SUB-AORTIC VSD CLOSURE USING AN AMPLATZER-TYPE PDA OCCLUDER-A CASE REPORT
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Abstract:
VSD is the most common form of congenital heart disease and accounts for 15 to 20 percent of all congenital heart defects. Successful percutaneous transcatheter device closure of muscular and perimembranous VSDs have been reported. The closure of perimembranous VSD is technically more challenging because of its proximity to aortic valve and the conduction system. We report a case of subaortic VSD with good rims being closed successfully with a Cardiofix PDA device. We used Ansels sheath (Cook) for the deployment of the device. Due to its hydrophilic covering Ansel sheath has excellent trackability and can be maneuvered across the VSD without a stiff wire support.

Keyword: VSD, Amplatzer type PDA occluder, Ansels sheath, Cardiofix PDA device

Sub-Aortic VSD closure using an Amplatzer-type PDA occluder – A Case Report

Introduction:
Ventricular Septal Defect (VSD) is the most common form of congenital heart disease and accounts for 15 to 20% of all congenital heart defects. VSDs are classified into three major categories according to their location and margins. Muscular VSDs are bordered entirely by the myocardium and can be trabecular, inlet or outlet in location. Membranous VSDs have inlet, outlet or trabecular extension and are bordered in part by the fibrous continuity between the leaflets of an AV valve and an arterial valve. Doubly committed subarterial VSDs are situated in the outlet septum and are bordered by the fibrous continuity of the aortic and pulmonary valves. Successful percutaneous transcatheter device closure of muscular and perimembranous VSDs have been reported(1). Trabecular VSDs have proven more amenable to this technique because of their straight-forward anatomy and muscular rim to which the device attaches well and results in excellent closure rates with a low procedural mortality. The closure of perimembranous VSD is technically more challenging because
of its proximity to aortic valve and the conduction system (2). Here we report a case of subaortic VSD with good rims being closed successfully with a Cardiofix PDA device. Its also noteworthy that we used Ansel sheath (Cook) for the deployment of the device. Due to its hydrophilic covering Ansel sheath has excellent trackability and can be maneuvered across the VSD without a stiff wire support.

Case Report:
A 24-year-old female, diagnosed to have “cardiac disease” since childhood, presented to us with dyspnoea on exertion class II of 3 years duration. She gave history of recurrent respiratory infections during childhood. There was no history of cyanotic spells or squatting episodes during childhood. She had a relative asymptomatic phase during her adolescence. General examination was unremarkable. Cardiovascular system examination showed pulse 92/mt, regular in rhythm and of normal volume. Blood pressure was 116/88mmHg in the right upper limb in supine position. Jugular venous pressure was not elevated. Left ventricular apical impulse was felt in the left 5th intercostal space ½ inch lateral to the mid clavicular line. There was grade II left parasternal heave. There was a systolic thrill in left lower parasternal border. Grade 4/6 pansystolic murmur heard in the left parasternal region in the 3rd and 4th intercostals spaces, which increased during expiration, isometric handgrip and decreased with valsalva maneuver.

Electrocardiogram showed sinus rhythm 92/mt with voltage criteria for LVH. Chest X ray showed cardiothoracic ratio of 55% with left ventricular apex and plethoric lung fields. Echocardiogram showed a 3.4 mm subaortic ventricular septal defect, partially closed by the septal leaflet of tricuspid valve, with left-to-right shunt and a gradient 134mmHg across the defect. Left ventricular function was normal. There was moderate tricuspid regurgitation.

The aortic valve was tricuspid with no aortic regurgitation. Cardiac catheterization study showed oxygen step up at RV level, Qp/Qs 1.32 with PVR and PVRI 2.6 and 3.3 wood units respectively. Systolic, diastolic and mean aortic pressures were 122/75(93) mmHg and pulmonary pressures were 32/8(19) mmHg. Left ventricular angiogram in LAO cranial (55 degree LAO and 15 degree cranial) view showed 4mm subaortic VSD with left-to-right shunt. Closure of VSD was considered based on:

1. Past history of repeated lower respiratory tract infections
2. Electrocardiographic evidence of left ventricular hypertrophy by voltage criteria
3. Pulmonary plethora with cardiothoracic ratio of 55% in chest X ray
4. NYHA class II dyspnoea on exertion. It was decided to close the subaortic VSD with a PDA device in view of smaller VSD size and adequate rims. As the angiographic size of the VSD was 4mm, it was decided to choose 8-6mm Cardiofix PDA device for the septal closure.

Procedure:
After obtaining written informed consent, she underwent VSD device closure on 14/03/13 under controlled sedation after obtaining informed consent. A 6F 11cms sheath was inserted percutaneously in the right femoral artery (RFA) and a 7F short sheath in the right femoral vein (RFV). The RFV access was later changed over to Ansels sheath for femoral venous access. The VSD was crossed from the left.
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was later changed over to Ansels sheath for femoral venous access. The VSD was crossed from the left ventricular aspect using a 0.035 inch hydrophilic wire (Terumo, Japan) within a 5F Judkins Right (JR) catheter. The wire was then advanced into the left pulmonary artery. This was snared out from the femoral venous side using a 6F 20mm gooseneck snare. The RFV access was then exchanged for a 7F 55cm Ansel Sheath (Cook) which was then advanced across the septal defect and positioned in the ascending aorta (over the exteriorized wire while using traction on both ends). A 8-6mm Cardiofix PDA device was attached to a delivery cable and taken through the Ansel sheath. Keeping the aortic disc deployed, the device was pulled back from the aorta into the left ventricle and deployed against the septal defect. The position was confirmed by check LV angiograms and transthoracic echo. After deployment, echo showed optimal apposition of the device and no residual shunt. 3500 units of unfractionated Heparin was given intravenously during the procedure. She was given 1g cefazolin injection as infective endocarditis prophylaxis prior to the procedure. The arterial and venous sheaths were removed immediately after the procedure and hemostasis attained by manual compression. There were no vascular or any other immediate post procedure complications. She was observed for 24 hours in the hospital. She was initiated on double antiplatelet therapy (DAPT – Aspirin 75mg + clopidogrel 75mg) and was advised to continue for 6 months. 2D echocardiography done after 24 hours showed good apposition of the device and no residual shunt. She was discharged in a hemodynamically stable condition and advised to review after 6 months for echocardiographic assessment.

Discussion:
VSD is the most common form of congenital heart disease. Based on the hemodynamic significance, VSDs can be divided into:

1. Restrictive VSD is a defect that is associated with significant pressure gradient between the left ventricle and the right ventricle with pulmonary to aortic systolic pressure ratio <0.3 and is accompanied by a small shunt with Qp/Qs <1.4.

2. Moderately restrictive VSD is accompanied by a moderate shunt with Qp/Qs 1.4 to 2.2 and pulmonary to aortic systolic pressure ratio less than 0.66.

3. A large or non restrictive VSD is accompanied by a large shunt with Qp/Qs >2.2 and pulmonary to aortic systolic pressure ratio more than 0.66. Eisenmenger VSD has a systolic pressure ratio of 1 and Qp/Qs less than 1 or a net right-to-left shunt (1).

Spontaneous closure occurs in 30 to 40% of patients with membranous and muscular VSDs during the first 6 months of life. Congestive heart failure develops in infants with large VSDs but usually not until 6 to 8 weeks of age. Pulmonary vascular obstructive disease may begin to develop as early as 6 to 12 months of age in patients with large VSDs, but the resulting right to left shunt usually does not develop until the teenage years. A perimembranous defect in an immediately subaortic location or any doubly committed VSD may be associated with progressive aortic regurgitation. Infundibular stenosis may develop in some infants with large defects and result in a decrease in magnitude of the left-to-right shunt with an occasional occurrence of a right-to-left shunt. ECG mirrors the size of shunt and the degree of pulmonary hypertension. Small restrictive VSDs
usually produce a normal tracing. Moderate sized VSDs produce a broad notched p wave characteristic of left atrial overload as well as evidence of left ventricular volume overload namely deep Q and tall R waves with tall T waves in leads V5 and V6 and perhaps eventually atrial fibrillation. After repair, the ECG is usually normal with right bundle branch block. The chest radiograph reflects the magnitude of the shunt as well as the degree of pulmonary hypertension. A moderate sized shunt causes signs of left ventricular dilation with some pulmonary plethora. Transthoracic echocardiography can identify the location, size and hemodynamic consequences of the VSD as well as associated lesions like aortic regurgitation, right ventricular outflow tract obstruction or left ventricular outflow tract obstruction. Cardiac catheterization may be required when the hemodynamic significance of a VSD is questioned or when assessment of pulmonary artery pressures and resistances is necessary. In some centres therapeutic catheterization is performed for percutaneous closure. The presence of a significant VSD in the absence of irreversible pulmonary hypertension warrants closure of the defect. Significant VSD denotes Qp/Qs more than 1.5, pulmonary artery systolic pressure more than 50mmHg, increased left ventricular and left atrial size or deteriorating left ventricular function in a symptomatic patient. If pulmonary arterial resistance is less than 7 Wood units, closure can be safely undertaken if there is a net left-to-right shunt of at least 1.5:1 or strong evidence of pulmonary reactivity with challenge with a pulmonary vasodilator like oxygen or nitric oxide(3). Successful percutaneous device closure of the trabecular (muscular) and perimembranous VSD has been reported. Trabecular VSDs have proven more amenable to this technique because of their favourable anatomy and muscular rim to which the device attaches well and usually result in excellent closure rates with a low procedural mortality(1). Immediate as well as short-term results are good. The closure of perimembranous VSD is technically more challenging because of its proximity to valve structures and careful selection of patients is required. It should be performed only in centres with appropriate expertise(2). Short-term follow up data show complete closure in 96% patients with the development of aortic or tricuspid regurgitation or the development of complete heart block in less than 5% of patients(1). Even though the calculated left-to-right shunt was not significant (Qp/Qs 1.32), this was probably due to partial closure of the septal defect by the septal leaflet of tricuspid valve. It is likely that she had a much more significant left-to-right shunt earlier as indicated by persisting plethora in the lung fields and past history of recurrent respiratory infections.

References:
1. Braunwald’s Heart Disease – A Text book of Cardiovascular Medicine, 9th edition, Pages 1430-
2 Grossman’s Cardiac Cathetrisation, Angiography and Intervention, 7th edition, Pages 623-25
3 Pediatric Cardiology for Practitioners Myung k. Park, 5th edition, Pages 166-75.

Fig:1. 12 lead ECG showing sinus rhythm 92/mt with normal QRS axis and LVH by voltage criteria

Fig:2. Chest Xray PA View showing CTR 55% with LV apex and plethoric lung fields

Fig:3. 2D echocardiography in PLAX view showing 3.4mm perimembranous VSD with left-to-right shunt

Fig:4. 2D echocardiography in A5C view showing perimembranous VSD with left-to-right shunt

Fig:5. LV angiogram in LAO cranial view showing VSD 4.22mm with left-to-right shunt
Fig:6. Deployed device across the VSD prior to release. LV angiogram shows no significant shunt across.

Fig:7. VSD closed with PDA Occluder. LV angiogram shows no residual shunt.

Fig:8. Well apposed PDA occluder across the VSD in echocardiographic A5C view.

Fig:9. Device across the VSD with no colour flow across (echocardiographic A5C view).