of two patients with perimembranous ventricular septal defects, 60 years apart in age. A, A 5-year-old kid with a moderately restrictive ventricular septal defect, a 2.6-to-1 left-to-right shunt, and a pulmonary artery pressure of 43/13 mm Hg. An enlarged left ventricle (LV) occupies the apex, and a prominent right atrium occupies the right lower cardiac border. Pulmonary arterial vascularity is increased, the pulmonary trunk (PT) is moderately dilated, an enlarged left ventricle (LV) occupies the apex, and a prominent right atrium occupies the right lower cardiac border. B, A moderately restrictive perimembranous ventricular septal defect, a 2.7 to 1 left-to-right shunt, and a pulmonary artery pressure of 55/32 mm Hg were present in the 65-year-old woman. An enlarged left ventricle (LV) occupies the apex, and a prominent right atrium (RA) occupies the lower right cardiac border. Pulmonary arterial vascularity is increased, the pulmonary trunk (PT) is markedly dilated, an enlarged left ventricle (LV) occupies the apex, and an enlarged left ventricle (LV) occupies the apex [14]

12. MAGNETIC RESONANCE IMAGING

Cardiac MRI is effective in cases when the anatomy is complicated, such as when a VSD is present together with other congenital cardiac malformations, or when defects are present in atypical locations that are difficult to see with standard TTE (Fig. 5) [15].

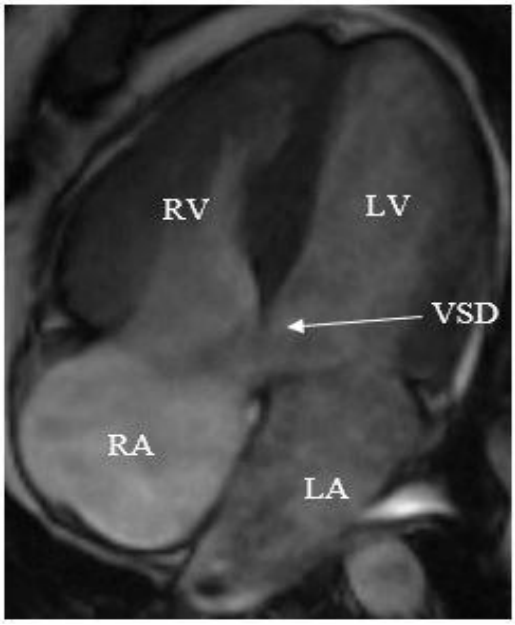

Fig. 5. An unrepaired massive membranous VSD with inlet extension caused Eisenmenger syndrome in a 28-year-old male [16]

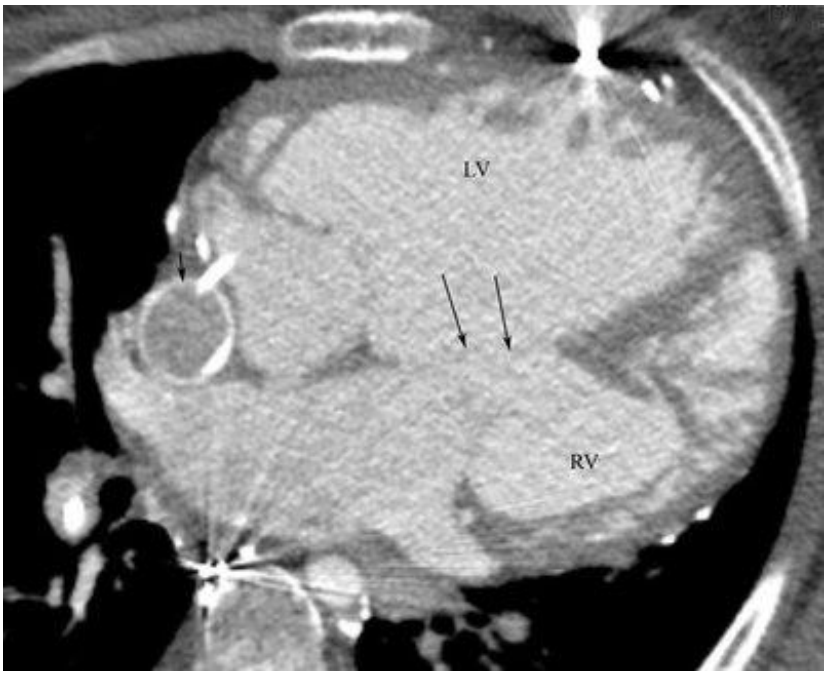

Fig. 6. VSD affects a 21-year-old lady. In a patient with levo-transposition of the great arteries and substantial dilatation of the morphologic left ventricle (LV), which gives birth to the pulmonary artery, an axial contrast CT picture reveals a massive membranous VSD (long arrows). After the Fontan shunt revision failed, a PTFE graft (short arrow) was inserted lateral to the right atrium (RA) [18]

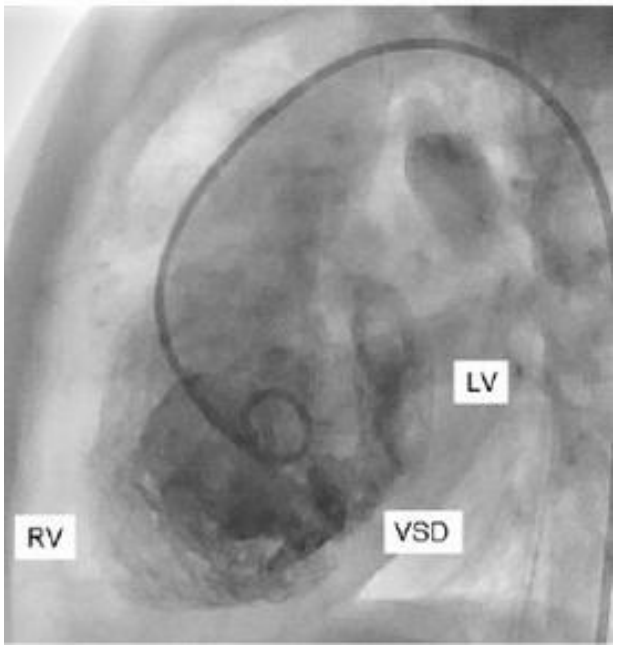

Fig. 7. Cardiac catheterization demonstrating VSD [20]

13. CT

The defect can be seen directly on a CTA with ECG-gating. Non-gated studies may reveal large VSDs (Fig. 6) [17].

14. CARDIAC CATHETERIZATION

Cardiac catheterization provides reliable hemodynamic information on pulmonary vascular resistance and reaction to vasodilators, which is very useful in patients undergoing surgical closure evaluation. It goes into greater detail about concomitant aortic regurgitation, multiple VSDs, and the possibility of coronary artery disease (Fig. 7) [19].

15. MANAGEMENT

During the first year of life, around 85 percent to 90 percent of small isolated VSDs close spontaneously. Patients with minor, asymptomatic VSDs who do not have PAH have a good prognosis without treatment. Otherwise, VSD closure is part of the management strategy. Due to the complexity of such instances, patients with Eisenmenger syndrome are frequently treated in advanced centers. VSDs were formerly only repaired surgically; however, recent improvements in interventional procedures have made percutaneous VSD closure conceivable. Antibiotic prophylaxis for infective endocarditis is no longer regularly provided for patients with unrepaired ventricular septal defects [21].

Endocarditis prophylaxis is recommended for people who have cyanotic congenital heart disease, have had previous episodes of endocarditis, and have prosthetic heart valves, or have had a prosthetic material repair. In general, VSD closure is recommended for medium to large defects with severe hemodynamic compromise, such as those with LV failure that are symptomatic. In cases of progressive aortic insufficiency or after an episode of endocarditis, an intervention should be considered [21].

According to the ACC/AHA 2008 recommendations, the following are the indications for surgical closure: Those who have experienced endocarditis. When the pulmonary to systemic blood flow ratio (Qp/Qs) is equal to or greater than 2 and there is clinical evidence of LV fluid overload. When there is evidence of LV systolic or diastolic dysfunction, or when the pulmonary artery pressure and pulmonary vascular resistance are less than two-thirds of systemic pressure and systemic vascular resistance, respectively, it is reasonable to intervene in milder shunts such as those with Qp/Qs above 1.5 [21].

Surgical repair lowers the risk of endocarditis, improves PAH, and boosts overall survival. In the absence of PAH, the operational mortality rate is around 1%. Residual or recurrent VSD, valvular incompetence such as tricuspid regurgitation and aortic insufficiency, arrhythmias, LV dysfunction, and PAH development are all potential complications. Atrial fibrillation, total heart block, and ventricular tachycardia are some of the arrhythmias that might

occur after VSD correction. The existence of irreversible PAH is the principal contraindication for surgical VSD closure, due to the significant surgical perioperative mortality and pulmonary comorbidities [22].

Device for use on the skin VSD closure is reserved for patients with severe PAH, many comorbidities, and those who have had previous cardiothoracic surgery with residual or recurring VSD. The main type of VSD responsive to this method is muscular VSDs; nevertheless, the proximity of other abnormalities to the inlet valves makes this technique difficult to conduct in such circumstances. Despite its unpopularity in the United States, current data reveal great results, including full closure and low mortality. The complete atrioventricular block is the most common problem, which is usually caused by perimembranous abnormalities (Fig. 8) [23].

16. DISCUSSION

The natural history of a VSD ranges from spontaneous closure to congestive heart failure (CHF) or the development of pulmonary vascular disease without heart failure symptoms and is directly proportionate to the size of the defect. Spontaneous closure happens regularly in toddlers, usually by the age of two. After the age of four, closure is unusual. Muscle defects are the most common cause of closure (80%), followed by perimembranous defects (35-40 percent). Outlet VSDs are less likely to close spontaneously, but inlet VSDs do not. Hypertrophy of the septum, growth of fibrous tissue, subaortic tags, apposition of the tricuspid valve's septal leaflet, or (in rare cases) protrusion of an aortic valve leaflet can all lead to closure. An aneurysm of the interventricular septum can develop when perimembranous VSDs close due to the growth of fibrous tissue or the apposition of the tricuspid valve [24].

A good prognosis is associated with a tiny VSD that does not close spontaneously. Although patients are at risk for infective endocarditis, tiny muscular VSDs do not offer any other risks. Small perimembranous VSDs, on the other hand, are linked to an increased risk of aortic cusp prolapse over time. In addition, the Second Natural History Study found a tiny but significant incidence of malignant ventricular arrhythmia. A total of 1000 patients were included in this investigation (about 76 percent of the original cohort). Between 1958 and 1969, the First Natural History Study included 1280 patients (mainly youngsters) with VSDs who were hospitalized after cardiac catheterization. During a 20-year follow-up of roughly 900 patients with perimembranous VSDs, Wu et colleagues found a 45 percent incidence of LV-to-RA shunts and a 6% incidence of subaortic ridges. This group later reported an increased incidence of infective endocarditis in patients who had LV-to-RA shunts [24].

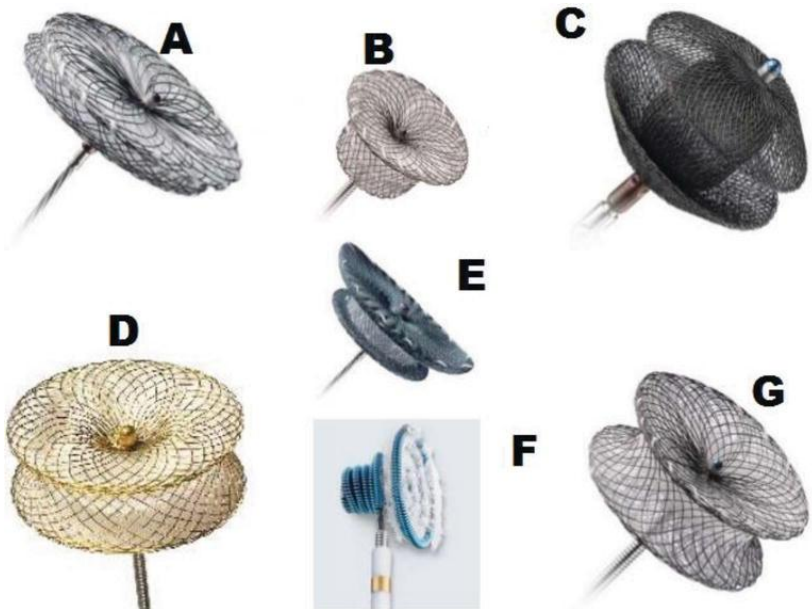

Fig. 8. (A) Amplatzer membranous VSD device, (B and C) Amplatzer Duct Occluder and Amplatzer Duct Occluder II devices. (D and G) Amplatzer and Occlutech muscular VSD device. (F) Nit-Occlud (Nickel-Titanium Spiral Coil) and (E) asymmetric membranous VSD device were used for transcatheter occlusion of VSDs [23]

VSDs afflicts 2-7 percent of live births in the United States, according to data. The prevalence of known VSDs may be influenced by the patient's geographic location. Small muscle VSDs, for example, are more likely to be discovered in urban areas, probably due to the availability of sophisticated healthcare in these areas. A high incidence of 5-50 VSDs per 1000 infants was discovered in an echocardiographic investigation. Small restrictive muscular VSDs were the defects in this investigation, which usually close spontaneously in the first year of life. Many chromosomal abnormalities, including trisomy 13, trisomy 18, trisomy 21, and very rare syndromes, have VSDs as the most prevalent lesion. VSDs, on the other hand, are not connected with a chromosomal aberration in moreover 95 percent of individuals. VSDs are slightly more common in female patients than in male patients, according to sex-related demographics (56 percent vs 44 percent). The incidence of abnormalities of ectomesenchymal tissue migration (ie, subarterial outlet VSD) is highest in boys [25].

Race-related demographics: Reports on racial differences in VSD distribution are ambiguous. The doubly committed or outlet flaw, on the other hand, is most common in Asians. In the United States, these faults account for 5% of all defects, but they account for 30% of all defects recorded in Japan. Small VSDs in children are asymptomatic and have a good long-term prognosis. Medical therapy for children with moderate or large VSDs has a variety of outcomes, as seen below. Between the ages of 6 and 24 months, many newborns improve, with evidence of a steady decrease in the extent of the left-to-right shunt. The cause of the decrease in left-to-right flow must be determined, as it could be due to an increase in pulmonary vascular resistance (PVR), a reduction in the relative size of the defect, or the development of RV outflow tract hypertrophy, all of which can result in functional or anatomic obstruction [25].

After infancy, the majority of infants with VSDs stay stable or improve. After childhood, heart failure is uncommon. A recurrence of symptoms might be caused by anemia, respiratory infection, endocarditis, or the development of an accompanying lesion (e.g., aortic insufficiency). Symptomatic therapy is required for a few patients who acquire severe pulmonary vascular obstructive disease with prominent right-to-left shunts (Eisenmenger syndrome) at the time of referral. Cyanosis worsens over time, and exercise ability declines. Lung or heart-lung transplantation may be an option for some patients with significant VSDs. Reduced red blood cell (RBC) counts with partial-exchange transfusion may help to alleviate symptoms of extreme polycythemia (eg, headache, extreme fatigue). Isolated VSDs have a surgical death rate of less than 1% [25].

17. SUMMARY AND CONCLUSION

A hole in the heart, known as a ventricular septal defect (VSD), is a common birth abnormality (congenital). The hole (defect) is in the septum, which separates the heart's lower chambers (ventricles) and permits blood to flow from the left to the right side. The oxygen-rich blood is then pumped back to the lungs rather than out to the body, putting more strain on the heart. A tiny ventricular septal defect may not cause any issues, and many VSDs close on their own. VSDs that are medium or bigger in size may require surgical treatment early in life to avoid problems.

VSD is best managed by a multidisciplinary team consisting of a pediatrician, cardiologist, heart surgeon, ICU nurse, physical therapist, and social worker. Parents and patients must be informed on the importance of following up. Aortic valve prolapse may develop in certain children with perimembranous VSD, necessitating surgery. Finally, all untreated VSDs have the potential to cause Eisenmenger syndrome by increasing pulmonary vascular resistance. Other than a heart and lung transplant, there is no other effective treatment option at this time. The majority of these patients die of increasing right heart failure and cyanosis due to a scarcity of organs for transplantation.

Young children with a minor VSD and no symptoms have a fair prognosis. Anemia, infection, or endocarditis, on the other hand, may cause symptoms in these youngsters. If a big VSD is not corrected, the patient's outcomes are dismal. Continuing the left-to-right shunt results in the development of pulmonary hypertension and the Eisenmenger syndrome. Most infants in North America today have their VSD corrected voluntarily within the first two years of their lives. The mortality rate is less than 1%, and the majority of patients live normal life.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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