Device closure of congenital ventricular septal defects with Amplatzer devices: first experiences in Turkey

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Ventricular septal defects (VSD), which cause volume overload, may be closed by interventional method. The success depends on the precise anatomic definition of the defect and its relation to other cardiac structures. We report our first experiences of transcatheter closure of perimembranous and muscular VSD.

Between May 2005 and September 2006, transcatheter closure of VSD was attempted in 38 patients. Implantation was successful in 37 patients. In one patient, the procedure failed because of the long sheath kinking. We observed important complications in three patients: severe tricuspid valve regurgitation, residual VSD and tricuspid valve regurgitation and right bundle branch block in the short-term follow-up.

Transcatheter device closure with Amplatzer device seems to be effective and safe in the treatment of perimembranous and muscular VSDs. Tricuspid valve incompetence may cause problems. Long-term results are required to determine the efficacy and safety.

Key words: ventricular septal defect, Amplatzer, perimembranous, membranous, perventricular.

Ventricular septal defects (VSDs) must be repaired to prevent pulmonary hypertension, ventricular dilatation, aortic regurgitation, and endocarditis. Various occlusion systems allow percutaneous closure of muscular and membranous congenital and acquired VSD[1-4]. Technically, VSD closure by a device is more difficult than with the other defects[1]. The success depends on the precise anatomic definition of the defect and its relation to other cardiac structures. Defects that are close to the semilunar or atrioventricular valves are not suitable for closure with device. With the development of new self-centering Amplatzer devices, an increasing number of defects could be closed in muscular and membranous septae[5-7,8-10]. We report our preliminary experience of transcatheter closure of VSD in different locations.

Material and Methods

Between May 2005 and September 2006, transcatheter closure of VSD was attempted in 38 patients (39 procedures). Ages of patients ranged between 4 months and 27 years (mean: 9 years, median: 8 years). We closed echocardiographically (enlargement of left ventricle [LV] on echocardiography) and angiographically significant VSDs. Mean body weight was 32.5 kg (range: 4-80 kg, median 21 kg) (Table I). Perimembranous VSD (PMVSD) was seen in 25 patients and muscular VSD in 13 patients (multiple muscular VSD in 1). Associated lesion was present in four patients: 1 operated tetralogy of Fallot, 2 patent ductus arteriosus (PDA) which were closed via transcatheter route, and 1 coarctation of aorta (Table I).

On echocardiography, the diameter of VSD ranged from 2 to 10 mm (median: 5 mm). Before transcatheter closure, the ventricular septal anatomy was examined by a detailed transthoracic echocardiography. The ratio of pulmonary to systemic blood flow (Qp/Qs) ranged from 1 to 2.9 (median: 1.8). Pulmonary artery systolic pressure ranged between 19-72 mmHg (median: 26.5 mmHg).

Table I. Demographic and Diagnostic Data

Total patients	38
Male	17
Female	21
Age at implantation	9 years (2 months-27 years)
Muscular VSD	13 patients, 14 interventions (multiple in 1)
Perimembranous VSD	24
Multiple VSD	1
Associated defects:	
PDA	2
Aortic coarctation	1
Operated tetralogy of Fallot	1
VSD size	2-10 mm (median 5 mm)
Qp/Qs	1-2.9 (median 1.8)
Pulmonary artery systolic pressure	19-72 mmHg (median 26.5 mmHg)
Device size	4-11 (median 6)
Failed case	1

PDA: Patent ductus arteriosus. VSD: Ventricular septal defect.

Procedure

Written informed consent for device placement and follow-up evaluation was obtained from the patient or parent/legal guardian. The catheterization procedure was performed under deep sedation and transthoracic echocardiography in 11 patients and general anesthesia and transesophageal echocardiography (TEE) in 27 patients. Heparin was administered during the procedure with 100 IU/kg and repeated if the procedure exceeded 90 minutes with a 50 IU/kg dose. Each patient underwent right and left heart catheterization through the percutaneous transfemoral route. The left anterior oblique (LAO) view with cranial angulation that best profiled the VSD was determined (Fig. 1). The size of the VSD and its distance from the atrioventricular valve was reconfirmed on cineangiography.

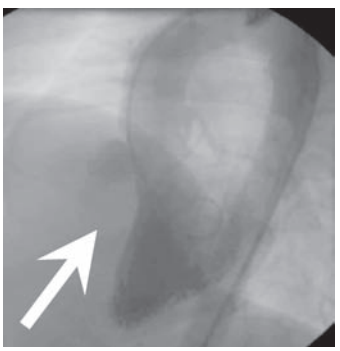

Fig. 1. Aneurysmal VSD: Left ventricular angiogram reveals large aneurysmal VSD (arrow).

Perimembranous VSD Closure

To cross the VSD from the LV, we used 5 Fr cut pigtail and 0.035" glide wire or Roadrunner and advanced it to the pulmonary artery. The catheter was advanced over the wire and exchanged with the noodle wire. This noodle wire was snared in the pulmonary artery or vena cava superior and pulled out through the femoral vein (Fig. 2). Delivery sheath was advanced over the noodle wire and the dilator slowly drawn back, and the sheath was pushed toward the apex of the LV. The device, which was 1-2 mm larger than the size of the VSD, was screwed into the

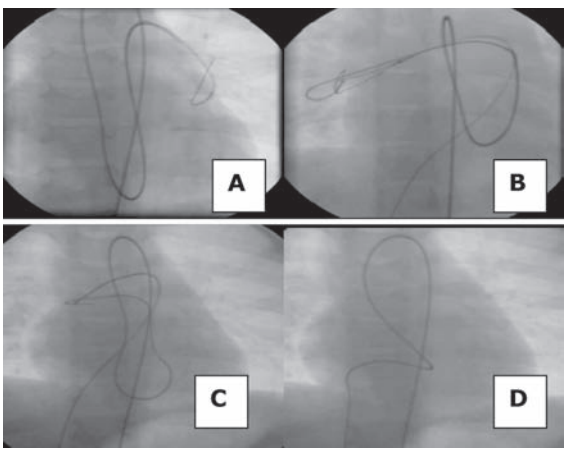

Fig. 2. Creation of arteriovenous loop: A: Cut pig-tail catheter was advanced to the left pulmonary artery via VSD, B: Noodle wire was caught at right pulmonary artery with a snare catheter, C and D: Snare catheter was pulled from the femoral vein and arteriovenous loop was created.

cable. Pin vise was tightened to the cable at the end of the hub of the pusher catheter to prevent premature dislodgement of the device. The device was then advanced through the sheath and the LV disc was delivered midway between the anterior mitral valve leaflet and the left ventricular outflow tract. The entire system is pulled back to the septum. After angiographic and echocardiographic control, the waist was delivered followed by the right ventricular (RV) disc (Fig. 3). Once a good position was confirmed, the pin vise was loosened and the pusher catheter was retracted over the cable. The device was then released by counter-clockwise rotation of the pin vise. A repeat angiogram in the LV was done to assess position of the device and any residual shunt (Fig. 4). If the catheter could be passed easily from the VSD via the RV, there was no need to cross the defect from the LV.

Muscular VSD

Femoral artery and vein were accessed routinely, but internal jugular vein was preferred in the case of VSD located in posterior or apical septum. Angiography was done to define the location and size of the defect. The appropriate device size was chosen 1-2 mm larger than the size of the defect. A long sheath was then placed across the VSD. Once the VSD was crossed, a 0.035 J exchange length wire was positioned in the pulmonary artery. This wire was snared and pulled out to form an arteriovenous loop. An appropriate-sized delivery sheath was then advanced from the femoral or right internal jugular vein over the wire to the apex of the LV and the exchange wire was removed. The left-sided retention disc was delivered in the mid-LV cavity and the entire assembly (sheath-device, cable) was pulled back to the septum. The waist was then delivered by retracting the sheath over the cable. Further retraction of the sheath over the cable will deploy the RV disc (Fig. 5). If the VSD was single, angiography in the LV was sufficient to delineate the position of the device and monitor the steps. However, if multiple VSDs were present, both angiography and TEE were used to verify the device position. Once the correct device position was confirmed, the device was released from the delivery cable using the pin vise.

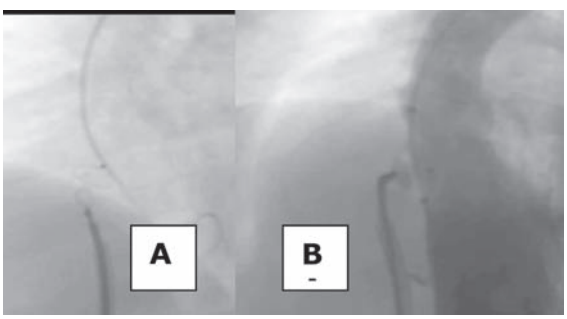

Fig. 3. Release of both discs: A: Both discs were opened, B: Angiography revealed closure of VSD.

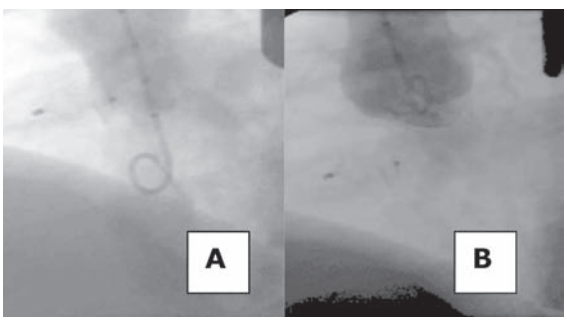

Fig. 4. After detachment: A: Disappearance of shunt on left ventriculogram, B: No aortic regurgitation on aortography.

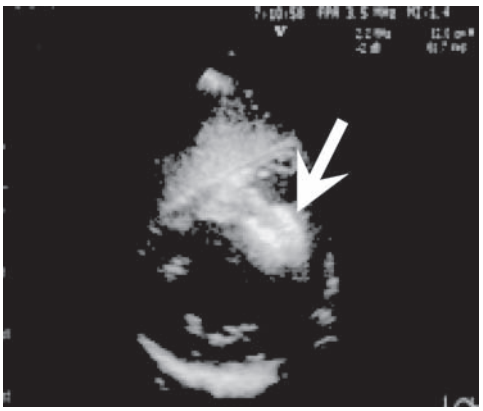

Fig. 5. Closure of midmuscular VSD: A: Both discs were opened, B: There was no shunt after the detachment.

Perventricular Closure Protocol

The perventricular approach was preferred in one four-month-old child with intractable heart failure. The procedure was done in the operating room under TEE guidance. The child was intubated via transnasal approach and a median sternotomy was done. The diameter of the defect was measured as 5 mm by TEE. For RV puncture, a 5-0 polypropylene purse-string

was placed at the carefully chosen location on the beating heart. An 18-gauge needle was introduced into the RV cavity and a 0.035" guide wire was passed through the needle and advanced into the LV cavity. A 8 Fr short introducer sheath with a dilator was fed over the wire into the LV cavity. The dilator was removed and the sheath tip positioned in the LV cavity. The 6 mm device was then screwed to the cable and pulled inside a 7 Fr loader. The device was released by the same method described in the previous method of muscular VSD closure. After the release of the device, TEE was repeated to control the residual shunt.

Cefazolin was administered during the procedure and subacute bacterial endocarditis (SBE) prophylaxis was recommended for six months following complete closure of the defect. All the patients except two - the hybrid procedure and patient with right bundle branch block - were hospitalized one day. Aspirin at a dose of 2-4 mg/kg/day and SBE prophylaxis for six months were advised. Patients were scheduled for follow-up at 1, 3, 6 and 12 months, with physical examination, telecardiogram, ECG, transthoracic echocardiography (Fig. 6) and 24-hour Holter monitoring.

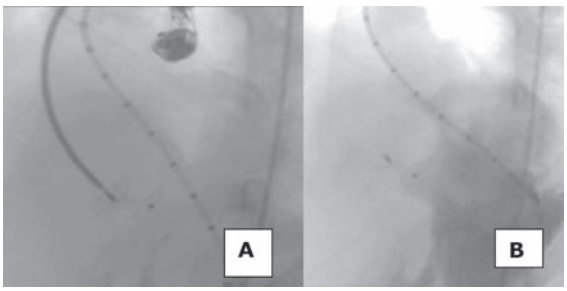

Fig. 6. Echocardiographic appearance of midmuscular VSD after closure with Amplatzer muscular VSD closure device: Both discs were visible (arrow).

Results

Transcatheter deployment of the device was successful in 37 of 38 patients (97.4%). VSD closure resulted in complete disappearance of flow immediately across the defect in 34 procedures (91%). Two procedures were performed in only one patient.

Perimembranous VSD was closed in 24 out of 25 patients (96%). In one patient, the procedure failed because of kinking of the long sheath in spite of repeated procedures. VSD was crossed from the LV and arteriovenous loop created in 23. In one, the VSD was crossed from the RV and procedure was uneventful (Table II). The mean diameters of VSD base and tip were 7.1±3.2 mm and 4.4±1.7 mm, respectively. The median device diameter was 6 mm. In this group, scopy and procedure time were 25±12 minutes and 109±26 minutes, respectively.

All cases were closed successfully in those with muscular VSD (Table III). In seven patients (54%) with anterior or midmuscular VSD, we used femoral vein to load the device. In the other five patients, four with midmuscular and one with multiple apical muscular VSD, internal jugular vein was used for closure protocol. In the patient with multiple muscular VSD and pulmonary vascular disease, who had a previous operation for perimembranous large VSD, two Amplatzer muscular VSD devices were implanted to apical muscular VSDs in separate procedures. During the third attempt, we found multiple tiny muscular VSDs and suprasystemic pulmonary artery pressure and we abandoned the procedure. In this group, the mean VSD diameter was 6.2±1.8 mm. The scopy time and procedure time were 30±16 minutes and 104 ±50 minutes, respectively. An infant with large midmuscular VSD was closed by hybrid procedure uneventfully.

At follow-up with a mean of 12.5±5.2 months, no patient had developed intravascular hemolysis or infective endocarditis. The device position remained stable and there was no evidence of problem in the device.

Complications

There were two major (1 in muscular and 1 in perimembranous group) and one minor complication in our patients. One patient with high muscular VSD had moderate tricuspid valve regurgitation and residual shunt on the VSD on echocardiography. This patient underwent open heart surgery for the closure of the VSD and device removal. During the surgery, we observed that the chordae tendineae of the septal leaflet of the tricuspid valve had been attached to the rim of the device. In another patient, with perimembranous VSD and aneurysm formation, the device caused moderate to severe tricuspid valve regurgitation six months after the implantation. Repair of

Table II. Clinical, Hemodynamic and Procedure Data in Patients with Perimembranous Ventricular Septal Defects

	Age	Weight (kg)	LVEDD (mm)	VSD Type	Qp/Qs	PAP (mmHg)	VSD Type (mm)	VSD Base (mm)	Device (mm)	ST (min)	PT (min)	Follow-up (mos)	Complication
1	16	56	39	PM	1.8	22	4	5.5	6	19	75	18	
2	9	36	43.6	PM-AP	1	22	2.5	12	8	41	120	18	
3	3	16	39	PM-AP	1	32	5	5.1	6	31	120	18	
4	12	78	53.3	PM-AP	1.08	32	4.4	13	6	65	180	18	
5	17	65	54	PM		34	8.8	8	11	10	120	18	
6	6	23.5	40	PM			6	5	8	26	105	18	
7	7	21	38	PM-AP	1.21		5	8	6	24	100	18	
8	8	20.5	45	PM-AP	2	22	4.2	8	6	22	120	15	
9	4	14	42	PM		19	6.4	8.5	8	12	60	18	
10	6	21	45	PM	1.5	26	4.7	2.5	6	25	105	12	RBBB
11	27	80	55	PM-AP	1	25	7.8	7.8	10	16	75	18	
12	18	72	53	PM	1.8	27	3.5	2.5	6	41	145	13	
13	5	16	59	PM	1.9	26	5	2.3	6	19	125	13	
14	9	29	44	PM	1.6	24	2	5.8	4	23	100	8	
15	7	24	45	PM	1.33	28	5		9	22	80	10	
16	0.8	8	35	PM	2	61	6		6	15	140	11	
17	14	42	45	PM	2	30	4		9	30	120	10	
18	26	64	51	PM			3.5	8	6	21.2	85	9	
19	19	57	47.6	PM	1.7	23	3	11	6	21.6	90	9	
20	8	25	42	PM-AP	1.1	13	2.3	9.5	4	44.8	110	9	
21	3	21	40	PM	1.6	25	3.1	9.3	6	22	110	10	
22	5	17	34	PM	1.8	25	2.2	5.6	6	27	120	6	
23	3	12	38	PM-AP	1.5	24	5	2.3	4	31	135	3	TR
24	6	19	42	PM	1	45	3	10.3	6	11	90	1	

AP: Aneurysmal perimembranous. LVEDD: Left ventricle end diastolic dimension. PAP: Pulmonary artery systolic pressure. PM: Perimembranous. PT: Procedure time. RBBB: Right bundle branch block. ST: Scopy time. TR: Tricuspid regurgitation.

Table III. Clinical, Hemodynamic and Procedure Data in Patients with Muscular Ventricular Septal Defects

	Age (years)	Weight (kg)	LVEDD (mm)	Qp/Qs	PAP (mmHg)	VSD Diameter (mm)	VSD Type	Device (mm)	ST (min)	PT (min)	Follow-up (mos)	Complication
1	5	17	41	2	28	10	MM	12	61	150	72	
2	7	21	43	2	24	5.9	AM	8	39	120	1	TR
3	10	26	44	2.8	28	8.6	MM	8	11	100	3	VPB
4	4	16	35.2	2.9	32	7	AM	8	27	60	10	
5*	8	21	48	2	72	5	A	8	50	225	8	
6	8	32	44	1.5	38	7.3	AM	9	44	135	1	
7	2	12	38	1	25	5.4	AM	8	15	90	10	
8**	0.2	5	25	2.5	60	5	MM	6	–	35	5	
9	17	70	56	1.8	28	6	MM	8	19	70	2	
10	2	9	37	4.8	36	4	MM	6	34	135	–	
11	12	25	38				MM	10	39	100	2	
12	16	50	46	2.3	24	6	MM	10	13	75	1	
13	17	52	67	1.9	26	4	MM	6	19	60	1	

*: Multiple apical VSD. **: Hybrid procedure. AM: Anterior muscular. A: Apical. LVEDD: Left ventricle end diastolic dimension. PAP: Pulmonary artery systolic pressure. MM: Midmuscular. ST: Scopy time. PT: Procedure time. TR: Tricuspid regurgitation. VPB: Ventricular premature beats. VSD: Ventricular septal defect.

RV disc-associated tricuspid septal leaflet motion abnormality was attempted by valve plasty procedure. In one patient, we observed right bundle branch block on the ECG on the day of the procedure. There was no sign of second- or third-degree atrioventricular block in Holter recordings during a follow-up of 10 months.

We observed two temporary problems. There was moderate tricuspid regurgitation in one patient with high muscular VSD following device closure, which disappeared after one day. Frequent ventricular ectopic beats following closure of midmuscular VSD disappeared at eight months of follow-up.

Discussion

This report describes the experience of a single center in transcatheter closure of congenital perimembranous and muscular VSDs with Amplatzer perimembranous and muscular VSD devices. Amplatzer muscular VSD closure device has gained worldwide acceptance since it was first used and there have been several reports describing the effectiveness and safety of this device[8,11-15]. However, this device is not suitable for closure of perimembranous VSD due to close proximity of this type of defect to aortic valves[11]. After the development and use of Amplatzer perimembranous VSD closure device, the closure rate of perimembranous VSDs has increased. This device has a special design that prevents aortic valve damage. Since its first use, this device has emerged as having a very high implantation rate and a very low acute complication rate[15]. In our patient group, we successfully used this device in 97% of patients. One patient was abandoned because of sheath kinking. In one patient, VSD was crossed from the RV, which was very useful regarding procedure time, and arteriovenous loop creation was not needed.

Amplatzer PMVSD device has a unique design for closure of these defects, but due to the close proximity of the conduction system and perimembranous VSD, atrioventricular conduction problem may occur in a significant number of patients[16,17]. Atrioventricular conduction problems may occur during the procedure, in the immediate period after closure, or late during follow-up[18-20]). The block may occur after a very straightforward procedure. Occurrence of intraprocedural rhythm

disturbance or difficulty in crossing the VSD with long sheath may forewarn the increased possibility of heart block. The mechanism of conduction problem is not known exactly, but edema or mechanical rubbing of device retention discs are among the suggested theories[18,20]. The resolution of atrioventricular block after treatment with high-dose corticosteroids suggests edema as the main mechanism[18]. The most dreadful complication regarding the conduction system is late atrioventricular block[19]. In one large study, left anterior hemiblock and incomplete right bundle branch block were observed in 4% and 3.7%, respectively. There was early complete atrioventricular block in 1.1% of patients. In these two patients, complete AV block was resolved after one and two months with residual left anterior hemiblock[16]. In a multicenter study, complete AV block, which was treated by pacemaker implantation, was observed in one patient (2.5%)[13]. In our study group, we observed complete right bundle branch block in one patient (4%) during the procedure that persisted after six months of follow-up without sign of second or complete atrioventricular block. We did not see advanced atrioventricular block in the study group, which was evaluated by serial 24-hour ambulatory ECG monitoring at every follow-up exam.

The morphology of perimembranous VSD has major importance on closure rates. The closure rate was found as 100% in patients with single VSD and as 98% in patients with single hole with aneurysmatic VSD at follow-up. However, closure rate was approximately 89% in patients with aneurysmal VSD with multiple holes. In our study group, one patient with aneurysmal VSD with multiple holes had mild residual shunt at eight months of follow-up. The presence of aneurysm with single VSD is a very good anatomy for device closure, since the left disc and aortic valve apparatus are not in close proximity to each other.

We observed moderate to severe tricuspid regurgitation in one patient with perimembranous aneurysmal VSD after six months. The development of tricuspid regurgitation is an important concern for device closure of PMVSD. This might be related to entrapment of the tricuspid valve by the device. This complication may be prevented by careful entry to the RV using a balloon catheter[15]. Before the device release, a thorough echocardiographic assessment of the tricuspid valve is mandatory[15,21,22].

In patients with superiorly located muscular VSD, one should be very attentive regarding tricuspid valve apparatus and device interaction. In our study group, we observed one temporary tricuspid valve incompetence, which disappeared in 24 hours. This finding may be explained by tricuspid valve septal leaflet chorda trapping by the right retention disc. Surgery was performed in one patient with superiorly located muscular VSD in our study group. There was a large residual shunt and moderate tricuspid regurgitation in this patient. This may be related to malposition of the right retention disc of the device due to tricuspid valve chorda entrapment. We were unable to overcome this malposition during the procedure. After that patient, we used balloon catheters to pass the tricuspid valve to reach the RV and pulmonary artery for snaring the noodle wire.

Small infants with large VSD should be treated by hybrid procedures in the operating room[23-27]. We treated one infant with large midmuscular VSD by this method, and the child recovered uneventfully.

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