Percutaneous Closure of Perimembranous Ventricular Septal Defect with Amplatzer Device

Col P Bharadwaj*, Col A Banerji⁺, Col R Datta vsm[#], Col H Singh^{**}, Lt Col AK Ghosh⁺⁺,
Wg Cdr G Keshavamurthy^{##}

Abstract

Background: The Amplatzer perimembranous ventricular septal occluder is an innovative device for percutaneous closure of perimembranous ventricular septal defects (PMVSD). In appropriately selected cases this procedure is safe and effective.

Methods: Fourteen patients with the mean age 10.53 years (range 18 months to 55 years) and mean body weight 20.64 kg (range 6 to 52 kg) underwent PMVSD closure.

Result: The PMVSD mean diameter was 5.28 mm (range from 4 to 9 mm). Implantation was successful in 92 % of the cases and all patients had complete occlusion of the shunt within three months.

Conclusion: Device orientation was excellent in all cases. Device-related aortic insufficiency, tricuspid insufficiency or left ventricular dysfunction was not observed. One patient had embolisation of the device and another had complete heart block which required a permanent pacemaker implantation. The excellent short term results need to be confirmed over long-term follow-up.

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Key Words : Perimembranous ventricular septal defect; Arrhythmias, Percutaneous closure

Introduction

Ventricular septal defect (VSD) accounts for approximately 20% of all forms of congenital heart disease [1]. About 75% of all VSDs are located in the mid portion of the upper region of the ventricular septum and are related to the aortic valve. Such VSDs are termed perimembranous trabecular defects or infracristal, subaortic, or type II Kirklin. Children with volume-overloaded left atrium and ventricle due to a VSD require surgical repair to prevent pulmonary artery hypertension, ventricular dilation, arrhythmias, aortic regurgitation, development of double chambered right ventricle and the risk of endocarditis [2]. Until recently the only way of doing this was open-heart surgery. This is a major procedure that necessitates a thoracotomy, heart lung bypass, blood transfusion in some cases, permanent scar and potential risks of complete heart block, early and late arrhythmias, post pericardiotomy syndrome and even death [3]. Surgery at present can be accomplished with minimal risk (less than 1%), but because of the obvious discomfort and relatively long recovery period, alternative procedures have been developed.

The development of a transcatheter occlusion technique that can safely and effectively close these

defects would be welcomed by cardiologists, patients, and their families. The use of the button and Rashkind devices to close perimembranous / membranous VSDs has been reported with acceptable results [4,5]. However, both the Rashkind and the button devices are cumbersome to use and they are associated with a high incidence of residual shunt. The excellent initial results of catheter closure of single and Swiss cheese muscular VSDs with the Amplatzer muscular ventricular septal defect occluder [6–8] has led to the development of a new device specifically designed for VSDs located in the perimembranous (Kirklin type II) portion of the septum. This device addresses the issue of the peculiar anatomical characteristics of the membranous ventricular septum, including its thickness and the vicinity to the tricuspid and aortic valves and the conduction system. We report a series of the first fourteen patients at our institution who underwent VSD closure using this device.

Material and Methods

Fourteen patients (five males and nine females) with perimembranous ventricular septal defect (PMVSD) underwent transcatheter closure using the Amplatzer perimembranous VSD occluder (PMVSDO) from December 2003 to April 2007. Informed consent was obtained from the patients or their guardians. Patients who were either

*.+.#Senior Advisor (Medicine & Cardiology), MH (CTC), Pune. **.Senior Advisor (Medicine & Cardiology), 166 MH,C/O 56 APO. **.Classified Specialist (Medicine) & Cardiologist, CH (CC), Lucknow. **.Classified Specialist (Medicine & Resident Cardiology), MH (CTC), Pune.

Received : 1.5.2007; Accepted : 01.3.2008 e-mail-prashantbharadwaj@gmail.com

symptomatic or had clinical and transthoracic echocardiographic diagnosis of significant isolated PMVSD with left ventricular volume overload (increased left ventricular end-diastolic dimensions indexed for age and body surface area) were selected for the procedure. Patients with aortic regurgitation, aortic rim to PMVSD distance less than 2 mm, ventricular septal aneurysm or septal tricuspid leaflet closing the PMVSD and severe pulmonary hypertension were excluded from this study. The salient characteristics of amplatzer perimembranous VSD occluder device are given in

Fig. 1: Side profile and en face views of the Amplatzer perimembranous ventricular septal occluder. In A, the 0.5 mm superior rim oriented toward the aorta is visualised with the longer, 5.5 mm inferior rim below. The short, 2 mm waist is also well seen. In B, the circular left ventricular disk with minimum shoulders that could interfere with the aortic valve is seen.

Fig. 2: Pusher catheter and Amplatzer perimembranous ventricular septal occluder showing flattened area of the socket in the pusher catheter and the matching flattened superior surface of the microscreen on the device.

Fig. 3: A: Left ventricular (LV) angiogram (LAO view) demonstrating perimembranous VSD (arrow). B: Cine image demonstrating the arteriovenous loop. C: LV angiogram after the LV disk has been deployed demonstrating good device position. D: Final angiogram showing no residual shunt.

Fig. 1,2. The procedure were performed under general anaesthesia in ten patients. The other patients underwent closure under conscious sedation and trans-thoracic echocardiography (TTE) guidance. The standard imaging locations were parasternal long axis and short axis respectively, apical four-chamber and five chamber views. Specific attention was paid to the size and location of the PMVSD, the distance of the PMVSD from the aortic rim, atrioventricular and semilunar valve competence. Access was obtained in the femoral artery (4–6 Fr sheath) and the femoral vein (6–8 Fr sheath). The patients were given heparin 150 IU/Kg (maximum 7500 units) to achieve an activated clotting time of 200–250 seconds at the time of device placement. Routine right and left heart catheterisation was performed to assess the degree of shunting and to evaluate the pulmonary vascular resistance. Left ventricular (LV) angiography in single plane (60°LAO/20° cranial) was performed to define the location and size of the PMVSD (Fig 3 A). The appropriate device size was chosen to be 1–2 mm larger than the VSD size as assessed by the TTE size.

The first step in the closure sequence is the crossing of the PMVSD from the LV side using a 4–6 Fr Judkins right catheter. A soft-tipped wire was advanced through this catheter to the pulmonary artery. The Judkins catheter was manoeuvred into the right ventricle (RV) and the soft tipped wire was exchanged for an exchange length 0.035" soft J-tipped Noodle guide wire (AGA Medical Corp, MN, USA) which was taken to a branch pulmonary artery. The tip of this wire was snared from the femoral vein using a gooseneck snare (Microvena, MN, USA). The wire was exteriorized out the right femoral vein in order to provide a stable arteriovenous loop (Fig.3B). A 7–8 Fr long TorqVue delivery sheath and dilator (AGA Medical Corp, MN, USA) were advanced from the femoral vein across the LV and positioned in the ascending aorta.

The dilator was removed and the sheath was then repositioned into the LV apex by pulling the sheath slowly from the ascending aorta and simultaneously pushing the wire from the arterial side. The appropriate size device was then loaded as mentioned above. The entire assembly (pusher catheter with the loader and cable) was then advanced through the sheath until the device reached the tip of the sheath. The sheath was then slowly pulled away from the apex until it reached the outflow tract, between the septum and the anterior

mitral valve leaflet. This was confirmed by TTE and LV angiography (Fig. 3C). Retracting the sheath over the pusher catheter and the cable deployed the LV disk in the correct orientation by ensuring that the platinum marker on the device faced towards the left ventricular apex. Further retraction of the sheath led to the deployment of the right disk on the right ventricular side of the defect. TTE was performed to ensure the complete closure of the defect after which the device was released by counterclockwise rotation of the pin vise. Following the device release, a repeat LV angiography (Fig 3D) and complete TTE study were performed to confirm correct device placement, to assess residual shunting and any obstruction or regurgitation induced by the device (Fig 4A-D).

The patients received a dose of cephazolin at 20mg/kg during the catheterisation procedure and two further doses at eight-hour intervals. The patients were hospitalised for five days and assessed daily for the development of conduction disturbances. Observation of subacute bacterial endocarditis prophylaxis was recommended for six months or until complete closure was obtained. Patients were instructed to take 3 mg/kg aspirin daily for six months and to avoid contact sports for one month. Patients were followed up monthly for six months and yearly thereafter with a complete clinical examination, a TTE, chest radiograph, and an electrocardiogram.

Results

The clinical presentation of the patients is given in Table 1. Eleven patients were symptomatic and three asymptomatic. The mean age of patients was 10.53 years (ranged 18 months to 55 years). The mean body weight of patients was 20.64 kg (ranged from 6 to 52 kg).

The mean diameter of VSD measured by transthoracic echocardiography was 5.28 mm (range 4 to 9 mm). The mean distance from the aortic valve to the rim of the ventricular septum was 5.3 mm (range 2.5 to 7 mm). The mean diameter of the VSD on cardiac catheterisation was 5.89 mm (range 4.0 to 9.6 mm). The mean pulmonary artery pressure was 21.71 mm of Hg (range 20 to 24 mm Hg), while the mean rate of quantity of pulmonary flow/quantity of systemic flow (Q_p/Q_s) was 1.65 (range 1.3 to 2.0). Table 2 shows echocardiographic and cardiac catheterisation data.

Thirteen patients underwent successful and complete closure of their defects. The device sizes employed are given in Table 3. In patient number two the device could not be placed because of the septal tricuspid leaflet impinging and closing the VSD and the resulting inability to deploy the right ventricular disk adequately. We experienced embolisation of the 6 mm device to the right femoral artery in one patient (no 12) which was successfully retrieved with the help of the 5F tissue biopsy forceps (Olympus endoscopic forceps). The patient underwent successful closure of the VSD using a 8

Fig. 4 : A: Apical five chamber showing PMVSD. B: Colour flow across the defect. C: Short axis view showing the device well seated across the defect. D: Device across the PMVSD in apical five chamber view.

Table 1**Demographic profile of patients who underwent closure**

Sex	Age	Weight (kgs)	Symptoms
Male	18 months	6	Recurrent respiratory infections, feeding difficulty, poor weight gain
Female	4 years	11	Recurrent respiratory infections, feeding difficulty, poor weight gain
Female	16 years	36	Nil
Female	16 years	46	Nil
Female	13 years	22	Screening in school examination for underweight
Female	8 years	19	Palpitations, cardiac murmur
Male	4 years	12	Recurrent respiratory infections, poor weight gain
Female	55 years	52	Non anginal pain chest
Female	4 years	11	Recurrent respiratory infections, poor weight gain
Male	3 years	9	Recurrent respiratory infections, feeding difficulty, poor weight gain
Female	6 years	15	Recurrent respiratory infections, poor weight gain
Male	7 years	16	Recurrent respiratory infections, poor weight gain
Female	7 years	15	Recurrent respiratory infections, poor weight gain
Male	3 years	9	Feeding difficulty, poor weight gain

mm PMVSD occluder after 10 days. There was residual trivial shunt in two patients which closed spontaneously in three months. There was no de novo tricuspid or aortic insufficiency after the procedure or during follow-up. LV function was normal in all patients at discharge and during follow-up.

The incidence of conduction disturbances is shown in Table 3. During sheath placement all patients experienced ventricular arrhythmias, eight patients developed transient right bundle branch block (RBBB), two developed left bundle branch block (LBBB) and one developed transient complete heart block (CHB). These arrhythmias were hemodynamically well tolerated and did not portend the development of post procedure blocks. We could not correlate the size of the device with the occurrence of arrhythmia. When the blocks were noticed post procedurally, most patients (4/5) were asymptomatic. These blocks were noticed on the second day except in one case (patient number 7), which appeared after five days. These blocks resolved spontaneously in seven to twenty two days. Two patients developed complete heart block and both were treated with oral prednisone 1mg/kg for five days which resulted in normalisation in one patient. The other patient had a persistent complete heart block at three months and a permanent pacemaker implantation was undertaken. Patient number 8, developed ischemic complication of the limb with pain, pallor and loss of pulses which was managed conservatively and improved to complete functional recovery over the next three months.

Table 2**Echocardiographic and cardiac catheterisation data**

Trans-thoracic echocardiography (TTE)			Cardiac catheterisation		
VSD size	Doppler gradient	Aortic valve rim-VSD distance	Qp/Qs	Pulmonary artery pressure	VSD size
5mm	64 mm of Hg	3 mm	2.0	37/18/23	6 mm
4mm	64 mm of Hg	3 mm	1.8	39/14/24	4 mm
4mm	68 mm of Hg	5 mm	1.3	40/15/23	4 mm
4mm	68 mm of Hg	4 mm	1.5	20/10/14	4 mm
5mm	60 mm of Hg	3.2 mm	1.9	32/14/20	5.5 mm
4mm	60 mm of Hg	2.8 mm	1.6	36/13/22	3.6 mm
9mm	68 mm of Hg	4.8 mm	1.8	39/15/23	9.4 mm
8mm	68 mm of Hg	5.8 mm	1.3	40/14/21	9.2 mm
6 mm	68 mm of Hg	4.2 mm	1.9	35/12/20	7.4 mm
6 mm	48 mm of Hg	4 mm	1.9	42/15/24	7.2 mm
4 mm	62 mm of Hg	3.5 mm	1.4	37/13/22	6 mm
5 mm	50 mm of Hg	2.5 mm	1.5	38/12/21	5 mm
6 mm	52 mm of Hg	2.5 mm	1.4	40/16/25	7.2 mm
4 mm	58 mm of Hg	4 mm	1.9	35/14/22	4 mm

Table 3**Size of VSD, device size and conduction blocks**

VSD size TTE	VSD size catheterisation	Device size	Post procedure transient blocks
5mm	6 mm	6	RBBB
4mm	4 mm	Failed	Nil
4mm	4 mm	6	Nil
4mm	4 mm	4	Nil
5mm	5.5 mm	6	Nil
4mm	3.6 mm	4	Nil
9mm	9.4 mm	10	RBBB
8mm	9.2 mm	10	RBBB, CHB
6 mm	7.4 mm	8	Nil
6 mm	7.2 mm	8	Nil
4 mm	6 mm	6	Nil
5 mm	5 mm	8	Nil
6 mm	7.2 mm	8	CHB
4 mm	4 mm	4	RBBB, LBBB, Permanent CHB

Discussion

Percutaneous closure of PMVSDs of different morphologies with Amplatzer PMVSD occluder was safe, and complete in our initial experience. The device is simple, requires a relatively low profile sheath for implantation, while the short waist of the device obviates protrusion into both ventricles and allows recapturing and repositioning if required [9-13]. The eccentric configuration of the device especially on the superior surface imparts a safety from impinging on aortic valve and its function. The device embolised in one case, but there was no incidence of air or thrombus embolisation. The rate of immediate and complete closure in this

series was 92% and 100 % after three months. Ongoing endothelialisation of the device is responsible for the decline in the rate of residual shunting with time, which is comparable to surgical repair [14-15].

We noticed conduction disturbances after five days of the procedure which is at variance with other reports. This mandates constant vigilance and perhaps revision of the advocated protocol of patient discharge on the next day of the procedure. Experience is now emerging with the use of this device in ventricular septal aneurysms, PMVSDs undergoing closure by the septal tricuspid leaflet and with prolapse of the aortic cusps [14].

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Conflicts of Interest

None identified

Intellectual Contribution of Authors

Study Concept : Col P Bharadwaj

Drafting & Manuscript Revision : Col P Bharadwaj, Lt Col AK Ghosh, Wg Cdr G Keshavamurthy, Col A Banerji, Col R Datta

Statistical Analysis : Col P Bharadwaj, Col H Singh

Technical Support : Col P Bharadwaj, Col A Banerji, Col R Datta, Lt Col AK Ghosh, Wg Cdr G Keshavamurthy

Study Supervision : Col P Bharadwaj

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