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Hybrid closure of Post Infarct ventricular septal rupture with perventricular Amplatzer Septal occluder reinforced with a composite patch

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ABSTRACT:
We describe a rare interventional procedure in which an 77-year-old gentleman underwent a successful per-op hybrid closure of post myocardial infarction ventricular septal defect and coronary artery bypass grafting.

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Background

Before the advent of thrombolytic therapy and acute percutaneous coronary artery interventions, post-infarction VSD developed in approximately 1% to 3% of patients(1). Following introduction of these therapeutic interventions, the frequency has been substantially reduced to less than 0.5% of patients(1,2).

Post-infarction VSD is usually located in the anterior or apical portion of the ventricular septum (≈60% of cases) as a result of a transmural anterior MI(3). About 20% to 40% of patients have a VSD in the posterior portion of the ventricular septum as a result of an inferior MI. Ventricular septal rupture usually occurs as a complication of a first acute MI(4). Also, a well-developed collateral coronary circulation is uncommon in hearts with a post-infarction VSD(5). The defect is generally associated with complete occlusion (rather than severe stenosis) of a coronary artery, usually the left anterior descending coronary artery(5). Important stenoses often coexist in the right coronary artery system. VSDs may be multiple, and rather than occurring simultaneously, they may develop separately several days apart. The importance of concomitant right ventricular infarction in patients with post-infarction VSD is now evident. For many years, evidence of RV dysfunction was thought simply to represent poor "adaptation" of the RV to the sudden increase in pulmonary blood flow imposed by the post-infarction VSD. Accumulated information indicates that actual infarction of the inferior RV wall, or at least severe ischemia of that area, is responsible for the dysfunction(6). A posterior VSD, in particular, may be accompanied by mitral valve regurgitation secondary to papillary muscle infarction or ischemia. In about 40% of patients who survive the early period after ventricular septal rupture, the remainder of the infarcted septum and adjacent ventricular wall may become aneurysmal(7).

Case Presentation

77 years old, male, presented with complaints of chest pain radiating to left arm, and sweating since 7 days. On evaluation he was diagnosed to have Acute Inferior wall MI with a VSR. Coronary angiography revealed significant triple vessel disease. 2D ECHO was suggestive of a 15 mm ventricular septal rupture with posterior wall hypokinesia, LVEF=55%. LA dilated[37m2] and LVEDD[54mm]. His coronary angiography revealed 95% stenotic lesions in left anterior descendent and obtuse marginal. Pre-operative elective IABP was inserted to support the hemodynamics.

Surgical Technique

Full midline sternotomy was performed and Aorto Bicaval bypass was established. Myocardial protection was achieved by both antegrade root cardioplegia and retrograde

cardioplegia. Distal anastomoses were done to LAD and OM and apical left ventriculotomy was done. As the defect was located in deep posterobasal region, a full left posterior ventriculotomy was done for a better operative field. VSR was measured to be 40 x 30 mm and the surrounding myocardial tissue was oedematous and fragile.

Multiple 4-0 pledgeted prolene sutures were taken along the margins of the defect with pledgets towards the RV side of the defect, a 36mm Amplatzer ASD Occluder was placed and a composite patch was sewn over the device from LV side to close the defect. Patient was cooled to 28 degrees, CPB time was 226 minutes and Aortic Cross clamp time was 125 minutes. Patient came off bypass on moderate intropic support. Post CPB, intra-op ECHO was suggestive of no residual flow across the defect.

Discussion

Hospital mortality after VSR repair is approximately 30-40%(8). Risk of death is greatest immediately after myocardial rupture and then gradually declines. Women and the elderly may be more susceptible(9).
The joint American Heart Association/American College of Cardiology (AHA/ACC) 2004 Guidelines recommend emergent repair of the VSD with concurrent coronary artery bypass grafting, as indicated, irrespective of hemodynamic status, with no change in this class I recommendation in the 2011 ACC/AHA guideline for coronary artery bypass surgery(10).

VSDs located in the posterior septum are more difficult to expose and repair. The heart is lifted out of the pericardium with traction on the LV apex. The defect is approached through a vertical incision in the infarcted LV myocardium. If the VSD is relatively small, the necrotic tissue can be excised, including the infarcted free wall of both the RV and LV, often with the overlying occluded posterior descending coronary artery. The VSD patch (collagen- or gelatin-impregnated polyester or bovine or autologous pericardium) is placed on the LV side of the septum and secured using mattress sutures of No. 2-0 poly- ester, with pledgets placed on the RV side of the septum. If little or no free wall myocardium has been excised, LV and RV edges are approximated, incorporating the septal patch and two strips of PTFE felt. A large defect in the free wall requires a second patch(11).The free edge of the septal patch is sutured to the free wall of the LV with interrupted mattress sutures of No. 2-0 polyester using pledgets on the patch and a strip of PTFE on the ventricular wall. The patch for closure of the RV is attached to the septal patch already in position and to the free wall of the RV. Pledgets of felt are placed on the inner surface of the RV, and a strip of felt is placed on the outer surface.

Lock and colleagues achieved successful percutaneous transcatheter closure of postinfarction VSDs in four patients with cardiogenic shock using the Rashkind double-umbrella device(12) The device embolized to the pulmonary artery in one patient, and the remaining three patients died with evidence of increased shunting across the defect over the ensuing several days.

Use of percutaneous closure as the definitive treatment for postinfarction VSD has been reported by Maltais and colleagues using the Amplatzer septal occluder device(13). Early mortality was 42% among 12 patients. One patient (8%) had a residual septal defect.

A residual VSD has been noted early or late postoperatively in 3% to 40% of patients(14,15). Residual VSD may be caused by reopening of a closed defect, presence of an overlooked VSD, or development of a new VSD during the early postoperative period.

The higher mortality associated with repair of defects located inferiorly in the septum reported in some series probably relates to the higher prevalence of important right coronary stenosis and RV dysfunction in this group(16). Mortality may also be related to the greater complexity of posterior repairs and to more frequent involvement of the mitral valve(16).

In our case, we did two bypass grafts to LAD and OM. Then we performed a total left posterior venticulotomy to visualise the poster basal PI-VSR. Pledgeted sutures with pledgets towards RV side were placed along the margins of the defect. An Amplatzer ASD septal occluder of 36mm was introduced from LV side into the defect and the device was additionally sewn with a composite patch from LV cavity and further reinforced by the sutures alongside margins of the defect.

Conclusion

Perventricular device closure of post-myocardial infarction VSDs (PI-VSD) appears to be a safe and effective method to close PI-VSD. This approach has established itself for management of high risk posterobasal VSDs. It has advantages over both surgical and transcatheter techniques. With this approach, immediate complete closure of PI-VSD with complete coronary revascularization is feasible without the ill effects of cardiopulmonary bypass or challenges of arterio-venous looping. However, many more cases will be required before this hybrid procedure becomes as established a procedure as it has become for congenital muscular VSD closure. Post MI VSR carry high mortality rates and approach to posterobasal defects are very difficult to repairing relation to its close proximity to the mitral valve apparatus and location. Hybrid closure of the defect provided extra-strength and minimised chances of device embolisation or patch dehiscence in view of oedematous and infarcted myocardium along the defect.

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