Case Reports

Percutaneous Repair of Post-Myocardial Infarction Ventricular Septal Defect:

Current Approaches and Future Perspectives

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Post-myocardial infarction ventricular septal defect is a devastating complication of ST-elevation myocardial infarction. Although surgical intervention is considered the gold standard for treatment, it carries high morbidity and mortality rates. We present 2 cases that illustrate the application of percutaneous closure of a post-myocardial infarction ventricular septal defect: the first in a patient who had undergone prior surgical closure and then developed a new shunt, and the second as a bridge to definitive surgery in a critically ill patient.
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The occurrence of a ventricular septal defect (VSD) after a myocardial infarction (MI) is an infrequent but serious sequela, which usually occurs within the first week.[1] In the years before reperfusion became available, the incidence of VSD after an MI was between 1% and 2%, with an in-hospital mortality rate of 45% with surgery, and 90% with medical management alone.[1-4] After the introduction of reperfusion therapy, the rate of post-MI VSD decreased to 0.2% to 0.34%.[4,5] However, mortality rates remain high after surgical intervention, ranging from 20% to 87%, depending on severity in the individual patient and on length of follow-up.[5-11]

Because surgery offers a better outcome than medical management alone, immediate surgical intervention is now a class I recommendation for post-MI VSD.[12,13] However, early surgical repair can be difficult because of the soft and friable tissue surrounding the area of infarction and the possibility of VSD expansion.[14-16] In addition, a residual shunt persists in 10% to 37% of patients despite surgical repair; 11% of those residual defects need further surgical procedures.[7] An approach that would enable immediate hemodynamic stabilization and closure of the defect is desirable. Although transcatheter closure of a post-MI VSD is relatively new, investigators have shown that this procedure is well suited for treating residual shunts after surgical closure and for stabilizing critically ill patients as a bridge to future surgery.[17-23] We report our experience with 2 cases of the percutaneous closure of post-MI VSDs, which illustrate these principles.

Case Reports

Patient 1

Transcatheter Closure of Recurrent Shunt after Surgical Repair. A 65-year-old man presented at our institution's emergency department with chest pain and shortness of breath. On physical examination, the patient exhibited signs of congestive heart failure, together with a grade 3/6 harsh holosystolic murmur. The patient was hypotensive, his cardiac enzymes were elevated, and electrocardiographic findings showed ST-elevation in the anterior leads. An echocardiogram revealed a VSD, an akinetic anterior wall, and a hyperdynamic posterior wall. Cardiac catheterization showed critical left main and multivessel coronary artery disease. The patient's pulmonary artery (PA) pressure was 42/22 mmHg, there was an oxygen elevation in the right ventricle (RV), and his PA oxygen saturation was 79%. The Qp/Qs was 2.2:1.

The patient underwent successful coronary artery bypass grafting (CABG), together with Dacron-graft repair of the VSD. One week postoperatively, he developed increasing dyspnea and had a recurrent holosystolic murmur. He was found on echocardiography to have a residual VSD (Fig. 1). Repeat angiography showed 2 defects,

localized to the mid and apical septum; the Qp/Qs shunt ratio was again 2.1:1. Because the patient was at high risk for a 2nd cardiac surgery, percutaneous closure was recommended.

With the patient under general anesthesia, we obtained bilateral femoral arterial and venous access and placed an intra-aortic balloon pump (IABP) prophylactically. Ventriculography performed in the left anterior oblique projection showed 2 separate defects (Fig. 2A). We placed a diagnostic catheter in the left ventricle (LV), and passed a wire into the PA, where it was snared and externalized to the right femoral vein, to form a continuous arteriovenous loop. The size of the defect was measured by transesophageal echocardiography (TEE) and fluoroscopy, via a sizing balloon. The VSD waist size was found to be 9 to 10 mm (Fig. 2B). We loaded an occluder device onto the delivery catheter and advanced it through the defect. A 28-mm CardioSEAL StarFlex® Septal Occluder was used, the distal portion of which was deployed on the LV side and anchored successfully by pulling back toward the RV. We deployed the proximal portion of the implant at the RV side of the interventricular septum. Repeat left ventriculography confirmed successful closure of the mid VSD, and both fluoroscopy (Fig. 2C) and TEE showed that the device was appropriately seated.

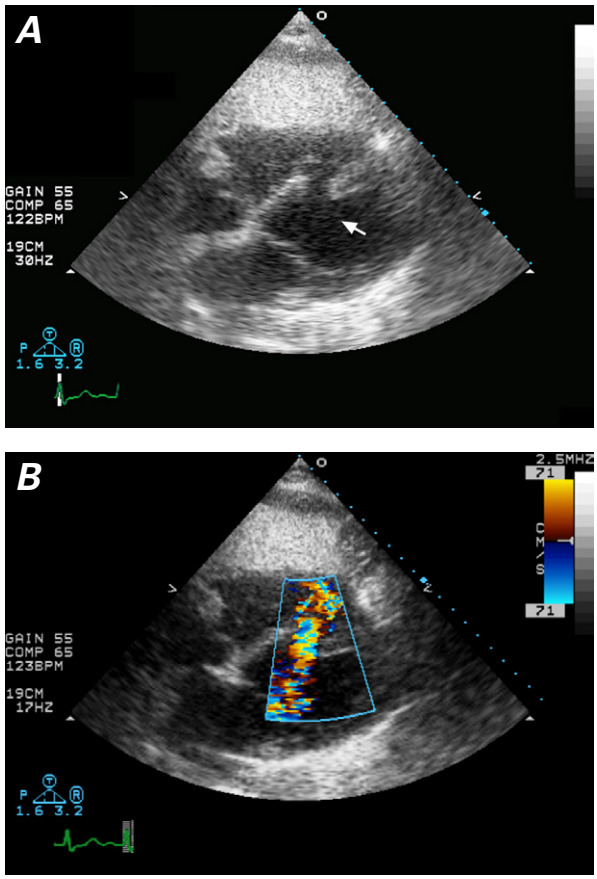

Fig. 1 *Patient 1. Transthoracic echocardiograms (subcostal view) show* ***A****) a ventricular septal defect (arrow) and* ***B****) further delineation with use of color-flow Doppler.*

The patient was stabilized and weaned from the IABP and mechanical ventilation. Over the course of the following year, the patient experienced recurrent congestive heart failure because of severe LV dysfunction, despite an intact septum. One year after septal defect closure, the patient underwent cardiac transplantation. Examination of the explanted heart showed complete sealing of the septal defect by the occluder device (Fig. 3).

Patient 2

Recurrent Shunt after Transcatheter Closure and Subsequent Surgical Repair. A 73-year-old woman was transferred from another hospital for the treatment of a VSD 4 weeks after an anterior MI. Upon cardiac catheterization, we found total occlusion of the proximal left anterior descending coronary artery (LAD) and 40% stenosis of the right coronary artery. The patient was revascularized with 2 drug-eluting stents in the proximal LAD. Left ventriculography showed an LV ejection fraction (LVEF) of 0.25, with anterolateral akinesia and apical dyskinesia. The patient had a prolonged hospital course complicated by cardiogenic shock, which required pressor support. She eventually was discharged from the hospital.

During a follow-up visit with her cardiologist approximately 3 weeks later, she was noted to have profound heart failure and a 3/6 harsh holosystolic murmur. After her admission to another hospital, her condition rapidly deteriorated, to the point that she was given inotropic support. An echocardiogram showed persistently decreased LVEF and a new VSD.

The patient was then transferred to our institution for further management of her VSD. A repeat echocardiogram showed a 14- to 16-mm apical VSD with an LVEF of 0.30 and a dysfunctional RV with moderate tricuspid regurgitation. Right-sided heart catheterization showed a PA pressure of 67/45, a PA oxygen saturation level of 77%, and a Qp/Qs ratio of 3.6. The patient needed an IABP with inotropic support to maintain hemodynamic stability. Because she was considered a high-risk surgical candidate, she underwent percutaneous closure of her VSD.

A TEE identified the defect, the diameter of which was 1.4 cm. This was followed by ventriculography. Crossing the VSD proved difficult, and multiple catheters were used. We crossed the defect from the LV and externalized a guidewire via the femoral venous sheath. A sheath was then brought into the RV from the right femoral vein and was introduced into the LV, where it was used to deliver an 18-mm AMPLATZER® Septal Occluder (St. Jude Medical, Inc.; St. Paul, Minn). Positioning of the occluder was optimized via TEE and

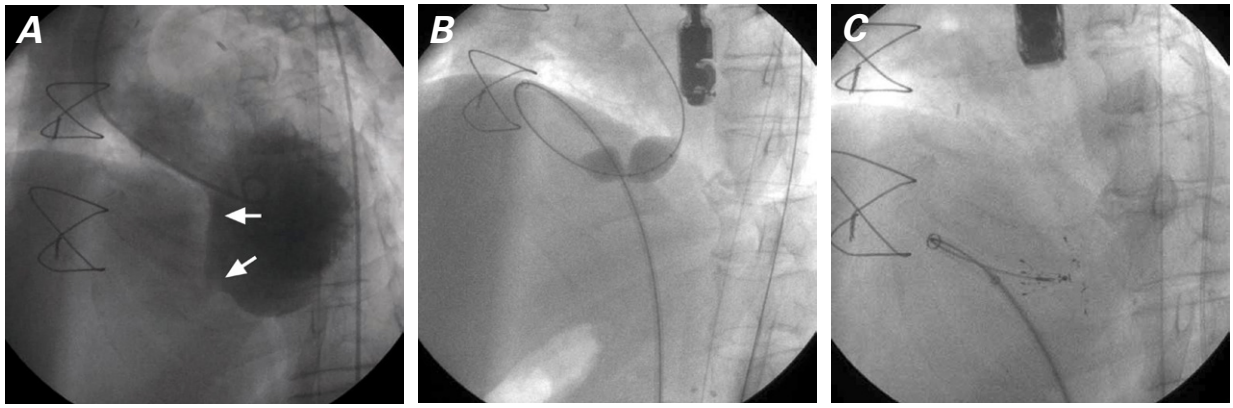

Fig. 2 *Patient 1. Coronary angiograms (left anterior oblique view) show* ***A****) 2 separate ventricular defects (arrows);* ***B****) the defect's waist size, measured via a balloon catheter; and* ***C****) the deployed septal occluder.*

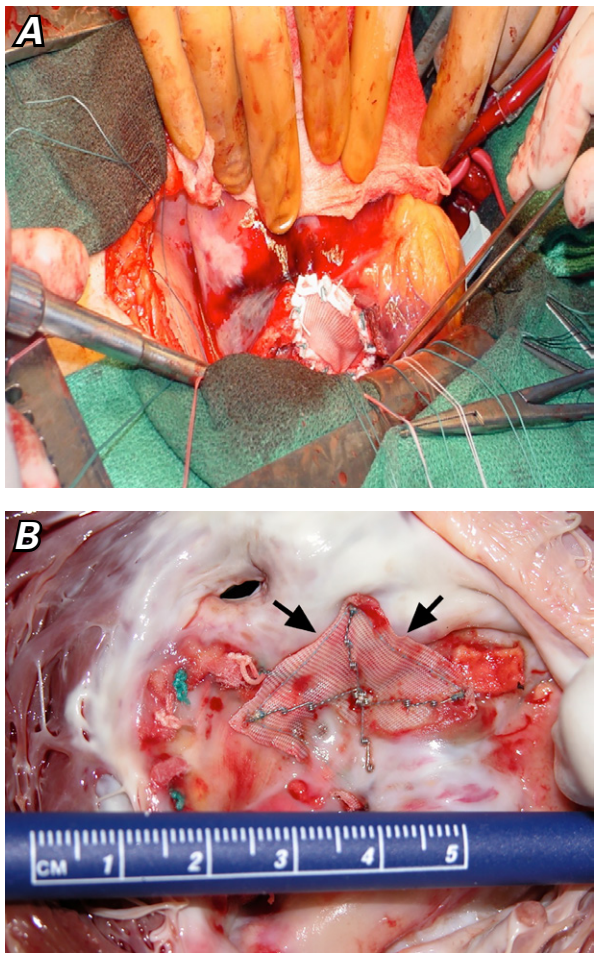

Fig. 3 *Patient 1. Intraoperative photographs show* ***A****) the explanted heart, with an intact septal occluder device; and* ***B****) a closer view of that device (arrows).*

ventriculography. We used a push-pull maneuver to confirm good seating of the device, then deployed it.

At first, the patient's clinical condition improved, but it steadily worsened and repeat imaging showed a persistent VSD. Two weeks after percutaneous closure (and 6 weeks post-MI), the patient underwent surgical repair. Intraoperatively, we found a residual VSD at the inferior border of the occluder device. We removed the AMPLATZER occluder and repaired the defect with a CorMatrix® patch (CorMatrix; Roswell, Ga). The patient tolerated the procedure and eventually was weaned from the IABP and inotropic support. She was discharged from the hospital free of angina, but her LVEF was persistently reduced, at 0.15.

Discussion

Surgical repair of a post-MI VSD is associated with a very high mortality rate.[19] The 2004 and 2007 American College of Cardiology/American Heart Association guidelines recommend emergent repair of the VSD with concurrent CABG, regardless of hemodynamic status.[12,13] Acute post-MI VSD repair is difficult because of the soft and friable myocardial tissue. It has been suggested that better results occur when the tissue edges surrounding the VSD have scarred and myocardial fibrosis has produced more favorable surgical conditions.[14-16] The improved surgical survival rate associated with delayed repair carries a significant selection bias, for it encompasses patients who have a relatively lower risk for surgery.

Within recent years, the percutaneous closure of post-MI VSDs has become a promising option in a variety of situations: to close a residual leak after VSD surgical repair, to stabilize high-risk patients for future surgery, and to serve as a primary intervention.

The application of occluder devices to recurrent post-MI VSDs has been reported in several series.[17,18,24] Recurrence of a VSD occurs in 5% to 20% of surgical repair cases.[25-27] The considerable advantage of a percutaneous approach to repeat VSD repair is that the patient does not undergo a 2nd surgery.

After an initial large study by Landzberg and Lock,[27] Holzer and colleagues[17] reported the immediate and

midterm results of a multicenter international registry of transcutaneous closure of post-infarction VSDs. The AMPLATZER muscular VSD device (St. Jude Medical) was successfully deployed in 16 of 18 patients (89%); 10 of those 18 patients (56%) had undergone a previous attempt at primary surgical closure and had experienced residual shunting. The median time between MI and transcatheter closure of the VSD was 25 days. At 24 hours after closure, the residual shunt was measured: 2 of the 16 VSDs with successful device deployment were closed (12.5%), 10 displayed trivial or small residual shunting (62.5%), and 4 displayed moderate or large shunts (25%). The 30-day mortality rate was 28%. Of the 16 patients, only 2 (12.5%) required a 2nd procedure to close a residual VSD.

Goldstein and associates[18] discussed the morbidity and death associated with surgical repair of post-MI VSDs. In their judgment, the impetus behind the development of percutaneous methods was the general reluctance of surgeons to attempt repeat surgical repair of post-MI VSDs. Several other investigators[24,28-30] have suggested the improvement in heart failure symptoms as an impetus, together with the option of a treatment alternative for critically ill patients.

We report our experience in treating Patient 1, who presented with a residual post-MI VSD one week after undergoing surgical repair of the VSD. His case illustrates the successful use of percutaneous techniques in order to close a post-MI VSD in a patient who is at high risk for repeat surgery. An unusual feature of the case is the availability of the patient's explanted heart, which enabled direct viewing of the successfully deployed closure device. Consistent with previous reports, this report shows that the staged approach was effective in avoiding a 2nd operation in a critically ill patient. We believe that this technique can be used for acute or permanent control of VSD expansion after initial surgical repair.

Two separate case reports recently discussed the importance of managing patients who are too ill to undergo early surgical closure of post-MI VSD.[22,31] Although delaying the closure of the VSD in such patients can result in a state of progressive multisystem failure, initial therapy through percutaneous closure can serve as a bridge to future surgical intervention.[31]

We also report the case of Patient 2, who presented with a VSD 4 weeks after the MI. The patient needed inotropic support to maintain hemodynamic stability and was at very high surgical risk. The decisions to avoid surgery because of the mortality rate associated with acute repair, and to stabilize the patient through percutaneous intervention, led to our successfully bridging her to surgery. We believe that the closure of post-MI VSDs in patients who are at extraordinary risk for operation affords extra time for improved tissue integrity, which can greatly strengthen future operative repair. As a bridge to surgery, a percutaneous closure device can initially stabilize a high-risk patient, provide additional tissue-recovery time, and afford easy removal at operation without impeding surgical closure.[22,31]

In the largest study to date, Thiele and colleagues[5] evaluated (in an acute setting, with 29 patients) the application of primary transcatheter closure of post-MI VSDs. Sixteen of the 29 patients (55%) presented in cardiogenic shock, and successful device deployment was achieved in 25 of the 29 (86%). The median time from MI to VSD was 2 days, and the median time from the VSD occurrence to percutaneous device closure was 1 day. The 30-day survival rate was only 35%. The long-term mortality rate (median follow-up, 730 d) of patients who developed a VSD, presented in cardiogenic shock, and underwent successful device implantation was 93%. However, patients who developed VSD, did not experience cardiogenic shock, and underwent successful device implantation had both 30-day and long-term mortality rates of 36%.

Our review of interventional postinfarction VSD procedures (Table I) is limited to case reports and small series.[5,17-22,24,28-46] Most such patients underwent VSD closure in the chronic or subacute phase, or experienced residual shunting after surgical patch closure.[5] This summary presents the results of a prospective series in which all patients with post-MI VSDs were treated in the acute setting, whether or not they were in severe cardiogenic shock. We attribute the high mortality rate to the selection of "all comers"; these outcomes cannot be compared with those of a surgical series, given the small number of patients and the inherent selection bias.

Concomitant cardiogenic shock, which might be present in these patients, is an important predictor of increased mortality rates. It seems reasonable to infer that outcomes in these patients (Table I) would have improved, had mechanical circulatory support been used to stabilize circulation of the blood before percutaneous or surgical repair. In one case series[47] not included in Table I, an IABP was used in 23 patients who had post-MI VSDs. Patients who underwent early surgical repair had an 83% mortality rate, whereas patients who were stabilized before surgical closure had an early mortality rate of 29% and an overall mortality rate of 56.5%. These patients were under IABP support for a range of 1 to 26 days. Conversely, in a study by La Torre and colleagues,[48] 5 patients in cardiogenic shock as a result of post-MI VSD underwent placement of the Impella® Recover® LP 5.0 Support System (ABIOMED Inc.; Danvers, Mass), in an effort to stablilize them before VSD repair. Two of the 5 died after surgical repair. In another case report,[49] the TandemHeart® percutaneous ventricular assist device (CardiacAssist Inc.; Pittsburgh, Pa) was used in a case of posterior post-MI VSD for a total of 18 days, which enabled hemodynamic stability and subsequent successful repair of the defect.

TABLE I. Reports of Percutaneous Transcatheter Closure of Post-Myocardial Infarction Ventricular Septal Defects

Reference	No. Patients	Acute	S/C	Prior Surgical Repair	Occluder Devices	Success Rate	Survival at 30 Days		Residual Shunt	
							Acute	S/C	Tr–Sm	Mod–Sv
Landzberg MJ and Locke JE[28] (1998)	18	NA	NA	11	CardioSeal	17/18	NA	NA	NA	NA
Lee EM, et al.[29] (1998)	1	0	1	1	ASO	1/1	—	1/1	1	—
Pesonen E, et al.[32] (2000)	2	0	2	2	ASO	2/2	—	2/2	2	—
Mullasari AS, et al.[30] (2001)	1	1	0	0	ASO	1/1	1/1	—	1	—
Pienvichit P and Piemonte TC[33] (2001)	1	1	0	0	CardioSeal	1/1	0/1	—	—	1
Parsi A, et al.[34] (2001)	2	1	1	0	MVSDO	2/2	0/1	1/1	—	—
Chessa M, et al.[35] (2002)	12	3	9	NA	MVSDO	11/12	0/3	8/9	NA	NA
Goldstein JA, et al.[18] (2003)	4	0	4	4	PIMVSDO	3/4	—	3/3	2	2
Szkutnik M, et al.[36] (2003)	7	0	7	1	4 ASO and 5 MVSDO	5/7	—	4/5	3	1
Zanchetta M, et al.[37] (2003)	1	1	0	0	ASO	1/1	NA	NA	NA	NA
Demkow M, et al.[38] (2005)	11	3	8	0	NA	10/11	0/2	9/9	8	2
Elsässer A, et al.[39] (2005)	1	0	1	0	PIMVSDO	1/1	—	1/1	1	—
Holzer R, et al.[17] (2006)	18	5	13	10	PIMVSDO	16/18	2/5	9/13	8	2
Martinez MW, et al.[40] (2006)	1	1	0	0	PIMVSDO	1/1	NA	NA	1	—
Martinez MW, et al.[19] (2007)	5	3	2	1	2 ASO, 2 MVSDO, and 2 PIMVSDO	5/5	2/3	2/2	3	1
Kaulfersch C, et al.[21] (2007)	22	22	0	0	NA	17/22	NA	NA	NA	NA
Costache VS, et al.[31] (2007)	1	1	0	0	NA	1/1	1	—	NA	NA
Bialkowski J, et al.[41] (2007)	19	1	18	2	17 ASO, 2 MVSDO, and 2 PIMVSDO	14/19	0/1	9/13	9	3
Marinakis A, et al.[42] (2007)	8	6	2	0	MVSDO	7/8	0/6	1/2	NA	NA
Ahmed J, et al.[43] (2008)	5	2	3	2	MVSDO and PIMVSDO	4/5	0/1	3/3	3	1
Giombolini C, et al.[44] (2008)	1	0	1	0	AMASDO	1/1	—	0/1	1	—
Thiele H, et al.[5] (2009)	29	29	0	0	ASO, MVSDO, and PIMVSDO	25/29	10/29	—	6	4
Maltais S, et al.[20] (2009)	12	12	0	0	PIMVSDO	11/12	7/11	—	10	1
Sun Y, et al.[45] (2009)	12	0	12	NA	NA	9/12	NA	NA	NA	NA
Gulkarov IM, et al.[24] (2010)	1	0	1	1	MVSDO	1/1	—	1/1	0	0
Avgerinos DV, et al.[22] (2011)	1	1	0	0	NA	1/1	1/1	—	0	1
Yalcin Y, et al.[46] (2011)	1	0	1	1	Cardio-O-Fix	1/1	—	1/1	0	0

AMASDO = Amplatzer Atrial Septal Defect Occluder; ASO = Amplatzer Septal Occluder; CardioSeal = CardioSeal Septal Occluder; MVSDO = Amplatzer Muscular Ventricular Septal Defect Occluder; Mod = moderate; NA = not available; PIMVSDO = Amplatzer Post-infarction Ventricular Septal Defect Occluder; S/C = subacute/chronic; Sm = small; Sv = severe; Tr = trivial

It seems plausible that percutaneous mechanical support might stabilize patients who are in cardiogenic shock as a sequela to their post-MI VSDs. Several case reports have described the management of hemodynamic dysfunction through the use of an IABP, an axial-flow pump, or a TandemHeart, with various results. The use of such devices in this circumstance will require more extensive study and experience. In addition, these devices can be used for high-risk percutaneous coronary artery intervention, and as an adjunct to surgical repair.[50] Myocardial revascularization in conjunction with VSD repair is imperative.

Conclusion

There is evidence supporting transcatheter device closure of post-MI VSDs. Although the gold standard is

still surgical repair with concurrent CABG, the application of this therapy to all patients might not be reasonable, especially when patients are critically ill or have multiple comorbidities. An interventional approach offers such patients the possibility of avoiding a major operation. The ability to manage a post-MI VSD in the acute setting can afford benefit—whether it be the closure of a residual defect after surgical repair or the provision of an opportunity to heal septal tissue in preparation for final closure. It should be emphasized that the mortality rate after transcatheter closure remains high, especially in the setting of cardiogenic shock. The high mortality rate in patients with post-MI VSD has not changed substantially during the last 2 decades.[51] It is feasible that the combined use of ventricular support, more complete revascularization, and earlier intervention will improve future outcomes.

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Erratum

With regard to the article by Maria D. Baldasare, et al., "Percutaneous Repair of Post-Myocardial Infarction Ventricular Septal Defect: Current Approaches and Future Perspectives," in Volume 41, Issue 6, pp. 613-9, the images for Fig. 2B and 2C were originally reversed but now appear online as they should be. The Figure 2 legend refers to all three images as coronary angiograms, but they are, in fact, left ventriculograms.