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Original Article

Transcatheter device closure of postmyocardial infarction ventricular septal defect

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Abstract

Background: Transcatheter device closure of postmyocardial infarction ventricular septal defect (PMIVSD) is less invasive than surgical repair. However, its feasibility, timing, outcome, and prognostic factors remain unclear.
Methods: This was a multicenter, retrospective cohort study. Between February 2012 and July 2015, a total of 10 (8 male and 2 female) patients with PMIVSD undergoing attempted device closure were enrolled retrospectively. The procedures were performed under general anesthesia with fluoroscopic and transesophageal echocardiographic guidance.
Results: The patients enrolled in the study were in the age range 50−85 years (median age of 76.5 years). The interval from infarction to device closure ranged from 6−147 days, with the median of 12 days. A total of 13 devices were implanted in 10 patients. There were five Amplatzer muscular ventricular septal defect occluders, four Amplatzer septal occluders, three Amplatzer PMIVSD occluders and one Amplatzer vascular plug II. Complications included transient ventricular tachycardia in three patients, device embolization in one patient, and tracheal bleeding in one patient. No procedure-related death, stroke, or cardiac tamponade was noted. During follow-up, two patients died of heart failure and two patients died of sepsis. Overall, subjects with age $\geq$ 80 years, systolic blood pressure $\leq$ 90 mmHg, and procedure time $\geq$180 minutes were significant predictor factors for mortality. All patients with the interval of infarction to device closure >12 days survived.
Conclusion: Our findings indicate that transcatheter device closure of PMIVSD is technically feasible, safe, and effective to reduce the shunt. The crucial prognostic factors were ascertained to be age $\geq$ 80 years, systolic blood pressure $\leq$ 90 mmHg, and procedure time $\geq$180 minutes.

Keywords: acute myocardial infarction; cardiac catheterization; transcatheter closure; ventricular septal defect

1. Introduction

Post myocardial infarction ventricular septal defect (PMIVSD) usually occurs within the 1st week following infarction, with an incidence of 0.2−0.34% since the advent of reperfusion therapy.[1,2] If the defect remains unrepaired, it has a high fatality rate of more than 90%.[1] Surgical repair is suggested by using current guidelines to avoid abrupt cardiovascular collapse; however, such intervention still possesses a

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high mortality rate of 20–87%.[1,3–7] Furthermore, cardiac surgeons typically prefer to wait at least 2–4 weeks for the firm scar to form over the margins of defect, which allows for better anchoring of suture and patch material.[2,8] Many patients die during this waiting period prior to surgery. Transcatheter device closure of PMIVSD is less invasive and can decrease the mortality rate to 14.3–42%.[8–13] However, its feasibility, timing, outcome, and prognostic factors remain unclear.

2. Methods

2.1. Study design

This is a multicenter, retrospective, cohort study and conducted in accordance with the Declaration of Helsinki and ethic regulation in our hospital.[14] From February 2012 to July 2015, a total of 10 patients (8 males and 2 females) with PMIVSD undergoing attempted device closure were enrolled retrospectively. Their age range was 50–85 years, with the median of 76.5 years. The interval from infarction to VSD found ranged from 2 to 146 days, with the median of 7.5 days. The interval from infarction to device closure ranged from 6 to 147 days, with the median of 12 days. The demographic data of these patients is summarized in Table 1. Vasoactive–inotropic score was calculated for the total equivalent dose of inotrope including dopamine, dobutamine, epinephrine, norepinephrine, and milrinone.[15,16] The Model of End-Stage Liver Disease Excluding International Normalized Ratior (MELD-XI) score was calculated using creatinine and total bilirubin according to the following formula: $5.11 \times \ln$ (bilirubin mg/dL) + $11.76 \times \ln$ (creatinine mg/dL) + 9.44, as an index of multiorgan system dysfunction.[17]

2.2. Procedure

Informed consent was obtained from all patients or their family members. The device closure procedure was described in a previous report and briefly as related below. Cefazolin (1 g) was given to the patients as a prophylatic antibiotic.[18,19] Vascular access was obtained from the right internal jugular vein and the right femoral artery. We performed the procedure under general anesthesia, with fluoroscopic and transesophageal echocardiographic guidance. Routine right and left heart catheterizations were done for the evaluation of pulmonary to system flow ratio (Qp/Qs). A Judkins right catheter was advanced retrogradely to cross the VSD. A 0.035-inch glide wire was advanced through the Judkins catheter into the pulmonary artery or superior vena cava, which was captured with a snare catheter through the jugular vein to establish an arteriovenous loop. A 24- or 34-mm compliant low-pressure sizing balloon (St. Jude Medical) was used to measure the stretched size of VSD and was subsequently exchanged for an appropriate sized delivery sheath. The size selection of deployed device was about 1.5–2 times the size of the stretched balloon. Ultimately, the device selection was based on the size of the device. The priority was the Amplatzer muscular VSD occluder (up to 18 mm), Amplatzer PMIVSD occluder (up to 24 mm, available since November 2013), and then atrial septal occluder (up to 40 mm, if bigger device needed). The Amplatzer vascular plug II was used if the defect was the long-tunnel type. If the left ventriculogram fully opacified the right ventricle after the 1st device implantation, indicating a large residual shunt, we would subsequently try to deploy a second device. After the procedure, oral aspirin, 100 mg daily, was prescribed for at least 6 months.

Table 1
Patient demographics before procedure.

No.	Age (y)	Sex	Occluded coronary artery	Infarction territory (wall)	Previous surgery	VIS score	IABP	ECMO	BP (S/D/M) (mmHg)	Interval AMI to VSD found (d)	Interval AMI to device closure (d)	NT-proBNP (pg/mL)	MELD-XI score
1	76	M	RCA	Inferior	VSD repair	14.8	+	—	112/62/75	9	75	NA	10.7
2	50	M	LAD	Anterior	CABG→ VSD repair	—	—	—	101/78/90	6	79	9290	7.8
3	80	M	LAD	Anterior	VSD repair	NA	+	—	90/41/60	3	10	NA	29.2
4	61	M	LAD	Anterior	—	60.6	+	—	103/63/75	71	80	> 35000	35.1
5	77	M	LAD	Anterior	—	—	—	—	104/67/83	4	10	5120	15.5
6	71	M	RCA	Inferior	CABG+ Mitral repair	97.6	+	+	74/50/56	9	12	168	35.4
7	85	F	LAD + RCA	Anterior	—	5.1	+	—	86/35/50	2	8	NA	29.3
8	80	F	LAD	Anterior	—	9.1	—	—	82/45/59	4	6	22900	19.1
9	70	M	LAD + LCX	Anterior	—	6.5	+	—	98/44/66	9	12	2180	8.8
10	77	M	RCA + LCX	Inferior	—	—	—	—	127/75/92	146	147	13101	24.8

AMI = acute myocardial infarction; BP (S/D/M) = blood pressure (systolic/diastolic/mean); CABG = coronary artery bypass grafting; ECMO = extracorporeal membrane oxygenation; F = female; IABP = intra-aortic balloon pump; Interval = interval from AMI to device; LAD = left anterior descending artery; LCX = left circumflex artery; M = male; MELD-XI = Model for End-stage Liver Disease Excluding International normalized ratio; NA = not available; NT-proBNP = N-terminal of the prohormone brain natriuretic peptide; PCI = percutaneous coronary intervention; RCA = right coronary artery; Time = time after acutemyocardial infarction; VIS = vasoactive–inotrope score.

2.3. Statistics

We stratified each predictor factor into two groups and used Fisher's exact test to correlate them with mortality.

3. Results

The results are summarized in Table 2. A total of 13 devices were successfully implanted in 10 patients (Fig. 1), and 3 patients received two devices (Fig. 2). There were five Amplatzer muscular ventricular septal defect occluders, four Amplatzer septal occluders, three Amplatzer PMIVSD occluders, and one Amplatzer vascular plug II. Three patients experienced transient ventricular tachycardia when the sheath crossed the VSD. In Patient 7, the 2nd device (16-mm Amplatzer muscular VSD occluder) was embolized into the pulmonary artery, which was successfully retrieved and replaced with a 30-mm Amplatzer septal occluder. Patient 2 had a large residual shunt with a sign of heart failure and underwent surgical repair 38 days later. Patient 1 exhibited bleeding from the endotracheal tube which was resolved by reversing the anticoagulation effect of heparin by protamine. There was no cardiac tamponade or stroke. During follow-up, two patients died of heart failure and another two died of pneumonia-associated sepsis. There was no late complication after the procedure. Five patients had good prognosis with New York Heart Association functional class II. Prediction of mortality of different factors is summarized in Table 3.

4. Discussion

The main findings of our study include the following. First, transcatheter device closure of PMIVSD is technically feasible. All patients received device implantation successfully. Second, the device closure procedure is safe without major complications of procedure-related death, stroke, or cardiac tamponade. Third, only one patient had a large residual shunt, thereby proving that the procedure can effectively reduce the shunt. Fourth, all patients with systolic blood pressure ≦ 90 mmHg prior to procedure died. All patients with systolic blood pressure > 90 mmHg survived. It indicates that blood pressure is a crucial prognostic factor ($p = 0.005$). Fifth, all patients with the interval of infarction to device closure >12 days survived. However, for patients with an interval ≦12 days, the mortality rate was high (67%). This indicates that intervention at the acute stage carries a high risk. Some cardiologists suggest that it is necessary to delay device closure for > 2 weeks after infarction.[20] In our opinion, this does not mean that early intervention should be avoided because device closure could be a salvage procedure for acute critical patients. Sixth, patients aged ≥ 80 years have a higher mortality rate than those aged < 80 years (100% vs. 14%, $p = 0.033$). Seventh, procedure time ≥ 180 minutes had a higher mortality rate than procedure times < 180 minutes (75% vs. 20%, $p = 0.048$).

Most prognostic factors are not statistically significant probably because the sample size was quite small. However, the mortality rate of MELD-XI score ≥ 20 was 60% and < 20 was 20%. The result was similar to those in Assenza et al, indicating that the mortality rate of MELD-XI score ≥ 20 was 62%.[13]

PMIVSDs are often serpiginous and multiple, and balloon sizing can determine the exact size and shape of VSD, which are important for device selection and deployment.[11] About half (26/53) of the patients in the UK registry received balloon sizing.[11] We prefer to use balloon sizing to measure the accurate size of VSD; a compliant low-pressure balloon is recommended to avoid an enlarged VSD. The size selection of the deployed device could be 1.5 times the stretched balloon size in subacute stage. However, it should be at least 1.5−2 times in the acute stage due to the fragile injured myocardium.

Table 2
Results of transcatheter closure of PMIVSD.

No.	Qp/Qs	VSD number	VSD size (mm)	Device type and size (mm)	Residual shunt[a]	Fluoroscopy time (min)	Procedure time (min)	Complication	ICU days	Outcome
1	2.50	1	8.0	MVSDO (12)	Small	25.2	92	Tracheal bleeding	15	Survival (NYHA class II)
2	2.95	2	4.2	MVSDO (6)	Large	168.8	302	no	3	Survival (NYHA class II)
3	2.08	2	17.1, 10.7	ASO (24), VPII (20)	Small	NA	NA	no	15	Died of heart failure at Day 15
4	3.57	1	16.1	ASO (24)	Moderate	42.8	140	VT	28	Survival (NYHA class III)
5	4.67	2	10.8, 10.7	MVSDO (18, 16)	Small	40.3	156	VT	3	Survival (NYHA class II)
6	2.53	1	26.4	ASO (38)	Absent	30.5	189	no	16	Died of heart failure at Day 16
7	1.71	3	5.8, 16.8	MVSDO (12), ASO (30)	Small	138	355	VT, device embolization	11	Died of sepsis at Day 11
8	4.09	1	8.6	MVSDO (16)→ PMIVSDO (24)	Small	49.9	200	no	8	Died of sepsis at Day 8
9	2.30	1	15.2	PMIVSDO (24)	Small	31.1	75	no	10	Survival (NYHA class I)
10	3.4	1	16.2	PMIVSDO (24)	Small	29.9	70	no	4	Survival (NYHA class II)

ASO = Amplatzer septaloccluder; ICU = intensive care unit; MVSDO = muscular ventricular septal defect occluder; NA = not available; NYHA = New York Heart Association; Qp/Qs = pulmonary to systemic flow ratio; PMIVSDO = postmyocardial infarction ventricular septal defect occluder; VPII = 2nd generation of vascular plug; VSD = ventricular septal defect; VT = ventricular tachycardia.

[a] Residual shunt measured by color Doppler echocardiography: small = < 2 mm; moderate = 2−4 mm; large = > 4 mm.

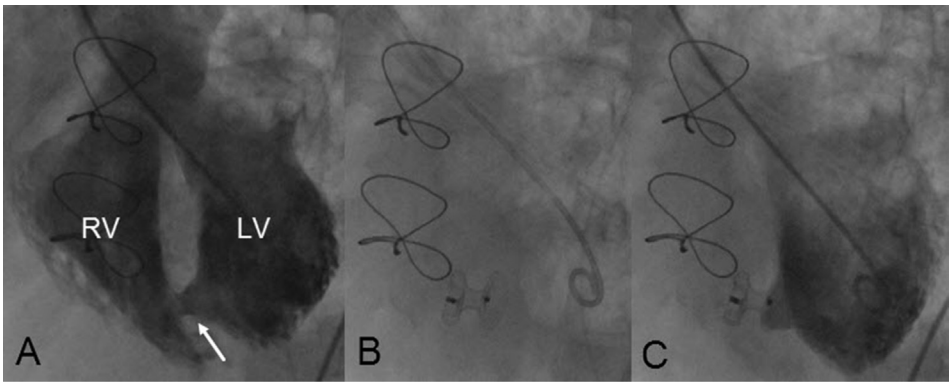

Fig. 1. (A) Left ventriculogram in patient 1 showing an 8-mm PMIVSD (arrow); (B) a 12-mm Amplatzer muscular VSD occluder was implanted; (C) left ventriculogram after device implantation showing only small residual shunt. LV = left ventriculogram; RV = right ventriculogram.

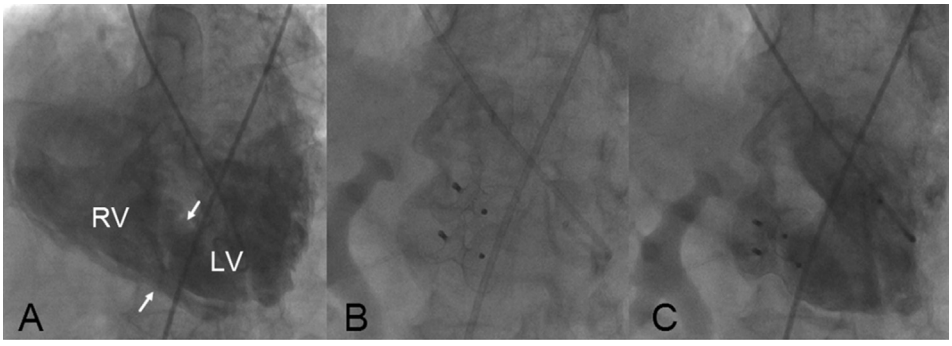

Fig. 2. (A) Left ventriculogram in Patient 5 showing two PMIVSDs (arrow); (B) Two Amplatzer muscular VSD occluders were implanted; (C) left ventriculogram after device implantation showing only small residual shunt. LV = left ventriculogram; RV = right ventriculogram.

This study does have certain limitations. First, it was retrospective in nature, with a small sample size. With the progression and broader availability of percutaneous coronary intervention, the incidence of PMIVSD is now quite rare. In fact, it is difficult to perform a randomized comparison with surgical repair.

In conclusion, according to our experience in this study, transcatheter device closure of PMIVSD is technically feasible, safe, and effective to reduce the shunt. We found that patients with age $\geq$ 80 years, systolic blood pressure $\leq$ 90 mmHg, and procedure time $\geq$180 minutes were significant predictor factors for mortality.

Table 3
Prediction of mortality in device closure of PMIVSD.

Factor		Mortality rate	*p*
Age (y)	< 80 : ≥ 80	14% :100%	0.033
Sex	M : F	25% : 100%	0.133
Infarction territory (wall)	Anterior : Inferior	43% : 33%	1.000
Previous surgical repair of VSD	Yes : no	33% : 43%	1.000
VIS score	<15 : ≥15	29% : 50%	1.000
IABP	Yes : no	50% : 25%	0.571
ECMO	Yes : no	100% : 33%	0.400
Systolic BP (mmHg)	≤ 90 : > 90	100% : 14%	0.005
Interval between AMI to device closure (d)	≤ 12 : > 12	67% : 0	0.076
NT-proBNP (pg/mL)	< 20000 : ≥ 20000	20% : 50%	1.000
MELD-XI score	< 20 : ≥ 20	20% : 60%	0.524
VSD number	1 : ≥ 2	33% : 50%	1.000
Maximum VSD size (mm)	< 17 : ≥ 17	25% : 100%	0.133
Residual shunt	Absent to Small : Moderate to large	50% : 0	0.467
Procedure time (min)	< 180 : ≥ 180	20% : 75%	0.048

AMI = acute myocardial infarction; BP = blood pressure; ECMO = extracorporeal membrane oxygenation; F = female; IABP = intra-aortic balloon pump; M = male; MELD-XI = Model for End-stage Liver Disease Excluding International normalized ratio; NT-proBNP = N-terminal of the prohormone brain natriuretic peptide; PMIVSD = postmyocardial infarction ventricular septal defect; VIS = vasoactive–inotrope score; VSD = ventricular septal defect.

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