Transcatheter Closure of Post-myocardial Infarction Ventricular Septal Rupture

Gabriele Egidy Assenza, MD; Doff B. McElhinney, MD; Anne Marie Valente, MD; Disty D. Pearson, PA-C; Massimo Volpe, MD; Giuseppe Martucci, MD; Michael J. Landzberg, MD*; James E. Lock, MD*

Background—Ventricular septal rupture (VSR) after acute myocardial infarction (AMI) is a potentially lethal mechanical complication of acute coronary syndromes. Given high surgical mortality, transcatheter closure has emerged as a potential strategy in selected cases. We report our single-center experience with double-umbrella device percutaneous closure of post-AMI VSR.

Methods and Results—In this single-center, retrospective, cohort study, patients who underwent transcatheter closure of post-AMI VSR between 1988 and 2008 at Boston Children’s Hospital were included. Data were analysed according to whether the patients underwent direct percutaneous VSR closure or closure of a residual VSR after a previous surgical approach. Primary outcome was mortality rate at 30 days. Clinical predictors of primary outcome were investigated using univariate logistic regression. Thirty patients were included in the study (mean age, 67±8 years). A total of 40 closure devices were implanted. Major periprocedural complications occurred in 4 (13%) patients. Cardiogenic shock, increasing pulmonary/systemic flow ratio, and the use of the new generation (6-arm) STARFlex device all were associated with higher risk of mortality. The Model for End-Stage Liver Disease Excluding international normalized ratio (MELD-XI) score at the time of VSR closure seemed to be most strongly associated with death (odds ratio, 1.6; confidence interval, 1.1–2.2; P<0.001).

Conclusions—Transcatheter closure of post-AMI VSR using CardioSEAL or STARFlex devices is feasible and effective. The MELD-XI score, a marker of multiorgan dysfunction, is a promising risk stratifier in this population of patients. Early closure of post-AMI VSR is advisable before establishment of multiorgan failure. (Circ Cardiovasc Interv. 2013;6:59-67.)

Key Words: catheterization | interventional closure | myocardial infarction | ventricular septal defect

Ventricular septal rupture (VSR) after acute myocardial infarction (AMI) is a potentially lethal mechanical complication of acute coronary syndromes.1 VSR classically occurs as a bimodal presentation with a higher incidence in the first 24 hours and then again 3 to 5 days after AMI. The incidence of VSR has substantially decreased since the widespread implementation of early reperfusion strategies, ranging between 0.2% and 0.34% in the modern era. However, when it does occur, VSR clinical presentation is often ominous, is commonly associated with extensive comorbidities, and results in poor cardiac output, multiorgan failure, and death.2 Early surgical closure is recommended. This current indication, irrespective of hemodynamic status, is based on natural history data that suggest a 90% mortality within 2 months of diagnosis in patients who do not undergo defect closure.3

Since the original description of post-AMI VSR in 1957, surgical closure remains a challenging procedure with mortality rates between 20% and 87%.4,5 Expert opinion and professional guidelines suggest that immediate closure should be considered to reduce the duration of poor systemic perfusion that results from left-to-right shunting, pulmonary over circulation, and systemic hypoperfusion that may eventuate in refractory multiple organ failure and death.6 Despite this awareness, there is a tendency for surgeons to wait several weeks before operating to allow tissue healing and more complete rupture remodeling, contributing to high interstage mortality and positive selection of more favorable cases. In this setting, transcatheter approaches for closure of post-AMI VSR have been introduced as a less invasive means of reducing or eliminating shunt and improving antegrade perfusion,
WHAT IS KNOWN

- Ventricular septal rupture after acute myocardial infarction is a lethal mechanical complication of acute coronary syndromes.
- Early surgical closure is recommended, but implementation of such an indication in clinical practice is heterogeneous among centers because of the excessive surgical risk perceived by operators.
- Transcatheter closure of ventricular septal rupture emerged as a possible alternative to surgery in selected cases. However, limited information is available for such a procedure, which is currently an off-label indication.

WHAT THE STUDY ADDS

- This is a single-center series reviewing transcatheter closure of post-myocardial infarction ventricular septal rupture in 30 patients.
- We found that this procedure is technically feasible and can be accomplished with relatively low risk of major periprocedural complication, including death.
- Multiorgan failure dictates high risk of 30-day mortality, irrespective of closure status.

This study was compliant with the Health Insurance Portability and Accountability Act, and a waiver of informed consent was approved by the Institutional Review Boards of Boston Children’s Hospital, and Brigham and Women’s Hospital.

Patients were classified into 2 groups according to whether they underwent primary VSR closure with no surgical attempt before the index transcatheter procedure (primary VSR) or closure of a recurrent defect after a previous surgical VSR closure (residual VSR). In all patients the indication for closure relied on the presence of severe heart failure and pulmonary over circulation not amenable to medical therapy. Although prospective criteria were not used to guide the eligibility for transcatheter VSR closure, a surgical option was denied in all patients by consensus of experienced cardiologists and cardiac surgeons.

OUTCOMES AND DEFINITIONS

All-cause mortality at 30 days was defined as the primary outcome. Cardiogenic shock at the time of postinfarction VSR closure was defined as need for intravenous vasopressor support to maintain a systolic blood pressure >90 mm Hg, need for an intra-aortic balloon pump to sustain an adequate circulation, and evidence of poor end-organ perfusion (eg, increasing creatinine, increasing lactate, urine output <30 mL/h). Time to admission was defined as the time between symptom onset and primary hospital admission. Time to VSR closure was the time between symptom onset and VSR closure. In patients who underwent surgical closure as the first option (residual VSR group), time to surgery was the time between symptom onset and surgical VSR closure.

The MELD-XI score was calculated using creatinine and total bilirubin at the time of VSR closure according to the following formula: 5.11×ln (bilirubin mg/dL) + 11.76×ln (creatinine mg/dL) + 9.44.13

Transcatheter VSR Closure Procedure

Catheterization and VSR closure were performed under fluoroscopic guidance. In selected cases, intraprocedural transesophageal echocardiography was used to improve visualization of intracardiac anatomy. Invasive hemodynamic measurements were obtained for every patient before VSR closure, including calculation of the cardiac index, pulmonary/systemic flow ratio (using Fick equation), and direct measurement of the left ventricular (LV) end-diastolic pressure. Vascular access was generally obtained in the femoral artery and femoral vein. Internal jugular venous access was used for recovery of the venous arm of the arterio-venous or veno-venous loop to reduce torsional tension of the wire loop in the setting of posterior or very apical defects. In many cases a veno-venous loop was established using trans-septal puncture (Brockenbrough procedure) to reach the LV cavity (Figure 1). The defect was typically crossed from the LV cavity to the right ventricular cavity using a balloon-tipped, Judkins, Amplatzer, or multipurpose catheter and a long, soft guide wire. After advancing the catheter to the pulmonary artery, the wire was snared from the venous access and subsequently exteriorized through the femoral or the internal jugular vein to establish a loop (Figure 2, Video I in the online-only Data Supplement).

An appropriate long sheath was then advanced over the wire and across the defect (Figure 2). The approach for sheath delivery and deployment depended on the location of the defect and other factors specific to individual anatomic constraints. In our early experience, the device chosen was 1.5-times to 2.2-times the maximal balloon, angiographic, or echocardiographic size, although in our more recent experience, we chose the largest device available that could fit in the specific anatomic location. After deployment and complete expansion of the proximal ventricular arms, LV angiography was usually performed through a separate catheter to assess the position of the device and assess for residual intracardiac shunt, valve interference, and device position.

Devices

Three generations of double-umbrella closure devices were deployed during this study: the first-generation Clamshell device (C.R. Bard,
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prior vena cava (see Video I in the online-only Data Supplement).

pulmonary artery and to exteriorize the venous arm from the inferior vena cava (see Video I in the online-only Data Supplement).

rupture and to advance the long soft guidewire into the pulmonary artery, where it is snared (B) to avoid potential ensnaring of the tricuspid valve subvalvar apparatus.

A long, soft guide wire is advanced across the ventricular septal puncture is used to advance a catheter into the left ventricle.

Between-group comparisons for clinical and outcome variables were performed using independent samples t test, Wilcoxon rank-sum test, \( \chi^2 \) analysis, or Fisher exact test. Receiver–operator characteristic curves were used to determine optimal thresholds for dichotomizing predictor variables. Univariate logistic regression was used to identify predictors of composite outcome. The following predictors have been included in the analysis: type of defect (residual versus primary); LV ejection fraction; cardiogenic shock at the time of closure; hemodynamics at the time of closure (LV end-diastolic pressure, cardiac index, and the ratio between pulmonary and systemic blood flow); MELD-XI score at the time of closure; white blood cell count at the time of closure; fever at the time of closure; time to closure; number of coronary arteries involved; and device type. Because of the small number of events, multivariable analysis of outcomes was not performed. Data are reported as mean±SD, median (first and third quartiles), or frequency (%). Odds ratios are presented with 95% confidence intervals. All tests are 2-sided. Given the exploratory nature of the study, no adjustment for multiple comparisons was performed.

Results

Patients

Thirty patients underwent transcatheter closure of post-AMI VSR during the study period. Table 1 summarizes demographic, clinical, and laboratory features of the entire study population and the 2 clinical subgroups. Twelve patients underwent primary VSR closure, and 18 had double-umbrella devices placed after an initial surgical procedure after a median of 52 days from the original operation (first and third quartile range, 21–170 days). On average, patients were in the sixth decade of life and had multiple coronary artery disease risk factors. The majority of patients received aspirin and heparin before VSR closure, but only a minority were treated with \( \beta \)-blockers or systemic thrombolysis. Cardiogenic shock was present in 17 patients (57%) at the time of VSR closure. Patients who underwent primary VSR closure had a shorter time to transcatheter closure than postsurgical closure patients. An abnormal serum creatinine level was common; the calculated MELD-XI score was significantly higher in those undergoing primary closure.

VSR was the result of myocardial necrosis attributable to ST elevation myocardial infarction in all but 1 patient. ST elevation myocardial infarction localization involved the anterior wall of the left ventricle in 15 patients (50%) and the inferior wall of the LV in 11 patients (39%). Table 2 summarizes hemodynamic, angiographic, and reperfusion strategy features of the cohort. In patients who underwent primary VSR closure, the VSR diameter by angiography was significantly larger than in patients with residual postsurgical VSR. In the 26 patients in whom coronary angiograms were available for review, 21 had stenoses or occlusions in 1 or 2 epicardial coronary arteries, and only 5 had multivessel obstruction. Myocardial reperfusion with percutaneous coronary intervention or coronary artery bypass surgery before VSR closure was performed in 21 patients. Percutaneous coronary intervention was never simultaneous with transcatheter VSR closure. In all but 1 of the patients who underwent coronary artery bypass grafting, surgical closure of the VSR was performed at the time of coronary surgery. In 3 other patients, surgical VSR closure was performed without concomitant coronary artery bypass grafting.

Data Analysis

Between-group comparisons for clinical and outcome variables were performed using independent samples t test, Wilcoxon rank-sum test, \( \chi^2 \) analysis, or Fisher exact test. Receiver–operator characteristic curves were used to determine optimal thresholds for dichotomizing predictor variables. Univariate logistic regression was used to identify predictors of composite outcome. The following predictors have been included in the analysis: type of defect (residual versus primary); LV ejection fraction; cardiogenic shock at the time of closure; hemodynamics at the time of closure (LV end-diastolic pressure, cardiac index, and the ratio between pulmonary and systemic blood flow); MELD-XI score at the time of closure; white blood cell count at the time of closure; fever at the time of closure; time to closure; number of coronary arteries involved; and device type. Because of the small number of events, multivariable analysis of outcomes was not performed. Data are reported as mean±SD, median (first and third quartiles), or frequency (%). Odds ratios are presented with 95% confidence intervals. All tests are 2-sided. Given the exploratory nature of the study, no adjustment for multiple comparisons was performed.

VSR Closure and Procedural Outcome

A total of 40 closure devices were implanted in the 30 study patients. Clamshell devices were used in our first experience with this procedure (between 1988 and 1997), CardioSEAL devices were used between 1994 and 2008, and StarFLEX device were used in the latest era of the study (after 2001).

In 8 patients, multiple devices were implanted because of persistence or recurrence of large-volume shunts after the first device was placed. In 5 patients, additional devices were implanted at the time of the first VSR closure procedure. In 2 patients, 1 additional procedure was performed a few days after the first intervention to implant a second device. In another patient, 2 additional procedures were performed.
(1 week and 11 months later, respectively), implanting a total of 3 devices because of a large residual shunt. Tables 3 and 4 summarize procedural details. Major periprocedural complications occurred in 4 patients (13%), as listed in Table 4. There was 1 procedural death occurring in a patient who had development of electromechanical dissociation during intracardiac manipulation of the delivery sheath, and who was not able to be restored to a perfusing rhythm. This patient presented with a large anterior ST elevation myocardial infarction and was treated with primary angioplasty a few hours before VSR closure. One other patient had development of transient complete heart block during manipulation of the long sheath and was treated with temporary pacing; heart block resolved after few minutes and a 38-mm STARFlex device was successfully implanted. A permanent pacemaker was not implanted.

In 2 patients, the device embolized from the optimal position. In 1 patient, a 40-mm Clamshell device dislodged from the initial position and became entangled in the tricuspid subvalvar apparatus. This device was retrieved a few days later using a transcatheter approach; the patient ultimately died a few days after this procedure because of multiorgan failure. The VSR diameter by angiography was 18 mm. In another patient, a 33-mm Clamshell device dislodged from the initial position but was retrieved and a 40-mm Clamshell device was then successfully implanted. In this patient, before the implantation of the second device, the VSR was tested with low-pressure sizing balloon inflation, giving a stretched diameter >20 mm.

In 3 patients (all presenting with primary VSR), surgical repair was performed after transcatheter closure because of persistence of large-volume shunt. In 1 case, surgical repair was accomplished the same day of the failed percutaneous repair. In the other 2 patients, surgical repair was completed 1 and 4 months after percutaneous closure, respectively.

### 30-Day Mortality
The primary outcome, death within 30 days of VSR closure, occurred in 7 patients (23%). Comparing the outcome rate in the first consecutive 15 patients with the rest of the cohort, no difference was noted (20% versus 27%; P=0.7). All deaths (with the exception of the intraprocedural event) were related to multiorgan failure in the setting of systemic hypoperfusion despite aggressive medical therapy, inotropic support, and intra-aortic balloon pump. There was a trend toward a higher 30-day mortality in patients who underwent primary VSR closure (5 of 12, 42%) compared with patients with a residual VSR after surgical closure (2 of 18, 11%; P=0.08). Factors significantly associated with 30-day mortality at a 2-sided 0.05 level of significance are summarized in Table 5.

Cardiogenic shock, increasing pulmonary/systemic flow ratio (at the time of percutaneous closure), and use of the 43-mm (6-arm) STARFlex device were all related to increased risk of mortality. The baseline MELD-XI score at the time of VSR closure was strongly associated with increased risk of death. On receiver–operator characteristic curve analysis, a MELD-XI score of 20 was associated with 100% sensitivity and 87% specificity for predicting the primary outcome. The association between baseline MELD-XI score and outcome remained significant even after repeated the analysis in the 2 clinical subgroups (primary VSR closure versus residual VSR closure), although with marginal probability values because of the small sample size (primary VSR group: dds ratio, 1.49
Discussion

Post-AMI VSR: Management Considerations

Our study reports on the use of double-umbrella devices (Clamshell, CardioSEAL, and STARFlex devices) in the transcatheter closure of post-AMI VSR. Our experience suggests that these devices can be used in these challenging interventional procedures achieving reasonable technical outcome and low rate of major complications. The high 30-day mortality (23%), which was attributable to multiorgan dysfunction and poor systemic perfusion, reflects the burden of cardiac dysfunction and extensive comorbidity, which is usually present in this group of patients.\(^8\) As previously reported, we confirmed that cardiogenic shock at the time of clinical presentation and the hemodynamic burden imposed by the left-to-right shunt are important predictors of unfavourable outcomes.\(^9\)

In an effort to better understand the risks associated with multiorgan system dysfunction in this setting, we examined the MELD-XI score\(^13\) and found it to be strongly associated with 30-day mortality. Assuming normal total bilirubin and creatinine values, the MELD-XI score is between 9.4 and 12 in normal subjects. In our study, 62% of patients with a MELD-XI score >20 died within 30 days of VSR closure. Our data suggest that multiorgan failure dictates a very high mortality rate irrespective of closure attempt. Recently, aggressive ventricular mechanical support using biventricular assist device or extracorporeal membrane oxygenators have been proposed to bridge patient presenting with post-AMI VSR to closure or cardiac transplantation in case of end-stage ventricular failure.\(^14\)–\(^16\) Recent data from the Society of Thoracic Surgeons database reported on >2800 surgical post-MI VSR closures performed in North America between

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Table 1. Demographics of the Study Population and the Two Clinical Subgroups

<table>
<thead>
<tr>
<th></th>
<th>Overall Cohort (n=30)</th>
<th>Primary Ventricular Septal Rupture (n=12)</th>
<th>Residual Ventricular Septal Rupture (n=18)</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male, n (%)</td>
<td>12 (41)</td>
<td>8 (67)</td>
<td>8 (44)</td>
<td>0.2</td>
</tr>
<tr>
<td>BSA, kg/m(^2)</td>
<td>1.8±0.2</td>
<td>1.9±0.2</td>
<td>2.2±0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>Age, y</td>
<td>67±8</td>
<td>68±6</td>
<td>67±9</td>
<td>0.6</td>
</tr>
<tr>
<td>Systemic arterial hypertension, n (%)</td>
<td>12 (41)</td>
<td>7 (58)</td>
<td>5 (29)</td>
<td>0.1</td>
</tr>
<tr>
<td>Hypercholesterolemia, n (%)</td>
<td>12 (41)</td>
<td>7 (58)</td>
<td>5 (29)</td>
<td>0.1</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>4 (14)</td>
<td>2 (17)</td>
<td>2 (12)</td>
<td>0.7</td>
</tr>
<tr>
<td>Active smoking, n (%)</td>
<td>4 (14)</td>
<td>3 (25)</td>
<td>1 (6)</td>
<td>0.1</td>
</tr>
<tr>
<td>Family history of CAD, n (%)</td>
<td>5 (17)</td>
<td>3 (25)</td>
<td>2 (12)</td>
<td>0.4</td>
</tr>
<tr>
<td>History of CAD, n (%)</td>
<td>4 (14)</td>
<td>2 (17)</td>
<td>2 (12)</td>
<td>0.7</td>
</tr>
<tr>
<td>History of stroke, n (%)</td>
<td>2 (7)</td>
<td>1 (8)</td>
<td>1 (6)</td>
<td>0.8</td>
</tr>
<tr>
<td>ASA before VSR closure, n (%)</td>
<td>20 (67)</td>
<td>9 (75)</td>
<td>11 (55)</td>
<td>0.4</td>
</tr>
<tr>
<td>Heparin before VSR closure, n (%)</td>
<td>18 (60)</td>
<td>8 (67)</td>
<td>10 (57)</td>
<td>0.5</td>
</tr>
<tr>
<td>GpIIb-IIIa before VSR closure, n (%)</td>
<td>2 (7)</td>
<td>1 (8)</td>
<td>1 (6)</td>
<td>0.8</td>
</tr>
<tr>
<td>Beta-blocker before VSR closure, n (%)</td>
<td>9 (30)</td>
<td>3 (25)</td>
<td>6 (67)</td>
<td>0.6</td>
</tr>
<tr>
<td>Systemic thrombolysis before VSR closure, n (%)</td>
<td>4 (13)</td>
<td>1 (8)</td>
<td>3 (17)</td>
<td>0.5</td>
</tr>
<tr>
<td>LVEF by echocardiogram, %</td>
<td>48±10</td>
<td>50±14</td>
<td>47±8</td>
<td>0.3</td>
</tr>
<tr>
<td>VSR diameter by echocardiogram, mm</td>
<td>13.6±6.7</td>
<td>16.7±9.1</td>
<td>11.2±3</td>
<td>0.2</td>
</tr>
<tr>
<td>At least moderate mitral regurgitation by echocardiogram, n (%)</td>
<td>3 (10)</td>
<td>2 (17)</td>
<td>1 (6)</td>
<td>0.3</td>
</tr>
<tr>
<td>Cardiogenic shock at the time of VSR closure, n (%)</td>
<td>17 (57)</td>
<td>7 (58)</td>
<td>10 (56)</td>
<td>0.6</td>
</tr>
<tr>
<td>IABP at the time of VSR closure, n (%)</td>
<td>14 (47)</td>
<td>5 (42)</td>
<td>9 (50)</td>
<td>0.7</td>
</tr>
<tr>
<td>Inotropic support at the time of VSR closure, n (%)</td>
<td>13 (43)</td>
<td>5 (42)</td>
<td>8 (44)</td>
<td>0.9</td>
</tr>
<tr>
<td>Time to admission, d</td>
<td>0 (0–8)</td>
<td>7 (0–22)</td>
<td>0 (0–5)</td>
<td>0.049</td>
</tr>
<tr>
<td>Time to surgery, d</td>
<td>NA</td>
<td>NA</td>
<td>4.5 (0–10)</td>
<td>NA</td>
</tr>
<tr>
<td>Time to VSR closure, d</td>
<td>27 (17–127)</td>
<td>19 (11–27)</td>
<td>54 (22–173)</td>
<td>0.045</td>
</tr>
<tr>
<td>Creatinine at the time of VSR closure, mg/dL</td>
<td>1.6 (1.2–2.45)</td>
<td>2.3 (1.2–3.6)</td>
<td>1.4 (1.15–1.7)</td>
<td>0.1</td>
</tr>
<tr>
<td>Bilirubin at the time of VSR closure, mg/dL</td>
<td>1.2 (0.7–1.5)</td>
<td>1.5 (0.7–1.7)</td>
<td>1.2 (0.7–1.2)</td>
<td>0.03</td>
</tr>
<tr>
<td>MELD-XI score at the time of VSR closure</td>
<td>16 (12–21)</td>
<td>19 (16–26)</td>
<td>14 (12–18)</td>
<td>0.03</td>
</tr>
<tr>
<td>WBC 10(^3)/μL</td>
<td>12.72 (7.32–14.93)</td>
<td>13.28 (7.36–16.23)</td>
<td>12.15 (7.28–14.47)</td>
<td>0.9</td>
</tr>
<tr>
<td>Fever at the time of VSR closure, n (%)</td>
<td>8 (29%)</td>
<td>2 (18%)</td>
<td>6 (35%)</td>
<td>0.4</td>
</tr>
</tbody>
</table>

Data are presented as as mean±SD, median (first and third quartiles), or frequency (%).

ASA indicates aspirin; BSA, body surface area; CAD, coronary artery disease; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; MELD-XI, Model for End-stage Liver Disease Excluding International normalized ratio; NA, not applicable; VSR, ventricular septal rupture; and WBC, white blood cell.

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per 1-unit difference; confidence interval, 0.99–2.26; \(P=0.01\); residual VSR group: odds ratio, 1.65 per 1-unit difference; confidence interval, 0.7–3.9; \(P=0.07\).
creatinine and need for preoperative dialysis were powerful predictors of cardiovascular death. Use of MELD-XI score may improve our ability to identify this high-risk group of patients earlier in whom aggressive mechanical ventricular support might improve overall outcome as a bridge to transplant or as adjunctive therapy in conjunction with surgical or percutaneous closure.

Eighteen patients were referred to our institution after failure to achieve adequate surgical closure of VSR. In this subgroup of patients, our results were most promising, with excellent technical outcome (immediate effective closure in every patient) and relatively low mortality at 30 days. One of the concerns that prevent early surgical post-AMI VSR closure is that the margins of the defect do not offer sufficient strength for suturing the patch, which could result in higher rate of recurrence after early surgery. However, waiting for complete healing of the VSR margins has significant risk.5 Our data suggest that a hybrid approach, with early surgery and potential transcatheter closure of residual or recurrent defects, is feasible. This strategy may prevent the detrimental effects of prolonged poor systemic perfusion and left heart overload, and seems particularly reasonable in patients with large defects in which a higher rate of device dislodgment and technical failure may be anticipated.10

Post-AMI VSR: Technical and Procedural Considerations

Our current practice is to perform VSR closure, as possible, within 24 hours of diagnosis. We tend to favor a dual venous access for establishing the wire loop with a trans-septal
approach because it lessens hemodynamic instability related to the retrograde crossing of aortic valve. In addition, the transseptal course of the delivery catheter allows a more favorable intracardiac manipulation of the device because it permits the opening of the distal device arms in the inflow portion of the right ventricle rather than in the right ventricular apex.

VSR are classically serpiginous ruptures rather than smoothly defined passages (Figure 3, Video II in the online-only Data Supplement). Often the LV entry and the right ventricular exit of the defect can be distant and posteriorly located. Particular attention to anatomic details and proximity to adjacent structures are required. Localized angiography (Video III in the online-only Data Supplement) and multiplanar imaging seem to be useful in this setting.

We usually defer balloon sizing the defect, given that necrosis and myocardial remodeling likely change defect size and shape, and that balloon manipulation may worsen tissue dehiscence or lead to rupture extension of the free (ventricular) wall. It is not uncommon to require additional device implantation either at the time of initial closure or within 3 to 6 days of the initial procedure, and patients and families should be counseled regarding this potential. The risk of subacute recurrence
of shunt after transcatheter closure of post-AMI VSR should prompt scheduled echocardiographic surveillance to improve early recognition and to guide therapeutic decisions.

**Post-AMI VSR: Device-Specific Considerations**

In our series, there was 1 intraprocedural death and major non-fatal complications in 4 patients. The majority of the reports on transcatheter closure of post-MI VSR have been limited to a single case or small series of patients.\(^1\)\(^-\)\(^2\)\(^3\) Larger series reporting on the use of Amplatzer occluder devices present a 30-day mortality rate between 28% and 42%.\(^8\)\(^-\)\(^1\(^0\)\(^1\) In those studies, the intraprocedural death rate ranged from 0% to 17%. Interestingly, the majority of intraprocedural deaths were related to myocardial rupture during intracardiac manipulation of the Amplatzer device.\(^9\)\(^,\)\(^1\(^0\)\(^1\) No direct comparison can be made between our results and the other series that reported on use of alternative devices because of the nonrandomized study design, the highly selected groups of patients, and different protocols. However, multiple concerns have been raised on the use of Amplatzer device in this setting, including rigid structure of the device, which may increase the risk of myocardial rupture; limited available sizes of the Amplatzer muscular ventricular septal defect occluders; and a suboptimal profile of the Amplatzer atrial septal defect occluder, with its relatively short waist that can lead to device deformation after deployment (cobra effect).\(^9\) In our experience, we did not observe any major mechanical complications related to device manipulation and deployment. We speculate that the lower and more flexible profile of CardioSEAL and STARFlex devices may reduce the risk of myocardial rupture during manipulation of the device in the infarcted ventricle during deployment phase, as well as during ventricular remodeling, as compared with the Amplatzer device.

**Limitations**

This is a retrospective study reporting on patients who underwent device closure of post-AMI VSR during a 20-year period. As such, it is certain that there are a number of limitations, including changes in management and unmeasured variables that might introduce bias. The limited number of patients in this series precluded any meaningful multivariable analysis to identify independent predictors of mortality.

We did not use 3-dimensional transesophageal echocardiography in this study. This advanced noninvasive imaging modality is increasingly recognized as a powerful tool to visualize complex anatomic structures, improving procedural outcome during cardiac surgery and complex catheter-based interventions, including post-AMI VSR percutaneous closure.\(^2\(^4\)\)

**Conclusions**

Post-AMI VSR is a lethal complication of acute coronary syndromes. The detrimental interplay between cardiogenic shock, high-volume intracardiac shunting, and pulmonary overcirculation frequently results in severe multiorgan failure and leads to high risk of mortality. Transcatheter reduction or closure of post-AMI VSR using double-umbrella, CardioSEAL, or STARFlex devices is feasible either as a primary therapy or in a hybrid approach including transcatheter closure of potential residual VSR after surgical repair. The MELD-XI score, a marker of multiorgan dysfunction, seems to be a promising risk stratifier in this population of patients.

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Disclosures
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Gabriele Egidy Assenza, Doff B. McElhinney, Anne Marie Valente, Disty D. Pearson, Massimo Volpe, Giuseppe Martucci, Michael J. Landzberg and James E. Lock

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SUPPLEMENTAL MATERIAL

Video captions

Video 1

The video shows the snaring of a long, soft guidewire occurring in the pulmonary artery. A snaring system was advanced into the pulmonary artery from the inferior vena cava.

Video 2

Intra-procedural, trans-esophageal echocardiography. This video refers to the same patient reported in Figure 3. Color-doppler shows the location and extent of the ventricular septal rupture.

Video 3

Left oblique, anterior, angiographic view of a post acute myocardial infarction ventricular septal rupture, using over-the-wire power injection through a long sheath. The sheath was advanced into the left ventricular cavity using trans-septal approach.