Case Reports

Transcatheter Closure of Iatrogenic VSDs after Aortic Valve Replacement Surgery:

2 Case Reports and a Literature Review

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We report 2 new cases of transcatheter closure of iatrogenic ventricular septal defects after aortic valve replacement surgery, together with our finding, in a literature review, of 9 additional patients who had undergone this procedure from 2004 through 2013. In all 11 cases, transcatheter device closure was indicated for a substantial intracardiac shunt with symptomatic heart failure, and such a device was successfully deployed across the iatrogenic ventricular septal defect, with clinical improvement.

Our review suggests that transcatheter closure of iatrogenic ventricular septal defects in patients with previous aortic valve replacement surgery is a safe and effective treatment option, providing anatomic defect closure and relief of symptoms in the short-to-medium term. ***(Tex Heart Inst J 2016;43(4):329-3)***

Iatrogenic ventricular septal defect (VSD), a rare sequela of aortic valve replacement (AVR) surgery,[1] is caused by accidental incision of the ventricular septum during dissection of the aortic valve. Patients might present in the postoperative period with breathlessness, reduced exercise tolerance, and signs of heart failure. Additional cardiac surgery to repair the VSD can cause substantial morbidity and death, but transcatheter device closure offers the possibility of a less invasive therapeutic option. We report 2 new cases of transcatheter closure of iatrogenic VSD and review the published literature on this procedure.

Key words: *Aortic valve insufficiency/replacement/surgery; catheterization; echocardiography, transesophageal; heart septal defects, ventricular/therapy; iatrogenic disease; septal occluder device; ventricular septum/injuries*

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Case Reports

Patient 1

A 70-year-old man presented with exertional dyspnea and a history of native aortic valve replacement (AVR) with a 23-mm Sorin MITROFLOW® bioprosthetic aortic valve (LivaNova PLC; London, UK), together with coronary artery bypass grafting 11 years prior. Because a transesophageal echocardiogram (TEE) revealed severe aortic regurgitation, he underwent repeat AVR surgery with use of a 23-mm Epic™ bioprosthesis (St. Jude Medical, Inc.; St. Paul, Minn).

The surgery was complicated by complete heart block and the consequent implantation of a dual-chamber pacemaker. A routine postoperative transthoracic echocardiogram (TTE) revealed a subaortic VSD estimated to be 5 mm in diameter, which was managed conservatively. The patient presented one month after repeat AVR surgery with breathlessness and peripheral edema. A TEE confirmed a subaortic VSD, now estimated to be 12 mm in diameter. Left ventricular (LV) systolic function was mildly impaired, but right ventricular dimensions and systolic function were normal.

The VSD closure was carried out with the aid of TEE and fluoroscopic guidance, and with the patient under general anesthesia. The VSD was crossed from the LV with a 260-cm Radifocus® hydrophilic guidewire (Terumo Medical Corporation; Somerset, NJ). The guidewire was advanced into the pulmonary artery, snared, and externalized via the right femoral vein to create an arteriovenous loop (Fig. 1). A 7F AMPLATZER™ TorqVue™ delivery sheath (St. Jude Medical) was advanced via the right femoral vein, and a 12-mm AMPLATZER™ Muscular VSD Occluder (St. Jude Medical) was successfully deployed across the VSD. An intraoperative TEE revealed only

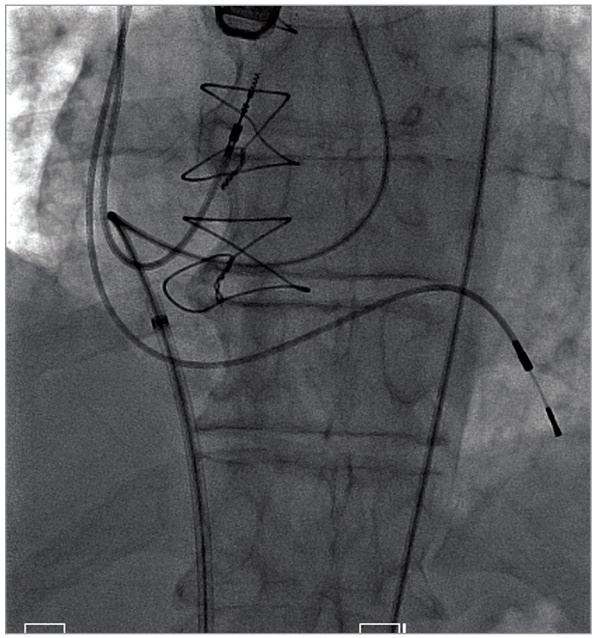

Fig. 1 *Patient 1. Fluoroscopic image shows the creation of the arteriovenous loop across the ventricular septal defect.*

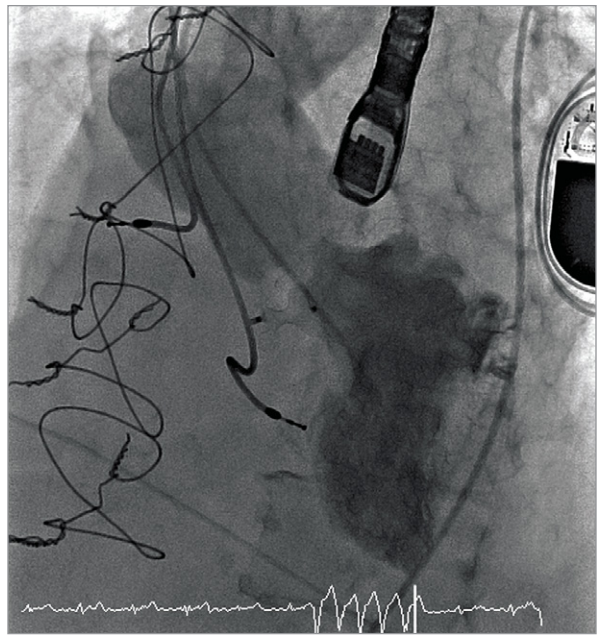

Fig. 2 *Patient 2. Final left ventriculogram shows no residual shunting.*

a minor residual shunt through the VSD. One month after closure of the VSD, our patient's breathlessness had improved. Subsequent echocardiography revealed the persistence of a minor interventricular shunt.

Patient 2

An 80-year-old woman with a bioprosthetic AVR presented with breathlessness secondary to concomitant severe mitral regurgitation. Right- and left-sided heart catheterization results confirmed a peak pulmonary artery pressure of 60 mmHg, and aortograms revealed a dilated ascending aorta (55 mm). She underwent mitral and aortic valve replacements with 29-mm and 23-mm Epic porcine bioprosthetic valves, respectively, and the aortic root was replaced with a 34-mm Dacron graft. Her recovery was complicated by complete heart block, and she underwent dual-chamber pacemaker implantation. She was discharged from the hospital but presented one month after surgery with clinical signs of congestive cardiac failure. A TTE revealed a VSD below the aortic valve.

Transcatheter closure of the VSD was carried out, as in the case above, with the aid of TEE and fluoroscopic guidance, and with the patient under general anesthesia. A TEE estimate of the VSD's diameter was 8 mm. The VSD was then crossed from the LV with a 260-cm Radifocus hydrophilic guidewire, which was advanced into the pulmonary artery. The wire was snared and externalized to create an arteriovenous loop. The Terumo wire was exchanged for a 300-cm AMPLATZER Noodle wire (St. Jude Medical), and an 8-mm AMPLATZER muscular VSD occluder was then deployed across the VSD, with a final good echocardiographic position. Repeat left ventriculography revealed no residual flow across the defect (Fig. 2).

After this procedure, the patient's breathlessness and peripheral edema improved. An echocardiogram 28 months after closure of the VSD showed no residual interventricular shunting.

Literature Review

We searched MEDLINE®, PubMed®, and Embase®, using (in multiple combinations) the terms "aortic, defect(s), iatrogenic, ventricular, septal, acquired" in a title search. The search yielded 1,059 articles, 8 of which we considered relevant. We identified reports of transcatheter closure of iatrogenic VSD after AVR in 9 patients (Table I).[2-8] A case series of 29 patients included 4 patients with iatrogenic VSD after AVR surgery, but this paper was excluded because data on individual patients were not reported.[1] There was a conference report of an additional 2 patients who had undergone transcatheter closure (via a transapical route) of iatrogenic VSD after an AVR, but these cases were also excluded because individual patient data were not available.[9]

Table I shows the symptoms and presenting diagnoses. The age range was 40 to 86 years. At the time of VSD closure, 6 patients had mechanical AVRs, and 5 had bioprosthetic. In 9 cases, the aortic valve was crossed retrogradely, and the VSD was crossed from the LV to the right ventricle. In 2 cases with mechanical AVRs, access to the LV was attained via puncture of the interatrial septum—after a failed transaortic ap-

TABLE I. Reports of Transcatheter Closure of Iatrogenic Ventricular Septal Defects after Aortic Valve Replacement in 11 Patients

Reference	Age (yr)	Symptoms/ Diagnoses	Valve Type		Closure Approach	Defect Size (mm)	Device Size (mm)	PPM Insertion	Complete Closure	Complications of VSD Closure	Length of Follow-Up	Findings at Follow-Up
			Initial AVR	Repeat AVR								
Holzer R, et al.[2] (2004)	40	Dyspnea and fatigue	Mech AVR	Mech AVR	TA	4–5	8	None	Yes	None	6 mo	Reduced LVEDD, increased fractional shortening, and no residual defect
	57	Dyspnea	Mech AVR	Mech AVR	TA	7	12	After AVR	Yes	None	1 mo	Reduced LVEDD with no residual defect
Klein AJ, et al.[3] (2007)	40	Dyspnea, CHF: reduced functional capacity	Bio AVR	Mech AVR	TS	8	10	After AVR	Yes	None	6 mo	Improved functional capacity
Dodos F, et al.[4] (2008)	64	Dyspnea	Mech AVR	NA	TA	8.5	10	After AVR	No (small residual shunt self-resolved)	Hemoptysis	NR	Reduced dyspnea
Chojnicki M, et al.[5] (2008)	70	Reduced exercise tolerance	Mech AVR	Bio AVR	TA	10	12	After AVR	Yes	None	1 yr	Improved functional class
Noble S and Ibrahim R[6] (2009)	69	CHF	Mech AVR and MVR	Mech AVR and MVR	TA	7	10	None	Yes	Periprocedural severe AR and hypotension	10 mo	Improved functional class; no cardiac events
Coceani M, et al.[7] (2012)	73	CHF	Bio AVR	NA	TA	NR	7	After VSD closure	Minimal residual shunt	Complete heart block	NR	NR
Matsumoto T, et al.[8] (2013)	72	Dyspnea	NR	Mech AVR	TS (TA failed)	9	14	After AVR	Yes	Periprocedural severe AR and hypotension	3 yr	Asymptomatic
	86	Fatigue and dyspnea	Bio AVR	NA	TA	4	6	After VSD closure	Yes	Complete heart block	3 yr	Asymptomatic
Our Patient 1	70	Dyspnea and peripheral edema	Bio AVR	Bio AVR	TA	12	12	After AVR	Minor residual shunt	None	1 mo	Reduced dyspnea and peripheral edema
Our Patient 2	80	Dyspnea	Bio AVR	Bio AVR and MVR	TA	8	8	After AVR	Yes	None	18 mo	Reduced dyspnea and peripheral edema

AR = aortic regurgitation; AVR = aortic valve replacement; Bio = bioprosthetic; CHF = congestive heart failure; LVEDD = left ventricular end-diastolic diameter; Mech = mechanical; MVR = mitral valve replacement; NA = not applicable; NR = not reported; PPM = permanent pacemaker; TA = transaortic; TS = transseptal; VSD = ventricular septal defect

proach, in one instance.[8] All procedures used an arteriovenous loop and delivered the VSD occluder from the right side. The diameters of the VSDs varied from 4 to 12 mm. All VSDs were closed with an AMPLATZER VSD occluder, in sizes ranging from 6 to 14 mm.

Sequelae included intraprocedural hypotension, transient severe aortic regurgitation related to the interference of equipment with a mechanical aortic valve, and complete heart block (2 cases) that entailed permanent pacemaker insertion.[3,7] All patients survived and were discharged from the hospital. No late sequelae were documented. Follow-up information was available for 10 patients and ranged in duration from 1 month to 3 years. Symptomatic improvement (reduced breathlessness and peripheral edema, and increased functional capacity) was documented in 8 of 10 patients; the other 2 patients had echocardiographic reduction in LV end-diastolic diameter. In 8 patients, the procedure resulted in complete VSD occlusion; 2 others had a minor residual shunt, and 1 of these closed spontaneously.

Discussion

Iatrogenic VSDs are a rare sequela of AVR surgery. When they do occur, they typically are located in the membranous region of the interventricular septum. The VSD tends to be relatively small and is thought to be caused during dissection of the aortic valve. In 8 of the 11 patients in this series, the VSD was detected after a repeat AVR operation. We postulate that the risk of iatrogenic VSD is higher during repeat AVR surgery because of scar tissue and the requirement to dissect beyond the suture line. This emphasizes the need to pay special attention to the interventricular septum during repeat surgery.

Intraoperative TEE can help to minimize the risk of septal injury and to facilitate earlier diagnosis and management of iatrogenic septal defects. Intraoperative TEE was not often used in our institution at the time of our reported surgical AVR procedures, but this practice has now become routine. In the future, color-flow mapping should also enable perioperative detection of iatrogenic VSD. Detection of septal injury in the absence of septal perforation might be more difficult. Indeed it is possible that in some patients the iatrogenic communication between the ventricles develops only during the postoperative period.

Transseptal, transaortic, and transapical[2] approaches for transcatheter closure of iatrogenic VSD have been used successfully after the placement of either metallic or bioprosthetic AVRs. The use of the transaortic approach through a bileaflet metallic valve has been described,[2] but in monodisc valves that approach is hazardous because of the risks of catheter entrapment and embolization of the valve disc.

As we have observed, the incidence rate associated with transcatheter closure of iatrogenic VSD appears to be low, but periprocedural sequelae have been reported. In particular, intravascular guidewires and catheters can interfere with prosthetic aortic valve function, causing severe aortic regurgitation and hypotension.[6,8] The procedure can be associated with new complete heart block,[8] and in this review we found that 8 of the 11 patients needed permanent pacemaker insertion as a sequela to aortic valve surgery or transcatheter VSD closure. This could arise from trauma during aortic valve surgery or from compression of the conducting tissues by the VSD occluder. The rate of permanent pacemaker implantation in our review series is high, which reflects the fact that we selected patients who had complex cardiac disease and sequelae of surgical AVR.

Conclusion. Iatrogenic VSD should be suspected in patients who continue to be symptomatic after aortic valve surgery. The diagnosis can usually be confirmed by means of TTE. Our review suggests that transcatheter closure of iatrogenic VSD is a feasible alternative to further cardiac surgery. In this small series, the risks of transcatheter closure were low, but these patients would have been at relatively high risk during repeat surgery. We suggest that transcatheter closure should be the preferred treatment option for patients with symptoms attributed to iatrogenic VSD after aortic valve replacement.

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