

Transcatheter closure of residual postinfarction ventricular septal defect after dehiscence of surgical patch repair

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Ventricular septal defect (VSD) is a life-threatening complication of transmural myocardial infarction. Urgent surgical repair and concomitant revascularization are the standard of care. Percutaneous catheter-based closure techniques have been reserved for patients with a high-risk surgery or a failed surgical procedure with residual shunting. This case report demonstrates the successful transcatheter closure of residual VSD using the Amplatzer muscular VSD device (Amplatzer, Minnesota, USA) after surgical patch dehiscence for postinfarction VSD and 3-and-a-half years' post-intervention follow-up.

Ventricular septal defect (VSD) is a known mechanical complication of acute myocardial infarction (MI), which tends to occur 3 to 8 days after MI but may occur within first 24 hours or as late as 2 weeks.¹ In the prethrombolytic era, its incidence was 1% to 2%, which reduced significantly to 0.2% in the era of thrombolytic therapy and primary percutaneous coronary intervention.² Despite reduction in incidence and recent advancement, mortality is very high with 35% survival after 1 month.³ Emergency surgery is the standard of care but is associated with high hospital mortality, which ranges from 10% to 60% depending on preoperative patient characteristics.⁴ Postsurgical shunt recurs in up to 10% to 20% of patients.⁴ Patch dehiscence, development of a new VSD, or an overlooked second VSD are among the important causes for recurrence.⁴ We present a case of transcatheter closure (TCC) for residual postinfarction VSD using the Amplatzer muscular VSD device 5 months after surgical repair in a patient presenting with decompensated congestive cardiac failure together with the review of published reports.

CASE

A 60-year-old female presented to the emergency room with an acute inferior MI and was thrombolized with streptokinase within 5 hours from the onset of chest

pain. She remained stable initially but developed pulmonary edema with oxygen desaturation on the third day. Vital signs were stable with elevated jugular venous pressure and pan-systolic murmur at the left sternal border. Echocardiography revealed akinetic basal to mid-inferior and posterior walls with the presence of basal septal defect and left-to-right shunt. Coronary angiogram revealed 3-vessel disease with mild left main involvement. Left ventriculogram confirmed the basal septal VSD. The patient was referred to our center for coronary bypass surgery (CABG) and VSD repair. On Dec 31, 2008, she underwent 4-vessel CABG and patch repair of VSD through the tricuspid valve using the exclusion technique. During surgery, the septum was found to be extensively damaged and friable, and a large patch of bovine pericardium was used to exclude the necrotic septum. Transesophageal echocardiography (TEE) during surgery showed successful closure, and subsequent echocardiography did not show any significant shunt across septum. She had a long postsurgical course with a severe heart failure, which gradually recovered and she was able to be sent home in a fair condition 4 weeks postoperatively. She was admitted with clinical features of pulmonary edema within a month. Echo revealed residual VSD with a left-to-right shunt involving basal inferior septum due to dehiscence of the

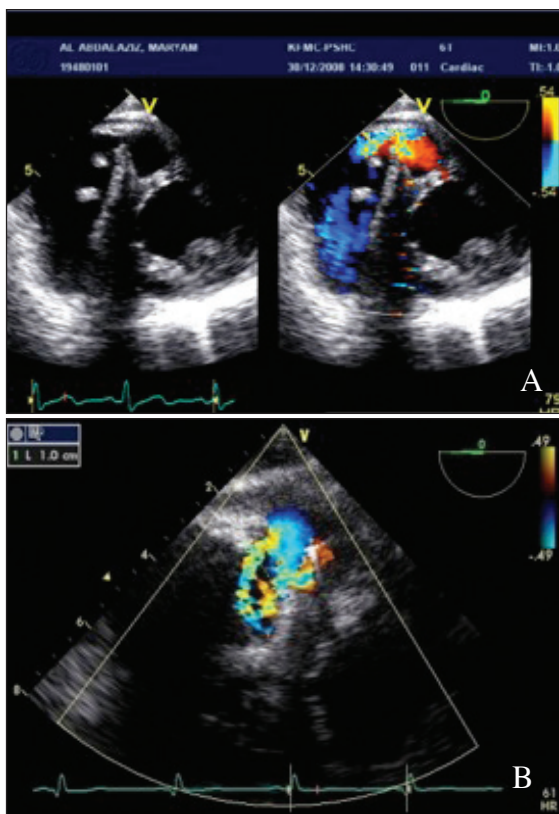


Figure 1. A) Short axis view at mid cavity level showing ventricular septal defect with left to right shunt. B) Color Doppler, short axis, mid cavity level showing left to right shunt.

patch, and the pulmonary-to-systemic flow ratio was 2.3 (Figure 1). She had multiple further admissions with congestive heart failure and remained in decompensated state for most of the time. She was planned for a percutaneous closure of residual VSD after a discussion in a combined cardiology and cardiac surgery meeting. The procedure was performed 5 months later to surgery.

Techniques

Right and left heart catheterization and left ventricular (LV) angiogram were performed to assess the shunt and define the anatomy. Angiographically, there was a significant left-to-right shunt. Then a JR-4 catheter and Terumo wire (Terumo Corporation, Tokyo, Japan) was used to cross the VSD retrogradely. The wire and catheter were advanced to pulmonary artery (PA). The wire was then exchanged for a long exchange wire. The wire was subsequently snared in the PA and was pulled back out of the femoral vein creating an arteriovenous loop. Then the VSD delivery sheath was advanced antegradely while pulling back the JR-4

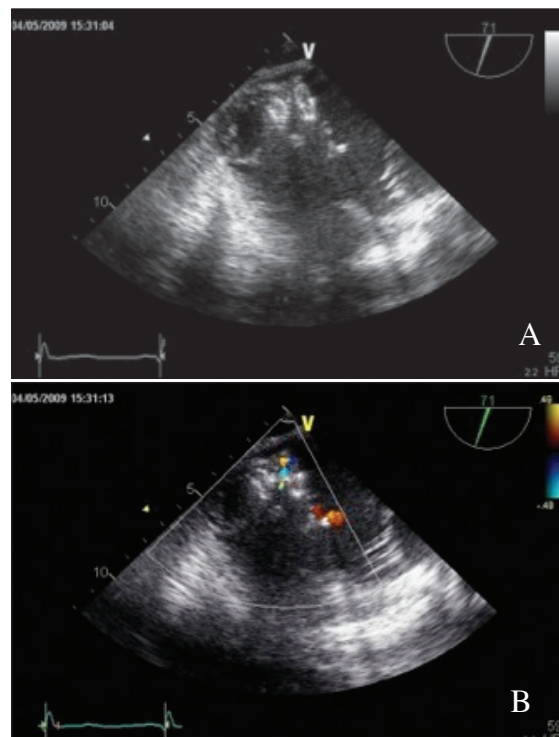


Figure 2. A) Closure of septal defect with percutaneous device. B) Color Doppler showing trivial shunt across the device.

catheter. The sheath was advanced all the way to the aorta and the wire was removed and then the sheath was pulled back into the LV. A pigtail catheter was introduced into the left side for subsequent angiography. TEE confirmed the size of the defect, which was 10×18 mm. An Amplatzer muscular VSD device of 12 mm was selected based on TEE measurement and was loaded into the delivery sheath. The LV disk was then deployed into the LV aneurysmatic pouch and the system was pulled back against the defect. After ensuring a good position with TEE and angiography, the right ventricle (RV) disk was deployed successfully. After confirming a good position and a good sealing of the defect, the device was successfully released. The repeat right and left heart catheterization revealed improved PA pressure, where it dropped from 55 mm Hg to 36 mm Hg with slight improvement in the left ventricular end-diastolic pressure. The repeat LV angiogram revealed only a trivial shunt. The total procedural time was 105 minutes and the total fluoroscopic time was 31 minutes. The total amount of contrast used was 150 mL (2 mL/kg). Following the procedure, she developed contrast-induced nephropathy and needed a short period of continuous renal replacement therapy. The follow-up echocardiography

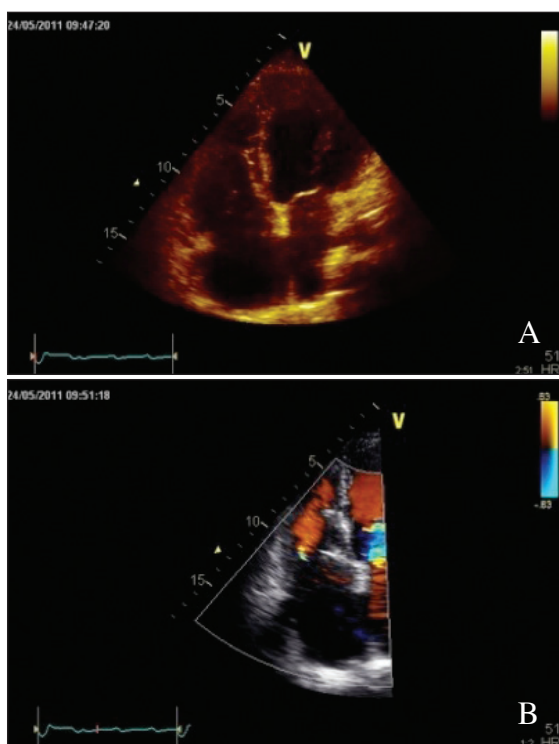


Figure 3. A) Apical view after 2 years showing well seated device. B) Color Doppler, Apical view after 2 years showing trivial shunt.

(Figure 2) showed a well-seated device with a minimal shunt and low normal LV systolic functions. The patient had a good recovery and was discharged in a stable condition within 2 weeks after the procedure and was put on proper anti-failure medications. She was followed in the heart failure clinic for 3-and-a-half years after the procedure, and a repeat echo showed no deterioration in the LV function, normal RV size and function, and small basal inferior VSD with left-to-right shunt (Figure 3). She developed right bundle branch block and atrial flutter during the follow-up and started on anticoagulation. She is almost asymptomatic with satisfactory quality of life.

DISCUSSION

Transmural tissue necrosis causes loss of structural integrity with a dynamic perforation of the interventricular septum at the interface of necrotic and non-necrotic myocardium.¹ Anterior wall MI complicates with shunt across the apical septum while inferior wall MI predisposes to shunt across the basal septum.² Our patient is diabetic and initially presented with inferior wall MI complicated with inferobasal VSD, which is a known complication of such MI. Her symptoms developed on the third day, which is the usual

time of such complications although it can be early or late. Current guidelines recommend immediate surgical VSD closure irrespective of the patient's hemodynamic status to avoid further hemodynamic deterioration.⁵ Early surgical VSD closure along with CABG was carried in our case according to the guidelines. However, many surgeons recommend surgical VSD closure after a delay of 3 to 6 weeks on the basis of retrospective studies to allow scarring of the surrounding tissue to occur, letting firmer anchoring of suture and patch material and resulting a surgical outcome⁶ but it more reflects natural selection bias. Delaying surgery even in hemodynamically stable patients increases the risk of unpredictable and catastrophic deterioration, and multiple studies support rapid intervention.⁷ A residual shunt persists in 10% to 37% of patient after surgery because of an overlooked additional defect or development of a new VSD.⁴ In our case patch dehiscence is the likely cause for residual shunt. There is limited data to develop consensus regarding the use of TCC of post-MI VSD. It has been used for a primary closure in the acute phase⁶ but has witnessed a less success rate, and when used for the closure of residual VSD after primary surgical intervention⁸ or in the subacute and chronic stages, it achieved a good success rate. The TCC of the VSD was initially reported in patients considered a too high risk for surgical repair because of their recent post-AMI status, advanced age, severe CAD, hemodynamic instability, and added comorbidities. The clinical experience is still limited. The TCC achieved a good success rate when used for the closure of residual VSD after primary surgical intervention, or in the subacute and chronic stages.⁹ In our case she had a trivial shunt across the device, which did not worsen during the follow-up and neither the patient condition deteriorated. Her symptoms of dyspnea and orthopnea became much improved. Device migration, trapping of mitral or tricuspid chordate, encroachment upon adjacent structure, and inadequate defect closure are the important complications of the procedure.⁹ After the initial report by Lock et al in 1988 who used the Rashkind double umbrella,¹⁰ various devices have been used including the clamshell occluder, the cardioseal, and the Amplatzer septal occluder device.¹¹ The Amplatzer device was also used in our case with a successful result for closure of residual VSD after surgical closure post-MI and has a good long-term survival. The surgical patch may provide better support for the occluder device than the necrotic tissue, resulting in a better procedural success.¹²

In conclusion, percutaneous transcatheter VSD closure is a promising technique for managing residual

shunt after the surgical repair of postinfarction VSD with a good success rate in the subacute or chronic phase. Further developments in devices and delivery techniques are required to optimize interventional outcomes.

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