Perioperative Use of TandemHeart Percutaneous Ventricular Assist Device in Surgical Repair of Postinfarction Ventricular Septal Defect

Igor D. Gregoric, Biswajit Kar, Tomaz Mesar, Sriram Nathan, Rajko Radovancevic, Manish Patel, and Pranav Loyalka

Mortality for patients presenting with acute myocardial infarction (AMI) complicated by ventricular septal defect (VSD) and cardiogenic shock is very high even with surgical repair. We report our experience regarding utilization of TandemHeart, a percutaneous ventricular assist device (pVAD) as an adjunct to the treatment of these patients. Retrospective case series study design included a total of 11 patients with post-AMI VSD and severe refractory cardiogenic shock who received pVAD support at our institution. Three patients underwent immediate surgical repair and received pVAD support for postcardiotomy cardiogenic shock for 2, 4, and 7 days, respectively. However, all three died. The other eight patients had pVAD implanted prior to surgical repair in order to rest the myocardium before operation. Hemodynamics improved immediately after pVAD placement, and after receiving pVAD support for 7 ± 3 days, they underwent surgical VSD repair. Their total pre- and post-surgical pVAD support was 14 ± 4 days. All eight survived 30 days postoperatively. At 6 months postsurgery overall survival rate was 75%. Our small series of these critically ill patients shows a trend toward better survival after immediate pVAD placement to stabilize the patient and allow for myocardial maturation before surgical VSD repair. *ASAIO Journal* 2014; 60:529–532.

Key Words: circulatory assist device, cardiogenic shock, ventricular septal defect

The mortality for patients presenting with ventricular septal defects (VSDs) and cardiogenic shock after acute myocardial infarction (AMI) is extremely high, as much as more than 90%.[1,2] Since Cooley *et al.*'s[3] first repair of a VSD in the 1950s, the treatment of choice for patients with postinfarction VSD has been immediate surgical repair. A prolonged interval between hospital admission and surgical VSD repair is a strong predictor of mortality for patients presenting with hemodynamic deterioration and cardiogenic shock.[4] Even if surgical repair of VSD occurs within the first few days after AMI in patients with severe refractory cardiogenic shock and a posterior VSD, mortality is still very high, which can go up by as much as 83%.[5–8] Thus, the timing and fine balance between waiting and surgical VSD repair are of crucial importance.

During the past decade, mechanical circulatory support therapy has been used in the setting of VSD and severe refractory cardiogenic shock to achieve hemodynamic stability and allow for myocardial scarring and maturation before attempting VSD repair. Examples include the Hemopump,[9] the ABIOMED BVS 5000 (Abiomed, Inc., Danvers, MA),[10–12] extracorporeal membrane oxygenation (ECMO),[13] and the Impella Recover microaxial-flow left ventricular assist device (Impella CardioSystems AG, Aachen, Germany).[14,15] Meanwhile, our group has published several case reports of percutaneous ventricular assist device (pVAD) support with the TandemHeart (CardiacAssist Inc., Pittsburgh, PA) prior to postinfarction VSD repair.[16,17]

We describe the outcomes of 11 patients who underwent surgical repair of post-AMI VSD and perioperative support with a TandemHeart pVAD.

Methods

For this retrospective case series study, we reviewed the records of 11 patients (8 men, 3 women; 52 ± 13 years) who presented with post-AMI VSD and severe refractory cardiogenic shock (CS). The study was approved by our institutional review board. At presentation, 9 of these 11 patients also manifested signs of pulmonary edema. Nine patients were receiving intraaortic balloon pump (IABP) support at presentation, and two patients were immediately placed on TandemHeart support due to profound severe refractory CS. All patients but one had a posterior VSD due to right coronary artery (RCA) occlusion and an ST-elevation AMI. The remaining one patient had an anterior VSD due to left anterior descending (LAD) artery occlusion and anterior ST-elevation AMI (**Table 1**).

Three of these 11 patients went directly to the operating room (OR), underwent urgent surgical repair of their VSDs, and received TandemHeart support for postcardiotomy CS (two patients while in the OR and one patient in the cardiac catheterization laboratory for hemodynamic deterioration 4 days after surgery). The remaining eight patients received TandemHeart support in the cardiac catheterization laboratory several days prior to surgical VSD repair, thereby allowing enough time for myocardial VSD tissue maturation and hemodynamic stabilization. The pVAD flow rate was adjusted to maintain mixed venous oxygen saturation >70 and mean arterial pressure >60 mm Hg and to facilitate aortic valve opening. Patients were weaned off the pVAD when showing adequate hemodynamics and improved end-organ function at a pVAD flow rate of 2 L/day for 2 days.

From the Department of Cardiopulmonary Transplantation and the Center for Cardiac Support, Texas Heart Institute at St. Luke's Episcopal Hospital, Houston, Texas.

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Correspondence: Igor D. Gregoric, MD, Department of Cardiopulmonary Transplantation and the Center for Cardiac Support, Texas Heart Institute at St. Luke's Episcopal Hospital 6400 Fannin Street, Suite 2350, Houston, TX 77030. Email: Igor.D.Gregoric@uth.tmc.edu.

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Table 1. Characteristics of Patients

	All Patients (N = 11)	pVAD Support Prior to Surgery (n = 8)	No pVAD Support Prior to Surgery (n = 3)	*p* Value
Age, years	52 ± 13	53 ± 15	50 ± 9	0.73
Gender				
Male, %	8 (73)	6 (75)	2 (67)	0.84
Female, %	3 (27)	2 (25)	1 (33)	
Heart failure etiology				
Ischemic, %	11 (100)	8 (100)	3 (100)	0. 94
IABP support, n	9 (82)	6 (75)	3 (100)	0.39
pVAD implant after AMI, days	3.6 ± 5.0	1.8 ± 1.8	7.0 ± 7.9	0.54
Urine output, ml/hr	40.3 ± 24.4	35.5 ± 25.9	52.5 ± 21.9	0.46
Blood urea nitrogen, mg/dl	2 5 ± 11	25 ± 11	29 ± 12	0.68
Creatinine, mg/dl	1.56 ± 0.37	1.56 ± 0.39	1.58 ± 0.45	0.95
Hemoglobin, g/dl	12.2 ± 1.6	12.6 ± 1.7	11.2 ± 1.1	0.84
Total bilirubin, mg/dl	0.9 ± 0.6	1.0 ± 0.6	0.6 ± 0.1	0.41

Results are presented as mean ± standard deviation or number of patients (%).
AMI, acute myocardial infarction; IABP, intraaortic balloon pump; pVAD, percutaneous ventricular assist device.

The TandemHeart and its method of implantation have been described elsewhere.[17,18] In short, the femoral artery and vein were cannulated in the standard fashion using the TandemHeart cannulas (15 to 17 French femoral artery cannula, 21 French left atrial venous cannula). The venous cannula was advanced to the inferior vena cava and guided over the wire into the right atrium and then into the left atrium with a transseptal approach through a previously made transseptal puncture. After both cannulas were positioned satisfactorily in the left atrium and femoral artery, the device's inflow and outflow were connected, and the device was started.

Data are expressed as the mean ± standard deviation. Paired Student's t-tests were used to compare hemodynamic and laboratory variables recorded before and after TandemHeart placement. For all analyses, *p* value less than 0.05 was considered statistically significant.

Results

Three patients (two men, one woman) received the TandemHeart postoperatively for postcardiotomy CS (two patients in the OR and one patient in the cardiac catheterization laboratory 4 days after surgery). Initially, these three patients were presenting with severe refractory cardiogenic shock with one patient having pulmonary edema. The average age of these three patients was 50 ± 9 years. All three patients were rushed on IABP support into the OR for surgical VSD repair. The VSD diameter was 1.56 cm in one patient and was assessed intraoperatively with transesophageal echocardiography (TEE). In the other two patients VSD diameter was unknown because the intraoperative TEE assessment was precluded by the emergent surgical procedure. The mean time to pVAD placement after myocardial infarction (MI) was 7.0 ± 7.9 days. Mean duration of pVAD support was 4.3 ± 2.5 days (2, 4, and 7 days, respectively). The two patients receiving the pVAD at the time of surgical VSD repair and then supported postoperatively (2 and 4 days, respectively) died on postoperative day (POD) 17 and 8, respectively. The patient who received the pVAD 4 days after surgical VSD repair remained on pVAD support for 7 days and survived 69 days after the pVAD's removal. All three patients died of multiorgan failure due to progressive heart failure.

Eight patients (six men and two women) with the average age of 53 ± 15 years received TandemHeart support several days before they underwent surgical VSD repair. Mean VSD diameter assessed with TEE was 1.37 ± 0.69 cm at presentation and 2.41 ± 0.71 cm at the time of surgical repair. All eight patients presented initially with pulmonary edema. Seven of eight patients had a posterior VSD due to RCA occlusion. The remaining one patient had an anterior VSD due to LAD artery occlusion. Six of eight patients presented with an IABP in place that was not removed before pVAD insertion. The other two patients had the pVAD implanted immediately for severe refractory cardiogenig shock. The mean time to pVAD placement after MI was 1.8 ± 1.7 days. The duration of presurgical and total (pre- and post-surgical) pVAD support was 7 ± 3 and 14 ± 4 days, respectively.

Hemodynamic and laboratory results before and after pVAD support, prior to VSD repairs, are presented in **Table 2**. In these patients systolic blood pressure, mean arterial pressure, and urine output statistically significantly increased after the pVAD placement. Creatinine levels decreased significantly, and although not reaching statistical significance, lactate dehydrogenase values and lactic acid values showed a decreasing trend. Also, these patients required less perioperative red blood cell transfusions as well as number of vasopressors.

Table 2. Results of Patients with TandemHeart pVAD Support Prior to Surgery

	Before pVAD	After pVAD	*p* Value
Cardiac index (L/min/m^2)	na	4.8 ± 1.8	na
PCWP (mm Hg)	28.9 ± 7.0	9 ± na	na
Systolic blood pressure (mm Hg)	77 ± 11	98 ± 13	0.012
Mean arterial pressure (mm Hg)	66 ± 7	78 ± 9	0.004
Vasopressors used (N)	2.1 ± 0.6	0.6 ± 0.5	0.003
Urine output (ml/hr)	35.5 ± 25.9	91.4 ± 48.5	0.044
Creatinine (mg/dl)	1.56 ± 0.39	0.98 ± 0.20	0.010
Lactate dehydrogenase (U/L)	1,035 ± 778	576 ± 301	0.250
Lactic acid (mg/dl)	26.2 ± 16.1	14.0 ± 6.4	0.106
RBC transfusions (U)	13.8 ± 7.7	30.7 ± 44.7	0.425

Results are presented as mean ± standard deviation or number of patients (%).
na, not available; PCWP, pulmonary capillary wedge pressure; pVAD, percutaneous ventricular assist device; RBC, red blood cell.

There were few complications associated with pVAD support. Two patients suffered mild stroke with diminished motoric function after pVAD removal. One patient regained complete motoric and neurologic function, and the other patient remained with residual weakness of the left upper and lower extremity and additionally developed postoperative pneumonia. Two of 11 patients had ipsilateral lower-limb ischemia immediately after pVAD insertion due to femoral artery cannula blood-flow obstruction. In both cases, the ischemia completely resolved after antegrade arterial femoral perfusion with an 8 French angiocatheter.

All eight patients with pVAD implanted prior to surgery survived 30 days postoperatively. Seven patients were discharged to home. The patient with stroke and pneumonia died in the hospital because of multiorgan failure on POD 40. Of the seven discharged patients, one was later readmitted to the hospital and died of multiorgan failure due to progressive heart failure on POD 37. The remaining six patients survived more than 6 months. One of them later underwent cardiac transplantation 1 year after VSD repair.

Discussion

All eight patients who received TandemHeart support preoperatively and were hemodynamically stabilized before surgical VSD repair survived 30 days postoperatively. Seven of these eight patients were discharged to home, and six survived more than 6 months (75% survival). On the contrary, patients who underwent classic surgical post-AMI VSD repair and were placed on pVAD support postoperatively for postcardiotomy CS all died. Only one of these three patients survived more than 30 days, and none survived long term. The short duration of postsurgical pVAD support (mean duration: 4.3 days) did not appear to improve long-term survival.

Mortality for patients with post-AMI VSD and severe refractory cardiogenic shock is extremely high due to prolonged congestive heart failure and end-organ hypoperfusion if VSD repair is not done promptly. To try to reduce mortality, some authors have used mechanical circulatory support devices such as the Hemopump, ABIOMED BVS 5000, Impella, and ECMO to stabilize patients with severe refractory cardiogenic shock and/or pulmonary edema before performing post-AMI VSD repair.[9–15] In our opinion, the TandemHeart may be more suitable than any of those devices for stabilizing such patients. The Hemopump is no longer manufactured. Extracorporeal membrane oxygenation may not completely unload the left ventricle and so may not resolve any left-to-right shunting in patients with a VSD. The Impella 2.5 does not provide enough flow to reverse severe refractory cardiogenic shock. The Impella 5.0 and ABIOMED BVS 5000 can supply adequate flow, but both are surgical devices that require additional time to implant, thereby increasing the time to cardiac unloading and reestablishment of end-organ perfusion.

In our experience, the TandemHeart pVAD offers additional advantages for patients with VSD and severe refractory cardiogenic shock. It rarely causes hemolysis and poses minimal risk of aspiration of necrotic myocardial debris into the pump. It does not cause right-to-left shunting since the inflow cannula is placed in the left atrium. It can also be used in patients with critical aortic stenosis and, unlike other devices, can truly be placed percutaneously. The concept of transseptal left ventricle unloading without surgery is not new. In 1962, Dennis *et al.*[19] reported the first clinical attempt to rest the myocardium before surgical VSD repair by means of transseptal cannulation and left heart bypass.

We initially used the TandemHeart pVAD in three patients to provide additional hemodynamic support and improve outcomes following VSD repair. Unfortunately, none of these three patients survived long term.

With more experience and a growing understanding that patients in severe refractory cardiogenic shock appear to have better outcomes when resuscitated and stabilized with the TandemHeart before undergoing definitive cardiac surgical repair,[18] we applied this approach to patients presenting with post-AMI VSD and severe refractory cardiogenic shock. We currently use a staged approach in treating patients with post-AMI VSD who also present with severe refractory cardiogenic shock and pulmonary edema despite inotropic and IABP support. First, the patient is taken immediately to the cardiac catheterization laboratory for standard TandemHeart pVAD insertion.[17] The patient is then stabilized, taken to the intensive care unit, and extubated as soon as this can be tolerated. After approximately 1 week, the VSD is again imaged with transthoracic echocardiography or TEE and repaired. Although relatively few centers around the world have been successful in repairing post-AMI VSDs percutaneously, this approach appears to improve survival.[16] If the VSD is amenable to percutaneous repair (*i.e.*, VSD diameter $\leq$2.5 cm, adequate septal margin for device anchoring, adequately thick myocardial free wall, central rather than apical septal position, and no proximity to the aortic valve), this is the preferred choice for closure. If coronary revascularization is required and can be done with stents at the time of the first catheterization, then percutaneous coronary intervention can be easily achieved while the patient is on pVAD support. However, if neither coronary revascularization nor VSD repair is feasible percutaneously, then we perform the surgical VSD repair and aortocoronary bypass (ACB) in the same setting. Six patients in our series received percutaneous stents and four patients required concomitant ACB. In one instance, the only coronary artery affected was the one supplying the infarcted area.

Prolonged preoperative support with the TandemHeart pVAD offers several benefits to patients with post-AMI VSD. First, it allows myocardial VSD tissue to mature, thereby allowing for better suture anchoring on the VSD's myocardial edges at the time of surgical repair. Second, it can prevent increased shunting with enlargement of the VSD. Furthermore, continued support for 1–2 weeks is desirable to hemodynamically stabilize the patient unless the patient develops systemic inflammatory response syndrome (SIRS). Once SIRS begins to develop the patient must undergo surgical VSD repair. However, if TandemHeart is used for longer durations the rate of risks and complications increase (the most common complications include bleeding, dislodging of the cannula, a femoral arteriovenous fistula, thromboembolism, atrial-septal defect, limb ischemia, wound infection, and lymphocele).[18] These complications are more common regarding pVAD implantation contrary to IABP support. However, these critically ill patients had profound cardiogenic shock despite IABP support, and, therefore, pVAD was preferred as the additional therapeutic choice given the fact that patients could not tolerate only IABS and vasopressor support. The most severe complication of limb ischemia was

easily overcome with antegrade percutaneous femoral artery perfusion.

Postoperative pVAD support also offers benefits. First, by unloading left ventricle it can help gradual ventricular reconditioning that follows VSD repair as a result of suture placement. Since the sutures need to be placed into the healthy septal and free wall myocardium in order to form a tight seal and prevent recurrence of the VSD, they can cause additional myocardial damage. Unfortunately, the larger the suture anchorage site, the larger is the area of septal myocardium that becomes dysfunctional. Second, it helps to prevent recurrent VSD formation early after surgical repair, which is important since such recurrence can lead to very high rate of mortality.[4] Third, the TandemHeart may help counteract the worsening of cardiac function that can result from the left bundle branch block or atrioventricular blocks that may be iatrogenically created by septal sutures during VSD repair, and, fourth, in the case of large VSDs, the TandemHeart pVAD postoperatively can help to recondition the right ventricle weakened by left-to-right shunting and to recondition left ventricle due to low resistance caused by VSD. If the entire left ventricle, including septal myocardium, is damaged and the patient's left and right ventricular function is severely depressed due to extensive MI, then VSD repair may be contraindicated and the patient should undergo implantation of the total artificial heart.[20]

Conclusion

At our institution the majority of patients presenting with post-AMI VSD and severe refractory cardiogenic shock received pVAD prior to surgical VSD repair with good results. Thus, our current institutional policy is pVAD placement prior to surgical VSD repair with continuing pVAD support postoperatively for approximately 1 week. The main limitation of this retrospective study is the small number of patients, and large patient cohorts are necessary to confirm our results in the future trials.

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