Ventricular Septal Defect

Ventricular septal defect is an abnormal communication between the right and left ventricles. It is the most common type of acyanotic congenital heart defect that accounts for 25% of all the congenital heart defects.

It ranges from small to large, single to multiple to membraneous or fibrous.

Normally, the left side of the heart only pumps blood to the body, and the heart's right side only pumps blood to the lungs.

In a child with ventricular septad defect, blood can travel across the hole from the left pumping chamber (left ventricle) to the right pumping chamber (right ventricle) and out into the lung arteries.

If the VSD is large, the extra blood being pumped into the lung arteries makes the heart and lungs work harder and the lungs can become congested.

Classification of ventricular septal defect

Anatomic classification

1. Perimembraneous (Subaortic, infracristal) accounts for 75% of all VSDs
2. Muscular (anteriour, midmuscular or apical) accounts for 5-20% of all VSDs
3. Inlet (Inflow, canal VSD) accounts for 5-8% of all cases.
4. Outlet (Subpulmonic)

Geodynamic Classification

1. Group 1: small VSD , normal PVR, small L to R shunt and most of the affected are asymptomatic
2. Group 2: Moderate VSD, variable PVR, significant L to R shunt. there is come FTT and cardiomegaly.
3. Group 3: Large VSD, moderately high PVR, significant L to R shunt. Symptomatic with CCF.
4. Group 4: Large VSD, very high PVR, small or no L ro R shunt, or R to L shunt. Symptomatic with cyanosis and PAH

ECHO-Based Classification

1. Large:= Diameter of aorta
2. Moderate: 1/3 to 2/3rd of diamteter of aorta
3. Small: <1/3rd of diameter
4. Pinhole: <2 mm (detectable by color doppler only)

Pathophysiology of ventricular heart defect

The blood flows from high-pressure left ventricle across the the VSD into the low pressure right ventricle and into the pulmonary artery resulting into pulomanry overcirculation

This leads to increased right ventricular pressure and increased pulmonary artery pressure.

The increased pulmonary venous return to the left sode of the heart sounds in the left atrial dilation

Eisenmenger's syndrome long standing pulmonary pressure results in build up in the pulmonary vascular resistance which reverses the shunt from the right to left and the defect is reversed.

The size of the left-to-right shunt depends on two determinants,

- The size of the VSD and
- Pulmonary vascular resistance (PVR) in relation to systemic vascular resistance.

In case of a restrictive ventricular septal defect (under 0.5 cm^2), higher pressure in the left ventricle is able to cause only a limited left-to-right shunt.

In case of a nonrestrictive VSD (large, usually over 1 cm^2), pulmonary vascular resistance at birth is higher than normal.

The magnitude of the shunt from left-to-right is, therefore, limited. However, with the reduction in the resistance in the next few weeks, the shunt magnitude increases. When the shunt magnitude becomes quite large, VSD becomes symptomatic.

With passage of time, pulmonary vascular obstructive disease begins to develop. As soon as ratio of pulmonary to systemic vascular resistance approaches 1:1, the shunt becomes bidirectional. At this point, the child becomes cyanotic with disappearance of CCF signs. This state is called Eisenmenger complex or syndrome.

The enlargement of the chambers depends on the shunts which further depend on the ratio of the pulmonary to systemic blood flow.

When the ratio is under 1.75:1, the shunt is small, appreciable enlargement of the chambers does not occur and pulmonary vascular bed is by and large normal.

When, on the contrary, the ratio is above 2.5:1, the shunt is large, and left atrial and ventricular volume overload and right ventricular and pulmonary arterial hypertension occur. The large volume of pulmonary blood flow causes enlargement of the pulmonary artery trunk, left atrium and left ventricle.

Clinical features of VSD

If septal defect is small, the patients are asymptomatic and the defect is detected incidentally during a routine clinical examination.

There is a high spontaneous closure during the first year of life

In children with large defects

Signs of congestive cardiac failure such as tachydarcia, tachypnea, excessive sweating associated with feeding , hepatomegally.

Frequent upper respiratory tract infections

Poor weight gain (failure to thrive)

Feeding difficulties

Exercise intolerance

exertional dyspnea, etc.

Diagnosis of VSD

In symptomatic patients, there is moderate biventricular heart enlargement.

On **auscultation** a ***loud pansystolic murmur*** will be heard maximally down the left sternal border (3rd, 4th and 5th intercostal spaces). This murmur is accompanied by a thrill.

Also a functional diastolic murmur may be heard because of a large blood flow across the mitral valve, and its usually heard best over the apex.

There may be presence of splitting of the pulmonary second heart sound (P2) in the presence of pulmonary hypertension. In such patients, a pulmonary diastolic murmur may also be found.

In older children, the additional findings may be in the form of wide pulse pressure and an early diastolic murmur at the base.

These findings suggests development of aortic regurgitation as a complication of VSD (usually subpulmonic).

A chest x-ray is usually normal but may reveal minimal cardiomegally and slight increase in pulmonary vascularity. In large VSD, it shows a large left-to-right shunt with enlarged heart (both ventricles and left atrium), enlarged pulmonary artery and plethoric lung fields (overvascularity) with or without hilar dance

Electrocardiogram is usually normal in small defect but shows features of left ventricular hypertrophy

In large VSDs it shows biventricular hypertrophy with notched or peaked P waves.

A **2-dimensional echocardiogram** shows features of left atrium and ventricle volume. An echocardiogram is also important in showing the position and size of the septal defect.

Cardiac catheterization and **selective angiocardiography** are important in locating the site of the shunt.

Treatment

Treatment of ventricular septal defects depends on whether there is a small or larger ventricular septal defect.

General treatment measures for VSD include attention to good nutrition with treatment of iron-deficiency anemia and other nutritional deficiency states.

Management of congestive cardiac failure (CCF) and recurrent chest infection are treated on usual lines

Small VSD

Cardiac catheterization is performed to insert a ventricular occlusion device for muscular defects

Prophylaxis antibiotics for upto 6 months for infective endocarditis prevention

No surgical repair or cardiac drugs required.

Large VSD

cardiac failure management protocol is initiated with the use of cardiac glycosides such as digoxin and diuretics preferably frusemide.

Avoid oxygen because oxygen is a potent pulmonary vasodilator and will increase blood flow to the pulmonary artery

Increase carolic ntake; fortify formula or breast milk and give supplemental nasogastric feeds as needed.

Infective endocarditis prophylaxis for 6 months after surgery or insertion of a ventricular occlusion device.

Cardiac catheterization

Surgery is usually done before the age of one year.

Follow up to monitor ventricular function

Indications of surgery in VSD

Surgery is indicates in :

1. Symptomatic patients with VSD where medical therapy has failed to control symptoms, regardless of age
2. Large ventricular septal defect with PAH
3. Supracristal VSD of any age
4. VSD subject over 2 years of age with Qp: Qs ratio >2:1

Complications of ventricular septal defect

1. Frequent upper respiratory tract infections
2. Repeated episodes of congestive heart failure
3. Failure to thrive
4. Infective endocarditis
5. Pulmonary hypertension and its complications (Eisenmenger syndrome)
6. Pulmonary stenosis (Gasul’s VSD)
7. Pulmonary hypertension
8. Aortic insufficiency
9. Aortic regurgitation.