Novel Insights from Clinical Experience

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Unplanned Staged Hybrid Management of Postmyocardial Infarction Ventricular Septal Defect

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Established Facts

- Outcomes with postinfarction ventricular septal defect are poor with current management strategies.
- Ventricular septal defect recurrence after repair is due to infarct extension or friable myocardial tissue at the repair site.

Novel Insights

- A planned, staged hybrid approach with percutaneous followed by surgical intervention may improve outcomes.

Key Words
Coronary heart disease · Myocardial infarction · Ventricular septal defect

Abstract
Ventricular septal defect (VSD) is an uncommon but potentially deadly complication of transmural myocardial infarction (MI). Emergency surgical treatment has traditionally offered the best chance for survival. However, operative intervention is associated with high mortality and can be complicated by a recurrent VSD due to tissue friability around the infarcted area. Percutaneous catheter-based closure techniques can be used to treat these critically ill patients, offering a less invasive and less morbid technique. This case demonstrates the successful application of an unplanned, staged hybrid approach utilizing initial percutaneous and subsequent surgical repair after recovery of tissue integrity.

Acute ventricular rupture is a grave complication of myocardial infarction. Survival with medical therapy alone at 1 year is <10%. In-hospital mortality after surgi-

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cal repair ranges from 20 to 50% [1, 2]. Increased mortality rates relate primarily to end-organ consequences of cardiogenic shock, right heart failure and patch dehiscence with recurrent ventricular septal defect (VSD) and shunt. Surgical repair is difficult due to the presence of friable tissue surrounding the infarction, particularly in the acute setting. Infarct extension after repair can also affect tissue integrity and compromise repair. Thus, dehiscence is common and estimated to occur in 1 out of 5 surgical repairs [3–7]. This recurrence carries early mortality as high as 45% in some series [1, 6, 8–10].

There has been recent progress with percutaneous transcatheter treatment of postinfarct VSD. Various occlusion devices have been employed successfully to achieve closure. However, experience with the transcatheter approach is limited and its role in the acute setting is not well delineated.

We describe a case of a patient who underwent primary percutaneous device closure of a postinfarct VSD, followed weeks later, by operative repair for recurrent VSD as a de facto staged hybrid technique with excellent final outcome.

Case Report

A 72-year-old female with a known history of hypertension and coronary artery disease presented with chest pain, shortness of breath and diaphoresis. Electrocardiography on presentation was notable for anterolateral ST-segment depressions, and laboratory findings were significant for cardiac troponin I elevation of 2.5 ng/ml. She was started on aspirin, clopidogrel and unfractionated heparin infusion and was taken urgently for coronary angiography. This revealed 3-vessel coronary artery disease with subtotal occlusion of the mid left anterior descending artery and 85% stenosis of the proximal left circumflex artery in a left dominant system. Thrombolysis in myocardial infarction 3 flow was present in all 3 coronary vessels. Left ventricular function was severely decreased (ejection fraction 35%). An anteroapical VSD was diagnosed on left ventriculography, measuring 1.0 × 1.2 cm. Right heart catheterization revealed a significant step-up in saturations, confirming a left-to-right intraventricular shunting, with a calculated ratio of pulmonary (Qp) to systemic (Qs) blood flow of 2.0.

An intra-aortic balloon pump (IABP) was placed and the patient was transferred to the coronary care unit in stable condition. After multidisciplinary discussion between surgeons, interventional cardiologists and the patient's family, the decision was made to proceed with transcatheter closure of the VSD. Forty-eight hours later, VSD closure was performed with a 12-mm Amplatzer occluder device (AGA Medical Corporation, Plymouth, Minn., USA) in the cardiac catheterization laboratory. Post-procedure Qp/Qs ratio was 1.3. There was a minimal residual shunt present on transesophageal echocardiogram at completion of the percutaneous repair. The patient did well periprocedurally and the IABP was removed within 24 h of procedure.

The patient then underwent staged percutaneous angioplasty and stenting of the mid left anterior descending and proximal left circumflex arteries 48 h after VSD closure. She tolerated percutaneous coronary interventions well and was discharged home shortly thereafter in stable condition.

The patient presented to the emergency department on post-discharge day 8 with shortness of breath and peripheral edema. Diagnostic work-up revealed hepatic and renal insufficiency and worsening pulmonary hypertension. Transthoracic echocardiogram at this time revealed recurrent VSD with a significant increase in shunt fraction (Qp/Qs ratio 1.9).

Operative repair was recommended. An IABP was placed for hemodynamic support, and over the ensuing 4 days, end-organ function improved and the patient was then taken to the operating room for a patch repair.

At operation, the Amplatzer device was found to be occlusive over two thirds of its circumference with a clear gap posteriorly (fig. 1a). It was carefully freed from surrounding tissues and was extracted without difficulty. Tissue integrity was excellent. A Hemashield patch (Maquet, Inc., Wayne, N.J., USA) was used to close the VSD (fig. 1b) via a right ventricular approach. She was weaned from cardiopulmonary bypass without difficulty. There was no evidence of shunt at completion of the operation. Postoperatively, the patient did well with normalization of end-organ function. She was discharged on postoperative day 21.

Discussion

Postmyocardial infarction VSD is a dreaded, but fortunately rare, complication of myocardial infarction. Survival with medical therapy is abysmal while surgery is beset with mortality rates as high as 50% [1, 10]. VSD recurrence occurs in up to 20% of patients. Infarct extension after closure is a clear culprit. Myocardial friability in the tissues surrounding the infarct causing patch dehiscence after open repair also contributes to high recurrence rates. The morbidity and mortality associated with surgical repair has provided an incentive to develop percutaneous methods of closure for these defects.

Advances in the development of closure devices have allowed for successful treatment of various complex ventricular and atrial septal defects. While specific clinical experience with postinfarction VSDs is limited, it is a clear alternative to risky surgical intervention.

Our case is unique in that while we were not able to achieve definitive management percutaneously, we were able to achieve closure as a temporizing measure. During this time, the patient demonstrated right heart recovery and, importantly, development of adequate tissue integrity which greatly facilitated operative closure and our ability to achieve an excellent result. The decision to initially treat the patient with a percutaneous approach was

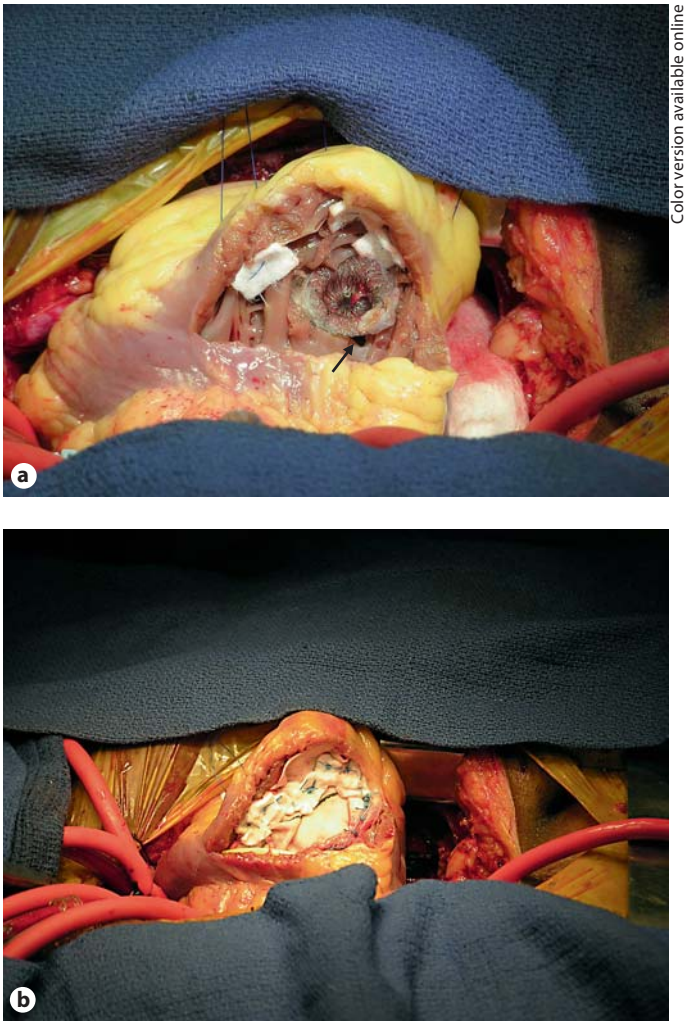

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Fig. 1. a Intraoperative photo from the right ventriculotomy demonstrating the Amplatzer occluder device and residual VSD (black arrow). **b** Final Hemashield patch closure.

dictated by her poor clinical condition and favorable intracardiac pathology, and also clearly by the fact that operative repair of acute postinfarct VSD carries a high mortality across the board [11]. As a hybrid approach, the use of this technique was instrumental in achieving a successful outcome as we benefitted greatly from the additional time for healing of the infarcted tissues. This is in contrast to our previously published case report where the percutaneous technique was employed after dehiscence of the patch that was placed operatively in the acute setting [12].

While our intentions were to definitively treat the postinfarct VSD percutaneously, we have made some significant observations about our eventual management strategy. First, clearly, a percutaneous approach is less traumatic to the patient's general physiology and recovery is much more rapid. Additionally, the delivery of the percutaneous closure device did not burn any bridges surgically. The closure device was easy to remove at operation and did not impede with our ability to achieve surgical closure. In fact, the device gave the patient additional time to recover and heal prior to operative repair which arguably allowed us to achieve an excellent technical result and overall outcome. Lastly, as previously mentioned, infarct extension is a clear problem and source of morbidity after repair in the acute setting. By taking a staged approach, infarct extension can occur with the percutaneous device in place prior to definitive surgical therapy which will be at some delayed interval, presumably after infarctions have completed their course. This, in turn, further decreases chances of dehiscence and recurrent VSD.

In the future, larger series with short- and long-term follow-up and comparisons between surgical and percutaneous management of these patients, as well as comparisons of different devices, are needed to better clarify the role of percutaneous repair in the treatment of postmyocardial infarction VSDs. The potential combination with open surgery as a staged hybrid model, as in our case, is particularly exciting. These approaches may allow us to eventually lower the historically high mortality rates associated with this difficult pathology.

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