

Transcatheter Closure of a Large Postinfarction Ventricular Septal Defect Complicated With Perforation of Ventricular Aneurysm

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Abstract

Ventricular septal defect concurrent with left ventricular aneurysm is unusual in postinfarction patients. The complex anatomy is challenging for occluder deployment in transcatheter closure and associated with high risk of aneurysm perforation. We present a case of transcatheter closure of a large postinfarction ventricular septal defect with an Amplatzer septal occluder in a 71-year-old woman, complicated with aneurysm rupture and cardiac tamponade and corrected with surgical repair.

Keywords: Left ventricular free wall rupture; Myocardial infarction; Ventricular septal defect; Cardiac tamponade; Amplatzer septal occluder; Transcatheter closure

Introduction

Ventricular septal defect (VSD) is a devastating complication of Acute myocardial infarction (AMI), occurring in less than 0.2% of patients, and patients with postinfarction VSD have extremely high mortality rate of 94% if only treated medically than those treated with surgical repair (47%) [1]. Transcatheter closure of VSD has been suggested to be a viable alternative to traditional surgical repair as a permanent treatment or a bridge to definite surgery [2,3]. Though anecdotal reports have demonstrated occluder implantation in small or medium postinfarction VSDs adjacent to an aneurysm [4], successful closure of large VSDs (≥ 15 mm) have not been previously reported. We present a case of transcatheter closure of a large postinfarction VSD complicated by apical left ventricle (LV) aneurysm rupture after device deployment which was confirmed by both realtime transesophageal echocardiography (TEE) and fluoroscopy and immediately corrected with primary suture repair. Written informed consent was obtained from the patient for publication of this article.

Case Report

A 71-year-old woman with past medical history of non-insulin-dependent diabetes mellitus presented to the emergency room with acute-onset chest pain and exertional dyspnea for two weeks. Electrocardiogram showed ST elevation in V2 to V5, signs of anteroapical AMI. Urgent coronary angiography revealed isolated disease of Left anterior descending coronary artery (LAD) with nearly total occlusion and a collateral circulation from posterior descending coronary artery without evidence of VSD. A drug-eluting stent was implanted in the middle portion of LAD. The next day, TEE revealed an apical VSD developed with left-to-right shunting, moderate pulmonary hypertension (right ventricular systolic pressure 55.7

mmHg), and LV systolic dysfunction evidenced by a kinetic apical anterior and middle anteroapical segments, apical aneurysm formation, and ejection fraction of 39%. Heart failure still persisted after coronary intervention but inotropic treatment was not necessary. In respect to the VSD close to the non-functioning apical aneurysm and viable myocardium might be greatly compromised after traditional surgery, patient and her family raised concern about high risk and postoperative mortality of surgical repair. Therefore, percutaneous transcatheter closure of VSD was scheduled 16 days after MI for scar formation in infarcted myocardium.

At the hybrid catheterization laboratory, standard anesthetic monitors and direct arterial blood pressure monitoring via the radial artery were applied before induction. After balanced general anesthesia, continuous TEE monitoring showed a large apical VSD lack of apical myocardial rim and adjacent to an apical aneurysm (Figures 1a and 1b). Under fluoroscopic and TEE guidance, the VSD was crossed from LV using a 6 French right diagnostic Judkins catheter introduced from the right femoral artery percutaneous sheath (Figure 2a). A 0.035 inch, 260 cm long Terumo guidewire was advanced into the pulmonary artery, then snared using an Amplatzer GooseNeck snare and exteriorized through a right internal jugular vein (RIJV) sheath. The VSD diameter was 15.8 mm, measured with TEE and confirmed with an Amplatzer sizing balloon (Figures 1a and 2b), and a 30 mm Amplatzer muscular VSD occluder was selected. A 10 French Amplatzer sheath was placed from the RIJV through the VSD into the LV using a veno-arterial wire loop. The septal occluder was advanced, deployed and positioned well with trivial residual shunting (Figure 2c). However, profound hypotension happened and TEE imaging revealed pericardial effusion and tamponade (Figure 1c). A pericardial window was emergently created through incising left chest wall, but active bleeding from the cardiac perforation persisted with compromised hemodynamics. Median sternotomy and longitudinal pericardiotomy were performed, and the suspected guidewire-related small perforation in the aneurysm was directly closed with 2-0 polypropylene pledgeted mattress sutures. TEE demonstrated no displacement of the Amplatzer septal device (Figure

1d). The patient was transferred to the Cardiac Surgical Intensive Care Unit (ICU) after surgery and was successfully discharged on postoperative day 45.

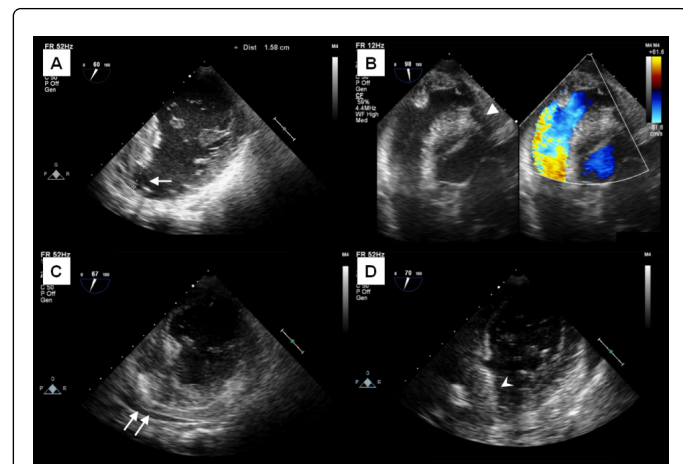


Figure 1: TEE: (a) modified mid-esophageal mitral commissural view showed the VSD concomitant with an apical aneurysm (arrow). (b) Deep-transgastric long-axis view showed an apical VSD (arrowhead) with left-to-right shunting. (c) Modified mid-esophageal mitral commissural view demonstrated pericardial effusion (double arrow) after LV wall perforation. (d) Modified mid-esophageal mitral commissural view showed that pericardial effusion was relieved after pericardiostomy and surgical repair without Amplatzer occluder displacement (hollow arrowhead).

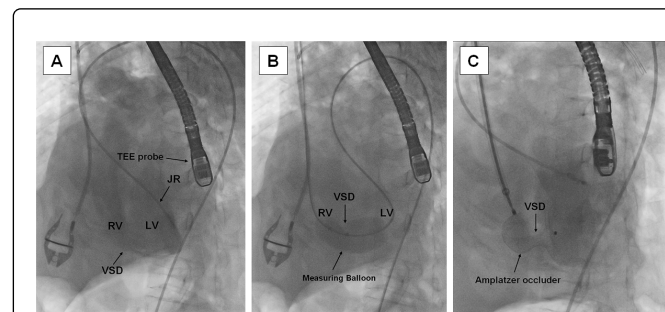


Figure 2: (a) Fluoroscopic left anterior oblique (LAO) view showed 6 French right Judkins catheter (JR) in the left ventricle (LV). (b) LAO view showed wire loop through the VSD with measuring balloon. (c) Right anterior oblique view showed the Amplatzer occluder deployed across the VSD.

Discussion

Large and complex postinfarction VSD usually requires surgical repair due to risk of device embolization or residual VSD after device implantation [5,6]. Although the American College of Cardiology and the American Heart Association guidelines reserved class 1A recommendation for immediate coronary artery bypass graft surgery and VSD surgical repair in AMI complicated with postinfarction VSD, it might not apply to all patients especially when they are high-risk, comorbid patients [7]. We concerned, in the present case, about the LV reservoir function after surgical repair of VSD since the VSD was

adjacent to the non-functioning apical aneurysm and the viable myocardium would be injured after surgery, and both poorly controlled diabetes and impaired pulmonary function would increase the major complications and mortality associated with surgery and prolonged ICU/hospital stay. After discussion, the patient and her family determined to receive the less-invasive transcatheter closure of VSD instead of traditional surgery.

Postinfarction VSD occurs in the apicoseptal wall more frequently after anterior infarction [8]. In addition, approximately 70 to 85 percent of LV aneurysms occurring following AMI with total occlusion of LAD are located in the apical or anterior walls [9,10]. Both postinfarction VSD and LV aneurysm formation contribute to hemodynamic impairment and are associated with poor prognosis in patients after MI. In the present case, the VSD was large and lack of enough muscular rim at the apical region of the VSD circumference, and the 30 mm Amplatzer muscular VSD occluder was well positioned with trivial residual shunt. Unfortunately, LV free wall rupture occurred at the adjoining aneurysm and was highly suspected to be caused by catheter or guidewire during transcatheter procedure. The site of the ischemic VSD was surrounded by necrotic tissue, and we believe that attempts to pass the closure device through the VSD may increase the size of the rupture and tear residual fragile tissue.

Catheter-related cardiac perforation is a devastating complication of percutaneous transcatheter intervention and associated with exceedingly high mortality due to the preexisting pathology of AMI patients. In an analysis of 29 patients undergoing transcatheter closure of postinfarction VSD, 3 patients developed LV rupture and died [11]. Early detection and immediate management are the keys to reduce hazard of death. Realtime TEE proves to be a valuable tool in both procedural guidance and recognition of complication during percutaneous transcatheter closure of postinfarction VSD [12]. It not only confirms the appropriate positioning of occluder but also detects residual shunting after device deployment. Compared with fluoroscopy, TEE offers continuous monitoring without the use of contrast dye. Besides, color Doppler TEE may be sensitive enough to detect small cardiac perforations in the immediate post-deployment period and prevent delayed cardiac tamponade.

In addition, surgical repair is an effective and well-established therapeutic technique for cardiac perforation. Although sporadic reports have demonstrated successful transcatheter closure of iatrogenic cardiac perforation during percutaneous procedures in selected patients, this technique can be challenging in large-sized and complex lesions [13,14]. Therefore, we suggest that percutaneous closure of VSD should be performed with on-site surgical standby at catheterization laboratory in complicated cases.

Summary

Surgical closure of ischemic VSD may be infeasible in selected patients of AMI. The postinfarction VSD adjacent to ventricular aneurysm presents challenges for occluder deployment and is at risk of iatrogenic cardiac perforation during or after transcatheter procedures, which necessitates early detection and prompt management. Realtime TEE monitoring and surgical standby are imperative in high-risk patients in case of catastrophic cardiac events. Standard surgical repair rather than percutaneous intervention might be more appropriate for patients having nearby aneurysmal myocardium to decrease the risk of aneurysm rupture.

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