Original Research

Improved outcomes in the treatment of post-myocardial infarction ventricular septal defect with percutaneous TandemHeart® left ventricular mechanical circulatory support

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Abstract

Background

Post-myocardial infarction (MI) ventricular septal defect (VSD) is associated with 40% - 50% of peri-procedural mortalities; however, it is amenable to catheter-based therapies. We retrospectively investigated the impact of state-of-the-art bridging percutaneous left ventricular mechanical circulatory support (MCS) using the TandemHeart® (TH) ventricular assist device (VAD) on a patient with post-MI VSD.

Results

From July 2008 to March 2014, 23 patients were referred for treatment of post-MI VSD. Initially, 18/23 patients required MCS; 12 received an intra-aortic balloon pump (IABP), while 6 received initial TH support. Seven of the IABP patients later required TH support. Catheter-based device VSD closure was performed in 18 of the patients; however, three patients required conversion to conventional open cardiac surgical repair via VSD patch closure due to failure of the catheter-based approach. Five patients with TH underwent planned open cardiac surgical repair

due to an anticipated lack of suitability for catheter-based treatment. Results revealed that delayed closure after MI correlated with improved survival. Overall, 30-day and 6-month survival rates were 83% (19/23) and 70% (16/23), respectively.

Conclusions

Further, Qp/Qs ratios of <2.4 correlated with successful percutaneous VSD repair, and this assessment should be further explored as an assessment to inform clinical judgment in patients with post-MI VSD treatment.

Introduction

Mechanical complications of acute myocardial infarction (MI) – ventricular septal defect (VSD), mitral regurgitation (MR), and free wall rupture (FWR) carry a grave prognosis and contribute to substantial mortality and morbidity despite current therapies. However, these complications are historically uncommon, with a reported incidence of 1 to 2% in the pre-thrombolytic agent era.[1,2] Furthermore, in the era of early myocardial reperfusion via percutaneous coronary intervention and thrombolytic agents, the incidence of mechanical complications is even lower; the incidence of VSD with thrombolysis is as low as 0.2%.[3,4] In addition, with the advent of reperfusion strategies, the time to ventricular rupture is significantly shorter, as documented by the GUSTO1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries/1 Day) and SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock/16 hours) trials.[3,5]

While FWR carries the highest mortality, due to its presentation with acute cardiac tamponade and obstructive shock, VSD and MR continue to carry high mortalities and morbidities as well. Unlike post-MI papillary muscle rupture causing MR, both surgical and catheter-based treatment options exist for VSD. Broadly, two types of surgical approaches have been used for post-MI VSD. The first approach, developed by Daggett and colleagues,[6] involves trans-infarct left ventriculotomy, infarctectomy, and closure of the VSD, which typically uses a patch prosthesis. The second and more recent approach is credited to David and colleagues,[7-9] and it employs trans-infarct left ventriculotomy with an oversized left-sided patch prosthesis sutured to the left-sided septum and anterior left ventricular (LV) walls to "exclude" the defect and commit the infarcted septum to the right ventricular (RV) side of the patch.

With respect to surgical treatment, Arnaoutakis and colleagues, in the largest study of its kind, reported outcomes of surgical repair of post-MI VSD from the

Society of Thoracic Surgeons database.[10] In this study, 2,876 patients underwent post-MI VSD repair, with a perioperative mortality of 42.9%.[10] Patients undergoing treatment within 7 days of hospital admission had a 54.1% mortality rate, while the minority of patients who underwent delayed treatment had an 18.4% mortality rate. While these different survival outcomes may reflect improved hemodynamic and clinical conditions of the patients undergoing delayed VSD repair, the magnitude of the findings is striking, and suggests that, if feasible, physiological optimization both pharmacologically and via mechanical circulatory support (MCS) should be undertaken prior to VAD repair. In fact, this was proposed by Dr. T. David in his discussion of Arnaoutakis et al.[10] Consistent with this hypothesis, we recently reported improved outcomes of delayed post-MI VSD surgical repair following a period of TandemHeart® (TH, LiveNova, London UK) percutaneous ventricular assist device (pVAD) support. Using this approach, we observed a 25% mortality rate at six months.[11]

The current guidelines recommend emergent surgical repair of the VSD with concurrent myocardial revascularization via coronary artery bypass grafting, if indicated, irrespective of hemodynamic status.[12] However, the high mortality and morbidity, as well as an increasing experience with catheter-based device closure of congenital VSDs,[13,14] have demanded the development of a comprehensive reassessment. Our proposed multi-pronged strategy for post-MI VSD management is as follows: (1) hemodynamic optimization using pharmacotherapy and MCS in the form of either intra-aortic balloon pump (IABP) counterpulsation or when needed, TH pVAD support, and (2) delayed catheter-based device and/or open cardiac surgical closure. To investigate the effectiveness of this strategy, outcomes of patients who underwent this approach of bridging MCS to delayed definitive treatment were retrospectively reviewed and analyzed.

Methods

Between July 2008 and March 2014, 23 patients were referred to our Center in a large metropolitan medical center for the management of post-MI VSDs. None of the patients had undergone emergent catheter-based or surgical VSD closure. All referred patients with post-MI VSDs were included in this study, without exclusion. The adequacy of end-organ perfusion and the presence or absence of cardiogenic shock was assessed as our Center has previously outlined in the surgical cases series by Gregoric et al.[11] Pharmacological optimization, comprised of LV afterload reduction and inotropic support, was initiated in all 23 patients. Patients who did not stabilize and had less severe, low-systemic cardiac output syndrome/shock underwent initial percutaneous IABP placement. In contrast, those with severe shock either initially or despite IABP support underwent percutaneous trans-atrial septal left atrial (LA) and common femoral arterial cannulation for TH pVAD support described in Gregoric et al.[11] This algorithm is outlined in Figure 1.

The decision to implement TH versus IAPB was exclusively based on clinical judgment. The primary step was to achieve satisfactory cardiovascular status, as assessed by systemic arterial blood pressure, Qs, central venous oximetry, and clinical and laboratory indices of end-organ perfusion. The decision to wean from
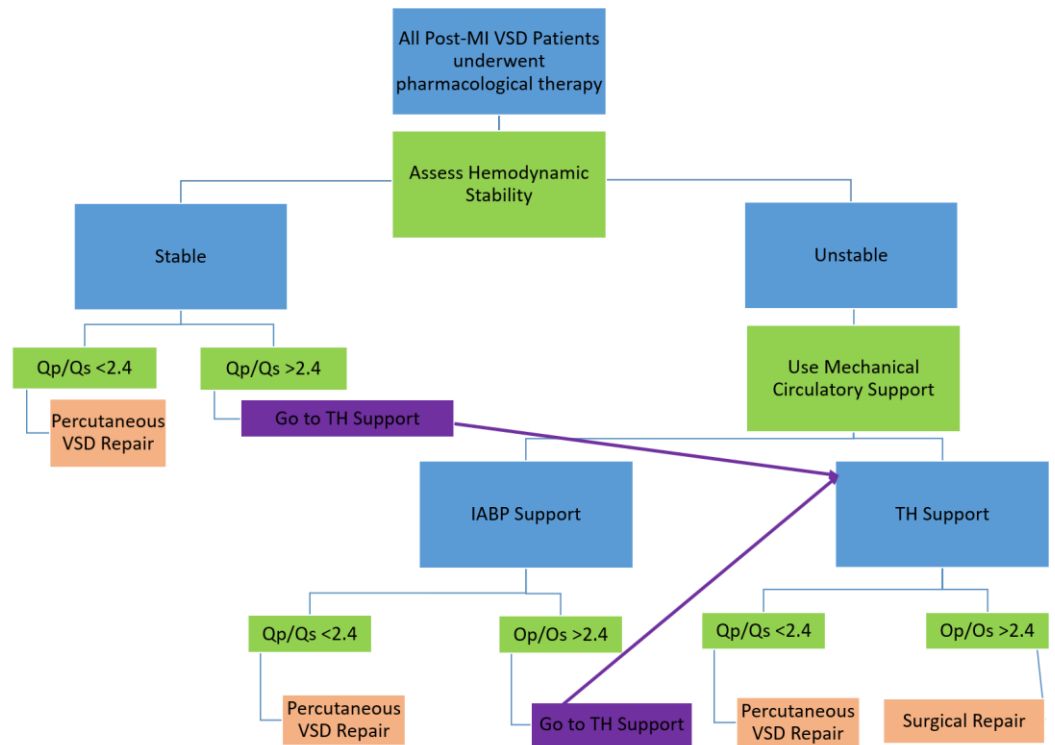
Figure 1. Strategy utilized for timing and therapeutic option in the management of post-myocardial infarction ventricular septal defects. MI (myocardial infarction); VSD (ventricular septal defect); IAPB (intra-aortic balloon pump); TH (TandemHeart™); Qp/Qs (pulmonary/systemic blood flow).

TH was attempted in the presence of adequate hemodynamics and an improved end-organ function at a pVAD flow rate of 2 L/day for two days. While on pVAD, the flow rate was adjusted to maintain mixed venous oxygen saturation >70 and mean arterial pressure >60 mmHg.

Qualitative and quantitative assessment of VSD physiology was accomplished using echocardiography and diagnostic right-sided cardiac catheterization with oximetry. Each of these modalities was used to determine pulmonary and systemic blood flow (Qp and Qs, respectively), thereby quantifying intracardiac shunt capability in all patients.

Patients who underwent successful percutaneous closure were implanted with the Amplatzer® post-infarction VSD occluder device, the Amplatzer® muscular VSD closure device, or the Atrial Septal Occluder (ASO) device (*all* St. Jude Medical, Inc., St. Paul, MN, USA). The implantation strategy included femoral arterial and right internal jugular venous access, with a 10Fr catheter from the systemic

venous side and a 7Fr catheter from the systemic arterial side. A Swan-Ganz catheter was navigated to the pulmonary artery to re-evaluate the degree of intracardiac shunting and was subsequently used to support a 300 cm 0.035" Storq wire (Cordis, Fremont, CA, USA) to cross the VSD from the RV. Retrograde access from the LV was obtained with a Judkins Right 7 Fr catheter (Cordis, Freemont CA, USA), and subsequently, the wire was exchanged for an endovascular snare (EN Snare Endovascular Snare System, Merit Medical, South Jordan, UT, USA). The Storq wire was then captured from the arterial access and served as a rail to deliver the Amplatzer® TorqVue 10Fr Sheath system (St. Jude Medical, St. Paul, MN, USA) in a "bodyfloss" fashion.

The Amplatzer® delivery sheath was railed across the tricuspid annulus and over the VSD in the LV. The Storq wire was externalized, the VSD occluder was loaded into the delivery system, and then it was deployed from the arterial to the venous side. Once properly positioned, intracavitary echocardiography was performed to confirm the occluder was stable, and there was no significant residual VSD.[15] Subsequently, the device was released, and the delivery sheath was withdrawn (Figure 2).

Figure 2. Intracavitary echocardiogram and fluoroscopic appearance of ventricular septal defect (VSD) device across ventricular septum after deployment. Left panel: Fluoroscopic image of the TorqVue delivery system and VSD occluder successfully deployed (arrow). Note transseptal cannula for TandemHeart® support in place (*). Right panel: Intracavitary echocardiogram of VSD occluder device (arrow) in place and released.
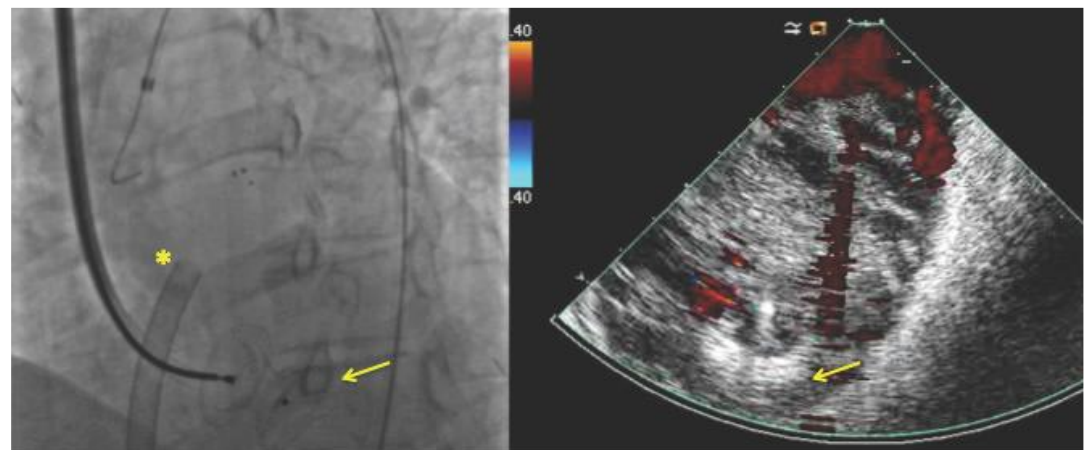
Patients with VSDs thought *a priori* not to be amenable to catheter-based device closure, or those who underwent a failed attempt at catheter-based device closure, underwent surgical repair as outlined in Gregoric et al.[16] In this subset of patients, a median sternotomy was completed, and repairs were performed using cardiopulmonary bypass (CPB) to provide total cardiac and pulmonary support with a bloodless operative field, via ascending thoracic aortic and bicaval

cannulation. The TH cannulae were maintained in place for postoperative LV MCS. Ascending thoracic aortic cross-clamping with the administration of cardioplegia (antegrade +/- retrograde) was used to render the operative field still. Trans-infarct left ventriculotomy was performed. The VSD was repaired using a woven Dacron patch sutured to a rim of healthy LV myocardium circumferentially using polypropylene suture. Depending upon intraoperative findings, either a standard or infarct exclusion patch repair was performed. After aortic cross-clamp release and initiation of inotropic and vasoactive support for the right-sided circulation, CPB was weaned and transitioned to TH pVAD support.

If the patient remained in cardiogenic shock despite TH assistance or after initial echocardiographic evaluation, defects were considered not amenable to percutaneous nor surgical closure, or in case of failed surgical closure due size or refractory biventricular failure TH was surgically explanted and a SynCardia temporary Total Artificial Heart (TAH) (SynCardia Inc., Tucson, AZ, USA) was implanted as a bridge to transplant, as previously described.[17]

After a successful percutaneous or surgical VSD closure, the TH was left in place for an additional 5-7 days to facilitate myocardial recovery and ensure end-organ perfusion and then surgically explanted, as previously described.[11]

Outcome parameters included: (1) technical success, which was defined as successful closure of the VSD without evidence of significant residual VSD or device embolization, and (2) survival at (a) 30 days, and (b) 180 days.

Eight patients included in the current manuscript were previously reported in Gregoric et al.,[11] as they were contemporaneous with our TH bridging and catheter-based device closure investigation.

Statistical Analysis

All statistical analyses were performed using Stata Version 12 statistical software (College Station, TX, USA). Categorical (frequency) variables were expressed as percentages or ratios. Continuous variables were expressed as means and standard deviation (SD) or medians with interquartile ranges (IQR). A comparison of Qp/Qs ratios between different types of MCS was performed with the two-sample Wilcoxon rank-sum (Mann-Whitney) test.

Results

Over the course of 5 years and 7 months, 23 patients were admitted for management of post-MI VSD. All patients in this group suffered VSD as a result of poor initial reperfusion or late presentation in the context of acute MI.

Patient baseline characteristics are reported in Table 1; briefly, this cohort includes 15 males and 8 females with a median age of 66 years (IQR 56 - 86). All patients had evidence of acute coronary syndrome a few days prior to the initial hospitalization. The date of MI and time to presentation could be identified in all patients except four due to the absence of typical signs and symptoms of angina.

Table 1. Baseline characteristics of patients in the entire group.

	N=23
Age, y	64 ± 14
Men (%)	15 (65)
Women (%)	8 (35)
EKG infarct location	
Anterior (%)	9 (39)
Inferior (%)	12 (52)
N/A (%)	2 (9)
VSD site ICE assessment	
Apical (%)	10 (42)
Basal (%)	13 (57)
Cardiogenic shock (%)	20 (88)
Median (IQR) pVAD time (days)	14 (10 – 25)
Pre-procedure pVAD time (days)	8 (5 – 10)
Post-procedure pVAD time (days)	6 (4-25)
Median (IQR) initial Qp:Qs	2.40 (1.63-3.80)

Abbreviations: VSD - ventricular septal defect, IQR - interquartile range, pVAD - percutaneous ventricular assist device, Qp - pulmonary blood flow, Qs - systemic blood flow.

Twelve patients (52%) developed an inferior infarction. Two patients (9%) had non-obstructive coronary disease as confirmed by a coronary angiogram.

Five patients (22%) were hemodynamically stabilized with pharmacological optimization (LV afterload reduction and inotropic support) alone, while 18 patients (78%) required mechanical hemodynamic support based on the primary assessment (Figure 3). Of those requiring MCS, 12 patients received IABP support, and 6 received TH support. Seven of 12 patients initially supported with IABP required a change to TH support, leaving only 5 patients on IABP.

Once hemodynamically stable, the less compromised patients (n=10; 5 on medical therapy and 5 on IABP support) underwent percutaneous VSD repair, and all survived 30 days, and 9 of 10 were alive at 180 days post-procedure.

In total, 13 patients received TH because they could not be hemodynamically stabilized either on medical therapy (n=6) or after IABP support (n=7). Of these, 8 underwent percutaneous repair initially, while 5 had a direct surgical repair. Of the 8 who underwent initial percutaneous repair, 3 failed and crossed over to surgical repair. Of the 5 TH patients who underwent percutaneous repair, 1 patient survived 30 days and was also alive at 180 days.

All 3 TH patients who underwent failed percutaneous repair and converted to surgical repair survived 180 days. Of the 5 TH patients who underwent initial surgical repair, all survived 30 days, and 3 were alive at 180 days post-procedure.

Figure 3. *A total of 23 patients were included in this study. The diagram displays their interventions and outcomes. IAPB (intra-aortic balloon pump), TH (TandemHeart™), VSD (ventricular septal defect).*
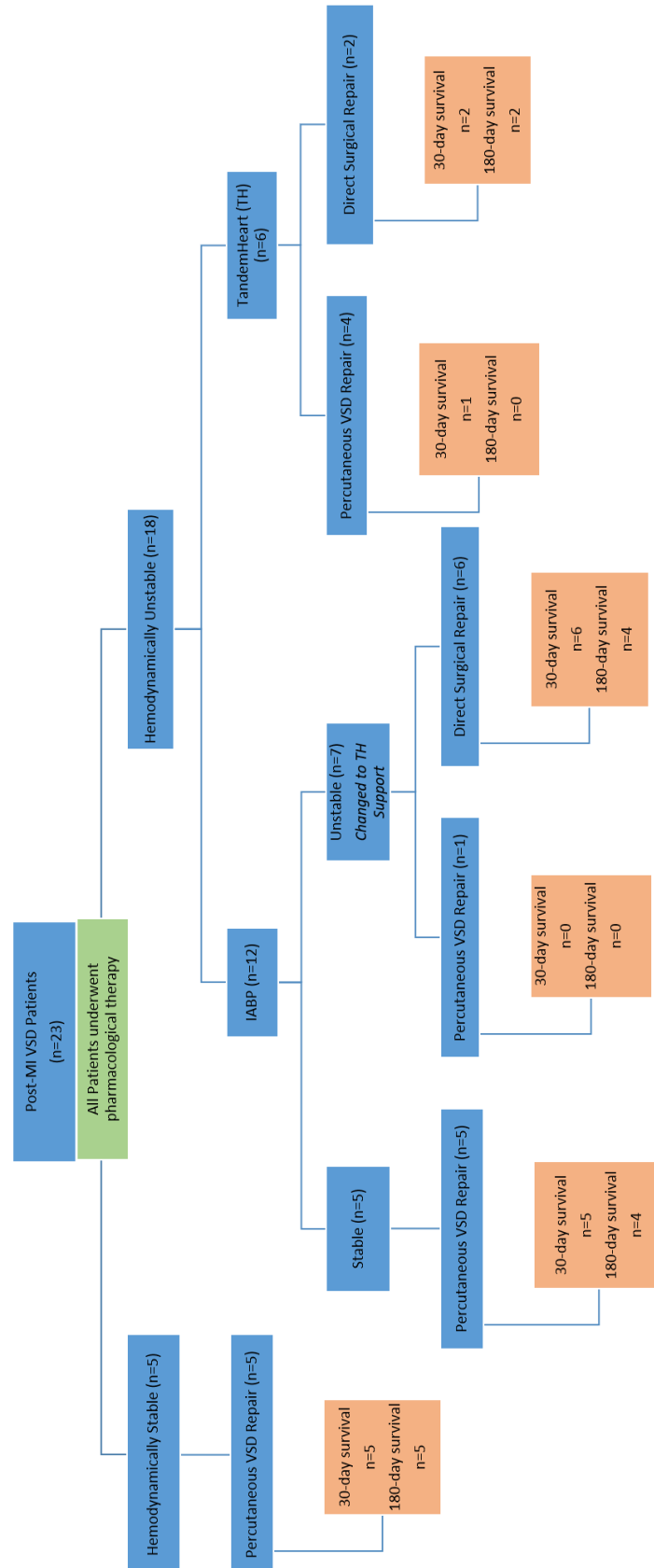
Based on our prior experience with surgical repair,[11] all patients on TH support prior to closure were continued on support after VSD closure. The median number of days on percutaneous ventricular assist device (pVAD) support was 14.0, with a median pre-procedural pVAD and post-procedural pVAD time of 6 days and 8 days, respectively.

The time-lapse between MI and defect closure ranged from 2 to 180 days, and only 4 patients underwent closure within 6 days. As a reflection of our delayed closure strategy (depicted in Figure 1), 18 patients with percutaneous closure had a procedural success rate of 78 % (14/18). A breakdown of these results includes 3 patients who were switched to successful surgical repair; 1 patient implanted with a Total Artificial Heart, as previously described,[17] due to severely depressed ventricular function secondary to the extended myocardial infarction; 4 patients with percutaneous closure died within 30 days. Five patients on TH underwent directly surgical VSD without attempting percutaneous closure due to the large size and location of VSD.

The magnitude of left-to-right intracardiac shunt across the VSD correlated strongly with requirements for and types of MCS. The median Qp/Qs for this patient cohort was 2.37 (IQR 1.50-2.70). There is a significant difference in Qp/Qs ratio between the 10 patients who hemodynamically stabilized with medical therapy (n=5) or with IABP support only (n=5) and the 13 patients who required TH support to hemodynamically stabilize [median: 1.95 (IQR 1.50-2.40) vs. 2.70 (IQR 2.10 - 3.50) respectively, P=0.046].

Most patients in this study underwent percutaneous closure with the Amplatzer® post-infarction VSD occluder device.[15] Three patients required Amplatzer® muscular VSD closure device and one ASO device for percutaneous closure for their septal defects.

There were no intraoperative or procedure-related deaths. The 30-day survival in all patients was 83%, with 180-day survival was 70%.

Discussion

Classical dogma regarding the pathogenesis of post-MI VSDs is that acute large trunk coronary arterial occlusion (generally the left anterior descending artery and right coronary artery, as they are responsible for interventricular septal blood supply) causes transmural septal infarction. This premise works in the setting of an isolated single coronary artery occlusive disease because robust coronary collateral circulation is commonly present in the setting of chronic multi-system coronary artery occlusive disease. After several days, large spatiotemporal gradients in the septal wall incur stress at the interfaces between infarcted and non-infarcted myocardium (due to the fact that the septum is exposed to high-stress gradients as it is situated between the LV and RV). This stress results in ventricular septal rupture and VSD formation--a dynamic perforation of the ventricular septum at the interface between the necrotic and healthy myocardium. VSD is commonly seen in the apical septum in anterior infarctions and in the basal

region in inferior infarctions.[18] Although relatively rare, VSD is associated with high morbidity and mortality if managed medically or with conventional surgery.

Lundblad and colleagues describe a case series of post-MI VSD patients over a 25-year study period, and report that concomitant surgical revascularization of all stenotic vessels, beyond the occluded one, decreased mortality at 30 days; this was attributed to the improved perfusion of the ischemic border zone and the consequently reduced risk of arrhythmias.[19]

Another study reported improved survival for patients in whom VSD surgical repair was delayed by two weeks after infarction to allow stabilization of the defect size and sufficient maturation of the scar borders to hold sutures.[20] Delayed post-MI VSD closure for proper defect sizing and effective anchoring was subsequently adopted by other groups,[21,22] and it constituted one of the foundations of our procedural strategy to surgically manage post-MI VSD.[11] Prior experience with patients undergoing surgical VSD repair[11] and prior necropsy observations[18] support a 7-10 day post-MI delay period before the VSD is closed. VSDs are often observed to enlarge. This is thought to be due to the structural weakness of the infarcted septal tissue, and consequently, negatively impact the outcomes of catheter-based or open surgical closure due to dehiscence – thereby yielding residual VSD. However, the David infarct exclusion technique may be less prone to dehiscence.[9]

The Amplatzer® post-infarction VSD occluder device, when properly sized, further circumvented some of the surgical challenges. The devise is a self-centering occluder that plugs the actual defect. The central part of the device fits the defect and is unaffected by the characteristics of the septal wall; its self-expanding properties allow for adjustments in the event of defect enlargement.

Currently, percutaneous closure is becoming a widespread and widely accepted method to correct post-MI VSD, and this manuscript describes our experience with percutaneous closure strategy. Our outcomes (Figure 3) are similar to the results previously reported by Landzberg and Lock,[23] Holzer et al.,[24] and Egbe et al.[21] In a recent series from the Mayo Clinic, investigators observed 83% survival at 30-days post-procedure and up to 5 years of follow up.[21]

In a US registry, 30-day survival of 18 patients with percutaneous closure of post-MI VSD was 72%; however, 10 (56%) of the patients developed shock prior to defect closure, and 8 (44%) of them needed to be hemodynamically stabilized with IABP. The majority of these patients underwent the procedure at least two weeks after diagnosis.[22]

Although there are still limited clinical data to support delayed VSD closure (i.e., after the acute phase), the timing of the percutaneous closure is significantly influenced by the hemodynamic status of the patient. The presence of cardiogenic shock has been repeatedly associated with a dismal survival rate. Thiele et al[25] have reported their experience in percutaneous post-myocardial closure in 29 patients performed within three days from presentation. Although the procedural success was 86%, the overall survival at 30 days was only 35% and was attributed to the large occurrence of left ventricular rupture, device late embolization, residual large shunt, and cardiogenic shock.

Kapur et al[26] used a male swine preclinical myocardial infarction model to show mechanically unloading the LV utilizing the TH reduced LV wall stress and activated signaling pathways that promoted myocardial salvage, even when coronary reperfusion was delayed. The TH provides direct left-sided cardiac unloading as opposed to venous arterial extracorporeal membrane oxygenation (VA-ECMO), which only indirectly volume-unloads the severely dysfunctional post-MI LV; thus, it was the chosen device for the post-MI VSD cases. VA-ECMO with an IABP in place reduces LV afterload, but it was theorized that the LV volume-unloading would be superior with TH pVAD support.

Our strategy of combining delayed percutaneous closure and percutaneous hemodynamic support ensured both higher short- and long-term survival rates, even in the presence of a significant fraction of patients (86%) in some degree of cardiogenic shock that required TH support, similar to previously published reports of pVAD support prior to VSD repair.[15,16,27,28]

Due to the retrospective nature of our analysis and the relatively small patient cohort, we cannot further define specific hemodynamic and/or clinical parameters whose presence may warrant TH implementation; however, our data do support a delay in VSD closure and the use of the Qp/Qs ratio. Patients who can be hemodynamically stabilized with medical therapy or IABP support will most likely still benefit from a post-acute intervention strategy (i.e., delay VSD closure for approximately two weeks to allow tissue granulation at the VSD). A high initial Qp/Qs ratio (i.e., greater than 2.4) might be a valuable predictive tool. We included the ratio along with other clinical variables in our algorithm as a trigger to mechanical support escalation and surgical VSD repair. However, future larger studies are warranted to confirm this algorithm.

Conclusion

In conclusion, post-MI VSD is associated with extremely high mortality in patients in cardiogenic shock. The introduction of mechanical support and delayed surgical intervention in hemodynamically unstable patients may improve outcomes. Larger and longer studies should be done to provide further evidence for guidelines to advance mechanical support in the early management of post-MI VSD.

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