Perventricular device closure of post myocardial infarction ventricular septal defect: Can it combine best of both worlds!

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ABSTRACT

Introduction: Post myocardial infarction ventricular septal defect (VSD) is a rare but dreadful complication of acute myocardial infarction. Current management of this complication has high morbidity and mortality rates. A hybrid approach (perventricular device closure) to high risk congenital muscular VSD has shown promising results. We report first case of a perventricular device closure of post infarct VSD by Amplatzer post infarct muscular VSD device. Case Report: A 52-year-old male was referred to us for rapidly progressive dyspnea. He had anterior wall myocardial infarction, complicated by post infarct VSD. His coronary angiography revealed 90% lesion in left anterior descending (LAD) artery. Perventricular device closure of VSD (Amplatzer post infarct muscular VSD device) and graft to the LAD were performed using a beating heart technique. His postoperative stay was complicated by an enlarged secondary post infarct VSD. It was closed by percutaneous technique. He was discharged in stable condition. He is in compensated heart failure at 1 year follow up. Conclusion: Perventricular approach to congenital muscular VSD is an established procedure. Applying this approach to post infarct VSD can also effectively manage this dreadful complication. Combination of surgical and percutaneous techniques might be less traumatic with better outcome.

Keywords: Closure, Device, Myocardial Infarction, Perventricular device, Ventricular septal defect (VSD)
INTRODUCTION

Acquired ventricular septal defect (VSD) is one of the three major mechanical complications of acute myocardial infarction (AMI), the other two being acute mitral regurgitation and rupture of the ventricular free wall. With the advent of early reperfusion strategies and adjunct medical therapy, the incidence of this complication has significantly decreased to <1% of cases but remains associated with a high morbidity and mortality. Although this decrease is encouraging, both early and long-term prognosis after AMI-related VSD remain unsatisfactory. Medically managed patients with post-myocardial infarction VSDs (PI-VSD) have 30-day mortality rates as high as 94% [1]. Given this high mortality rate, surgical closure has traditionally been advocated as the preferred treatment strategy. But even in surgically treated patients, mortality remains high, with reported rates ranging from 23–81% [1–3]. Even the incidence of a large residual shunt and re-rupture after surgery reaches up to 10–20% [3]. With advances in cardiac interventional techniques and devices, transcatheter closure of PI-VSD has become an alternative or bridge to surgical repair for patients with PI-VSD in the last decade [1, 3, 4]. However, both these methods do have their limitations in management of PI-VSDs [4]. A hybrid approach (perventricular device closure), performed both by surgeons and cardiologists has been described for congenital muscular VSDs [5, 6]. Although it is safe and effective in high risk cases, it has not been widely tried in PI-VSD cases. Only two cases of PI-VSD have been managed with hybrid approach till date [7]. We report a case of perventricular closure of PI-VSD with an Amplatzer post-infarct muscular VSD device. To the best of our knowledge this is the first reported case of perventricular device closure of PI-VSD by an Amplatzer post-infarct (PI) muscular VSD device.

CASE REPORT

A 52-year-old male without previous history of hypertension, diabetes mellitus and smoking, was referred to our hospital for rapidly progressive dyspnea. He was symptomatic for last 15 days, symptoms started with chest pain and shortness of breath. His dyspnea gradually worsened to NYHA class IV at the time of admission.

Clinically, he was in congestive heart failure with a pulse rate of 110/minute, blood pressure of 100/70 mm Hg, a respiratory rate of 30 per minute, bilateral basal crepitations in lungs and a grade III pan systolic murmur at left lower parasternal area.

Investigations: Blood investigations showed mildly deranged renal and liver function tests. His electrocardiogram (ECG) was consistent with evolved anterior wall myocardial infarction. Transthoracic echocardiogram (TTE) showed mid and distal interventricular septal akinesia and hypokinesia in apex and anterolateral segments, moderate left ventricular dysfunction and a large muscular VSD of 10 mm size in the distal part of the septum. One additional small muscular VSD was seen anterosuperior to the larger VSD. Coronary angiography (CAG) was performed next day which revealed 90% lesion in left anterior descending (LAD) artery involving ostium and proximal part. Other vessels were normal.

Treatment: The medical treatment was optimized with inotropic support, vasopressor drugs and diuretic therapy. He was put on intra-aortic balloon pump (IABP) support post coronary angiography. Perventricular device closure of VSD and graft to LAD were planned next day. Next day, for the procedure, the patient was anesthetized as per coronary artery bypass graft surgery (CABG) protocol and the chest and pericardium were opened in operation theatre. Using a beating heart technique, a Teflon pledgedt purse-string suture with Ethibond 2/0 (Johnson and Johnson, USA) was taken on right ventricle (RV) free wall, about one and half inches away from the RV apex. The site was selected using transesophageal echocardiography (TEE) guidance and after discussion with surgeon. A direct puncture was made in middle of the purse-string with an 18-gauge Jelco (Smiths Medical, Italia) in the direction of VSD. The needle was removed and a 0.035” short wire (Terumo, angled tip, RADIFOCUS, Terumo Corporation, Tokyo) was advanced to the LV over the wire. The dilator and wire were removed. A 16-mm Amplatzer post-infarction muscular VSD device (St. Jude Medical, Inc., USA) was selected and was advanced through the sheath. The left sided retention disc was delivered in operation theatre. Using a beating heart technique, a Teflon pledgeted purse-string suture with Ethibond 2/0 (Smiths Medical, Italia) was positioned in the left ventricle (LV) under TEE guidance (Figure 1). A 10-French (Fr) Terumo sheath (RADIFOCUS, Terumo Corporation, Tokyo) was advanced to the LV over the wire. The dilator and wire were removed. A 16-mm Amplatzer post-infarction muscular VSD device (St. Jude Medical, Inc., USA) was selected and was advanced through the sheath. The left sided retention disc was delivered in the mid LV cavity. The waist was then delivered and the entire assembly (sheath, device and cable) was pulled back to the interventricular septum. The right ventricular disc was delivered by active pushing as there was no sufficient space to passively deliver it, as the distance between septum and right ventricular free wall was short. These steps were performed under TEE guidance (Figure 2). The position of the device and residual shunt were checked on TEE. There was no significant shunt across the device and the position of the device was stable. There was no significant shunt across the additional VSD site at this point of time. The device was released from the delivery cable with counter clockwise rotation of pin vise. Thereafter a saphenous venous graft to LAD was performed using a beating heart technique. The absence of residual shunt was reconfirmed and the purse-string was tied after removal of Terumo sheath. The Ethibond...
purse-string suture was reinforced with a Prolene 3/0 (Johnson and Johnson, USA). The sternal wound was closed as per standard technique. The patient was shifted to intensive coronary care unit (ICCU) with IABP and ventilatory support. He was extubated on 2nd day and gradually weaned off from IABP support in next 4 days. A grade III pan systolic murmur persisted. There was no residual shunt across the device but shunt across the additional VSD was present. He had hepatic and renal dysfunction which required intensive treatment and he was shifted to ward on 5th postoperative day. His renal parameters gradually returned to normal and hepatic enzymes showed gradual downward trend. He required intravenous diuretics apart from usual postoperative medicines. However on 20th postoperative day, his clinical condition deteriorated considerably. He was put on intravenous inotropic and noninvasive ventilatory (BIPAP) support again. A repeat TTE revealed an enlarged VSD (size 10 mm) anterosuperior to the device. In view of rapidly deteriorating symptoms, percutaneous device closure of VSD was planned and was performed on the same day. The VSD was closed using an 18 mm Amplatzer muscular VSD (St. Jude Medical, Inc., USA) device using standard technique (Figure 3A–B). There was a residual shunt across the VSD but the step up in oxygen saturation was less than 5%. He was then shifted to ICCU and thereafter to the wards next day. He was discharged in stable condition after 5 days on aspirin, statin, warfarin and anti-failure treatment.

Follow up: A 12-month follow-up has been completed. The patient remains in NYHA class II symptoms with LVEF 30 %. He has a small residual shunt and has two devices across the interventricular septum (Figure 3C–D).

DISCUSSION

PI-VSD is a rare but serious complication of AMI. PI-VSD usually occurs 2–8 days after the infarction and often precipitates cardiogenic shock. The size of the defect determines the magnitude of the left-to-right shunt and consequently the hemodynamic deterioration, which affects survival. Compared to patients with AMI without VSD, patients with VSD are older, more likely to be women, has increased rate of chronic renal disease, congestive heart failure and cardiogenic shock, the absence of a history of angina or myocardial infarction, and severe coronary stenosis or total occlusion without compensatory collateral circulation and are less likely to be hypertensive or diabetic [3].

The pathogenesis of PI-VSD reflects two different types of rupture. The first, a simple rupture, is a direct through-and-through defect. Conversely, complex ruptures are believed to result from tracking of blood as it dissects through the septum with left ventricular entry sites remote from right ventricular exit sites – these tracks then enlarge over time due to the pressure gradient between

Figure 1: Puncture site on RV (Black arrow) with Terumo wire (green arrow).

Figure 2: Deployment of Amplatzer post-infarct muscular device by perventricular approach under transesophageal echocardiography (TEE) guidance. (A) Left to right shunt across the ventricular septal defect (VSD), (B) Terumo wire in left ventricle (yellow arrow), (C) Terumo wire in left ventricle across the defect (yellow arrow), and (D) No left to right shunt across the defect after device deployment (Device: green arrow)

Figure 3: (A) Left to right shunt across the secondary VSD, (B) No significant shunt after percutaneous device closure, and (C, D): 1: Device deployed by perventricular approach. 2: Device deployed by percutaneous approach.
the left and right ventricle. Multiple defects are found in 5–11% of cases [2, 8]. Incomplete closure of residual or secondary defects can account for postoperative recurrences. Fortunately, most residual shunts tend to be physiologically tolerated and spontaneous closure has been reported. Operative re-intervention is associated with a >60% mortality [2] and surgery is reserved for patients in heart failure failing medical management or those with large shunts (Qp:Qs > 2.0) [2, 8].

The mortality of PI-VSD is very high. 50% of these patients die within one week and about 85% within two months [2, 9]. Attempts to stabilize the patient’s condition with medical therapy often fail because most patients have a rapid deterioration and subsequently die [3]. The dismal prognosis of this subgroup of patients with AMI has elicited aggressive surgical intervention. The mere presence of a PI-VSD is considered an indication for surgery with the majority of patients undergoing urgent or emergent operative intervention. The primary goal of VSD closure is to reduce the end-organ damage from the combined insults of acute right ventricular overload/failure and systemic cardiogenic shock [2]. 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 [1, 10]. Current surgical management of this uncommon catastrophe includes: 1) deferring operation, if possible, until three weeks after infarction, 2) cardiac support with intra-aortic balloon pump insertion to allow preoperative definition of coronary and ventricular anatomy of patients with hemodynamic deterioration, and 3) a transinfarct incision with prosthetic replacement of excised ventricular free wall or septum, if necessary, and 4) possible repair of associated coronary or mitral valve pathology [8, 9]. Cardiac surgery is considered the gold standard in the management of these defects; however, its main limitation is that it carries a high risk of perioperative mortality and postoperative morbidity.

Percutaneous transcatheter closure of PI-VSD is an alternative method of repair [1, 3]. It is a less invasive option and allows immediate complete closure after initial hemodynamic stabilization. It has become an alternative or bridge to surgical repair for patients with PI-VSD. Immediate reduction of the left-to-right shunt, even if the VSD is not completely closed, may stabilize the patient enough to function as a bridge to surgery [1]. Specially designed devices for the closure of PI VSDs have also become available. The difficulty of transcatheter closure of ventricular septal rupture is how to push the catheter through the septum defect without injuring the surrounding friable myocardium [3]. As the site of septal rupture in patients with AMI is surrounded by fragile necrotic tissue, attempts to pass the closure device though the site may increase the size of rupture [8]. In addition, the occluders are usually difficult to fix because of the presence of the friable necrotic myocardium tissue around the defect [3]. Moreover, current interventional reports are mainly restricted to VSD closure in the sub-acute or chronic setting, or for residual shunts after initial surgical closure [1]. Due to scarcity of reports in the literature, there is limited data regarding survival data; however, the few reported series have shown an overall mortality rate of 44–60% within one year [11]. Furthermore, non-inferiority to surgery has been demonstrated in one case series [4]. Long-term follow-up studies are lacking, and thus long-term mortality has yet to be discerned.

Perventricular device closure for congenital muscular VSD (m-VSD) is a new approach. It is performed by both surgeon and cardiologist in hybrid suit or operating room combining catheterization and surgical techniques [5, 6]. Thakkar et al. concluded in their series of 24 infants who underwent perventricular device closure of mVSD that in selected high-risk infants, perventricular device closure of isolated mVSD is effective and may either substitute or complement the conventional surgical closure depending on the performance of institutional pediatric cardiac surgery program. The procedural safety can certainly be improved with more precautions for preventable complications. Until specifically designed hardware is available, very large defect or defects extending into inlet, posterior or apical septum are not suitable for perventricular closure [5]. The major advantages of hybrid approach are: from surgeon’s view; i) easy accessibility of m-VSD even in difficult locations ii) no palliative pulmonary artery banding or ventriculotomy required to close the apical m-VSD iii) no ill effects of CPB (cardiopulmonary bypass); from cardiologist’s view; i) no limitation for vascular access and sheath size ii) no hemodynamic instability due to arterio-venous looping iii) septum can be approached from anterior (perpendicular angle) but, not from a lateral (tricuspid valve) plane [6]. Thus, this hybrid approach appears to combine the positives from both surgical and percutaneous methods. Although this approach has become popular to close high risk congenital muscular VSDs, it has not been attempted extensively for PI-VSD. Only 2 cases of PI-VSD have been closed by this approach till date. Love et al has used this approach to close PI-VSD by Amplatzer septal occluder [7]. In our case the PI-VSD was closed with an Amplatzer post-infarct muscular VSD device. Previous experience with congenital VSDs has found the Amplatzer system to have a higher success rate than other devices and extrapolation of this experience has led to the tendency to also use this family of devices for the closure of acquired post-AMI ventricular septal ruptures [1]. The Amplatzer muscular VSD occluder is a self-expanding, single-unit Nitinol device with incorporated polyester fabric that comprises two discs connected by a 7-mm long waist portion, compared with a 4-mm waist in the atrial septal defect (Amplatzer septal occluder) device. The device is sized between 4 and 18 mm by the diameter of the central waist, with the discs being 8 mm larger than this segment. The disc sizes are larger in Amplatzer septal occluder;
mm on left atrial side (distal disc) and 10 mm on right atrial side (proximal disc). These devices are secured onto a delivery cable and implanted via a 5- to 12-Fr diameter sheath. It is self-centering and permits several positioning attempts because it is retrievable before release. Specially designed devices for the closure of post-AMI VSDs have also become available. The Amplatzer PI muscular VSD device has larger disks and a longer waist (10 mm) than the muscular VSD Amplatzer device to accommodate the thicker adult interventricular septum. It is available in sizes of 16 to 24 mm in 2-mm increments, as determined by the diameter of the waist section. The size of the device in our case was 50 % more than the size of PI-VSD as measured on TEE. The device closure in our case was performed with standard technique but our deployment method of the device was different from Love et al. Love et al deployed the proximal disc on the exterior surface of RV to ligate RV free wall to VSD. It minimized the residual shunt across the PI-VSD in their cases. The RV puncture site was closed with pericardial patch in these 2 cases [7]. In our case the proximal disc was deployed inside the RV cavity by active pushing of the device (see case report). There was no residual shunt on TEE in our case. The puncture site on RV was closed with Ethibond purse-string sutures with reinforcement from Prolene 3/0 sutures. Thereafter, graft to LAD and wound closure was performed with standard technique. The patient showed gradual stabilization in clinical status after the procedure. During postoperative period, our patient deteriorated due to increase in size of the additional VSD. As there was no flow across it on the day of hybrid procedure, it was not closed at that time. During early postoperative period, the pan systolic murmur was present. However as the patient was clinically stable, he was managed conservatively. Intervention was needed as there was sudden increase in size of VSD. This second VSD was closed using transcatheter technique. The second VSD was closed with Amplatzer muscular VSD device. The patient improved considerably and was discharged in stable condition. He has completed 1 year follow up and he is in NYHA class II.

CONCLUSION

Perventricular device closure of PI-VSD appears to be a safe and effective method to close PI-VSD. This approach has established itself for management of high risk congenital muscular 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.

Author Contributions

Alok Ranjan – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
Kalpesh Malik – Acquisition of data, Drafting the article, Final approval of the version to be published
Manik Chopra – Acquisition of data, Analysis and interpretation of data, Final approval of the version to be published
Arool Shukla – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Dr. Kanaiyalal Patel – Acquisition of data, Drafting the article, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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