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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

Conflicts of interest: The authors declare that they have conflicts of interest related to the subject matter or materials discussed in this article.

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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.