

Percutaneous Ventricular Assist Device Support

in a Patient with a Postinfarction Ventricular Septal Defect

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Complications of acute myocardial infarction have decreased in number and severity due to the application of early thrombolytic coronary revascularization techniques. Nonetheless, the mortality rate associated with these complications remains high. Ventricular septal rupture is one of the complications that can occur after myocardial infarction. In the treatment of postinfarction ventricular septal rupture, the need for immediate closure to avoid acute hemodynamic compromise must be weighed against the need for delayed repair to enable the acutely necrotic myocardium to organize and to develop fibrotic tissue.

We report the use of a minimally invasive TandemHeart® percutaneous ventricular assist device for 18 days in a 58-year-old man who experienced postinfarction ventricular rupture. The hemodynamic support provided by the device allowed time for left ventricular recovery before attempted percutaneous closure of the ventricular septal rupture and after definitive surgical repair of the septal defect. To our knowledge, this is the 1st reported use of the TandemHeart for support before and after repair of a postinfarction ventricular septal rupture. (Tex Heart Inst J 2008;35(1):46-9)

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Acute postinfarction necrosis of the myocardium can make surgical repair of ventricular septal defects (VSDs) challenging. The choice must be made between early surgical repair to avoid hemodynamic compromise and surgical delay to allow time for organization and fibrosis of the surrounding tissues. In selected patients, percutaneous closure of a VSD may be performed.

We describe the case of a man who experienced postinfarction ventricular septal rupture (VSR). He received hemodynamic support with a percutaneous ventricular assist device (pVAD) to enable recovery of the myocardium, both before and after repair of the ruptured septum. Postinfarction ventricular rupture and the benefits of pVADs in such cases are discussed.

Case Report

In April 2007, a 58-year-old man who had hypertension and hyperlipidemia presented with chest pain at another institution. Inferior wall myocardial infarction (MI) was diagnosed, and the patient underwent emergency cardiac catheterization. Complete occlusion of the right coronary artery was found; therefore, percutaneous coronary intervention was performed. Transmural inferior myocardial infarction (TIMI) grade 3 flow was established. The patient remained hemodynamically unstable, and an intra-aortic balloon pump was inserted. Transthoracic echocardiography showed a muscular VSD (approximately 5.5 mm in diameter) in the mid-ventricular inferoseptum, with a gradient of 55 mmHg (velocity, 3.7 m/sec). There was mild concentric left ventricular hypertrophy, but the left ventricular ejection fraction was normal (>0.60). Right ventricular function was also normal. No valvular abnormalities were noted.

The patient was subsequently transferred to our hospital for closure of the VSD. Physical examination at the time of admission revealed a coarse, grade 4/6 holosystolic murmur, with a precordial thrill. Rales were heard over the lower third of the bilateral lung fields. The patient was in acute renal failure (creatinine, 3.5 mg/dL). Coronary angiography showed a patent right coronary artery stent and nonsignificant stenosis of the left anterior descending artery and obtuse marginal branch. Because of the patient's unstable hemodynamic status, a TandemHeart® pVAD (CardiacAssist, Inc.; Pittsburgh, Pa) was inserted, which resulted in improved hemodynamic values. The patient was started on aspirin (325 mg/day), clopidogrel (75 mg/day), atorvastatin (20

mg/day), and a β -blocker (75 mg twice/day). Anticoagulation was achieved with intravenous heparin. Because of brief episodes of atrial fibrillation, intravenous amiodarone (1 mg/min for 6 hr, followed by 0.5 mg/min for 18 hr) was also administered. Four days after admission, repeat transthoracic echocardiography showed the VSD in the mid-ventricular inferoseptum, with a left-to-right shunt. The diameter of the defect had increased to approximately 1.3 cm. The pulmonary-to-systemic flow ratio (Q_p/Q_s) was 2.5. Overall, the patient's systolic cardiac function remained stable during the next week with pVAD support.

Nine days after the patient was admitted, he underwent attempted percutaneous closure of the VSD under transesophageal echocardiographic (TEE) guidance with a 33-mm STARFlex[®] septal repair implant (NMT Medical, Inc.; Boston, Mass). However, the defect was too large for percutaneous closure, and the procedure was aborted when the device slipped into the right ventricle. The closure device was retracted from the right ventricle into the right atrium, through the superior vena cava, and out via the right jugular vein. Device removal caused a jugular vein tear, which was surgically repaired immediately. After device removal, the patient developed severe tricuspid regurgitation due to chordal rupture, and it was decided that the VSD and the flail tricuspid valve leaflet should be repaired surgically.

Surgical Technique

The next day, the patient was taken to the operating room. Intraoperative TEE showed tricuspid regurgitation and a posterior septal defect. The TandemHeart was stopped, and cardiopulmonary bypass (CPB) was initiated. After the aorta was cross-clamped, antegrade and retrograde cardioplegia were used to stop the heart. The tricuspid valve, seen through a right atriotomy, had a torn posterior and septal papillary muscle and 3 ruptured chordae tendineae of the anterior leaflet. The left ventricle was opened through the infarcted portion of the inferior myocardium. The VSD was situated in the mid-posterior portion of the septum and was approximately 2 cm in diameter. Pledged transseptal interrupted polypropylene sutures were used to attach a woven Dacron Hemashield[®] patch (Boston Scientific Corporation; Natick, Mass) to the septum. The free edge of the patch was further attached to the septum with 3-0 polypropylene running sutures, creating a double-patch suture line. The septal tissue was firm enough to support a tight suture line; however, the septum still showed signs of postinfarction tissue organization. The left ventriculotomy was closed in the usual fashion, with felt strips for reinforcement.¹ The right atrial wall was then retracted and the tricuspid valve repaired. The torn papillary muscles were reattached to their base with 4-0 GORE-TEX[®] sutures (W.L. Gore & Associates; Flagstaff,

Ariz). The torn chordae tendineae were remodeled, and 3 neochordae were constructed with interrupted 4-0 GORE-TEX sutures. A DeVega tricuspid valvuloplasty was performed with 2-0 polypropylene sutures. Saline injection inside the right ventricle showed a competent tricuspid valve, and the atriotomy was closed.

The aortic cross-clamp was then removed. The heart was de-aired via the TandemHeart cannula side port, and the aorta was de-aired via a vent needle inserted in the aortic root. After ventilation was restarted and the heart was in normal sinus rhythm, the patient was slowly weaned from CPB. Intraoperative TEE confirmed that the tricuspid valve was competent, without residual leaks, that the heart was completely de-aired, and that there was no residual shunt across the ventricular septum. After the cannulae and the pump were completely de-aired and CPB was terminated, the TandemHeart was restarted. The chest was closed, and the patient was taken to the intensive care unit.

Postoperative Course

The postoperative period was complicated by bloody pericardial effusion, which was evacuated, and by acute renal failure; the patient recovered from both. He was successfully weaned from TandemHeart support 8 days postoperatively. However, after device explantation, he developed right hemiparesis due to a small left frontal lobe infarct. He made a steady neurologic recovery and was transferred to a rehabilitation facility approximately 3 weeks after surgery. He fully recovered and continued to do well 9 months later.

Discussion

We first used the TandemHeart pVAD in this patient for 10 days to provide hemodynamic stability before performing a VSD repair and to allow time for organization of the surrounding infarcted septum. In addition, the device was used to support the left ventricle during the attempted percutaneous VSD closure. The TandemHeart also provided postoperative ventricular support for an additional 8 days, which led to postsurgical ventricular recovery.

Complications associated with MI include free-wall rupture, papillary muscle rupture with resultant mitral regurgitation, and VSR. Before the use of thrombolytic therapy, the incidence of postinfarction VSR was from 1% to 2%.² However, with the use of modern coronary reperfusion techniques, including thrombolytic agents, the incidence of postinfarction VSR has markedly decreased. The Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-I) trial included 41,021 patients with acute MI who received thrombolytic therapy. Of those patients, VSR was suspected in 140 (0.34%) and was confirmed by a retrospective review in 84 (0.2%).² Remarkably, the 30-day

mortality rate in patients who develop postinfarction VSR remains high in this population—reportedly 47% in patients treated surgically and 94% in patients managed conservatively. Furthermore, VSR appears to occur more rapidly after thrombolytic therapy than it does before such therapy.^{2,3}

Ventricular septal ruptures tend to occur in a bimodal distribution—within 24 hours or between 3 and 5 days after an MI.⁴ The progression of myocardial necrosis affects the timing of the rupture, which in turn influences the timing of the repair. However, the timing of the repair creates a self-selection bias; patients whose repair is delayed are less hemodynamically compromised and their conditions are considered to be less critical. In cases of VSR, the use of mechanical circulatory support allows time for the hemodynamic status to stabilize and for the condition of more critically ill patients to improve before percutaneous or surgical repair.

In a series by Thiele and colleagues,⁵ intra-aortic balloon pump support in patients with VSDs reduced left-to-right shunting by decreasing left ventricular afterload. In that series, however, the mortality rate in patients undergoing early surgical repair was 83%. In patients whose conditions were stabilized and who then underwent surgical closure, the mortality rate was 29%. The overall mortality rate with use of an intra-aortic balloon pump support for postinfarction VSR was 56.5%.⁵ The presence of cardiogenic shock appeared to be an important predictor of death, and the ability to delay surgery to allow organization of the surrounding septum, an important factor in survival. Other methods of support have been used to delay surgery, including temporary closure of a VSD with a Swan-Ganz balloon catheter⁶ or calibrated sizing balloon.⁷ Definitive hemodynamic support has been undertaken with surgically implanted biventricular assist devices such as the AB5000[®] (ABIOMED, Inc.; Danvers, Mass).^{8,9} In patients who require this type of support, however, the perioperative risk remains high. Recently, Patanè and colleagues treated an acute postinfarction VSD patient with use of the minimally invasive Impella[®] left ventricular assist device (ABIOMED) as a bridge to cardiac transplantation, because the location of the VSD posed a surgical risk to the mitral valve and the surrounding tissue was too fragile to repair.¹⁰

The TandemHeart pVAD, with its continuous flow, has flow dynamics similar to those of surgically implanted axial-flow ventricular assist devices in that it augments end-organ perfusion. The TandemHeart unloads the failing left ventricle by directing blood from the left atrium to the pump via the device's inflow cannula, which is inserted into the femoral vein and advanced across the interatrial septum into the left atrium. Circulatory support is provided by oxygenated blood that is returned to the femoral artery via the device's outflow cannula.

The TandemHeart can provide additional safety during percutaneous VSD closure, when hemodynamic collapse is possible. In the patient described here, percutaneous VSD closure was attempted; unfortunately, the VSD was too large and the STARFlex septal repair device slipped into the right ventricle. The percutaneous retrieval of the device from the right ventricle caused the right ventricular papillary muscle and chordae to rupture, which led to tricuspid regurgitation and to a tear in the jugular vein. We then decided to surgically repair the VSD.

Residual transeptal shunts are another complication of VSD repair. DeJa and colleagues¹¹ observed a residual shunt across the septum in 40% of patients, despite a 15-day delay before surgical repair (mean time to VSD development after MI, 5–6 days; mean time to surgical repair after rupture, 9 days). Reoperation for residual shunt closure carried a mortality rate of 29% (4 of 13 patients died).¹¹

In the same group of patients, DeJa's group concluded that cardiogenic shock upon admission to the hospital and deterioration of hemodynamic status between the time of admission and surgery were strong predictors of death.¹¹ Stabilizing patients before surgical VSD repair can be beneficial, but this has to be weighed against the hazards of prolonging surgical repair. The use of a left ventricular assist device before percutaneous or surgical repair of a postinfarction VSD helps stabilize patients who are in cardiogenic shock and provides additional time for the septal myocardial tissue to mature. Postoperatively, residual shunting is possible, and any preserved left ventricular function can deteriorate when the interventricular septum is immobilized by sutures and a Dacron patch. Conduction abnormalities and cardiogenic shock also contribute to high surgical mortality rates, despite optimal perioperative management. The TandemHeart provides postoperative cardiac support during recovery from a stunned myocardium; in addition, unloading the left ventricle can prevent the postoperative occurrence of shunts across the infarcted septum.

Conclusion

The main challenge in treating patients with postinfarction VSDs is deciding whether to correct the defect immediately to avoid hemodynamic compromise or to delay treatment to allow time for tissue recovery and reorganization. The healing that ensues during this period is important because it aids in effective anchoring of surgical or percutaneous patches. Percutaneous ventricular assist device placement provides hemodynamic support for patients who are in cardiogenic shock from a myocardial infarction or a postinfarction VSR. Support from this device creates a therapeutic window during which the patient can recover before surgery and provides extra time for physicians to plan the optimal re-

pair technique, both of which can help avoid a residual transseptal shunt.

The TandemHeart can be lifesaving in patients who have conduction abnormalities caused by VSD closure procedures and in those who require retrieval of an embolized device after percutaneous VSD closure. The TandemHeart can also support the stunned ventricle after surgical VSD closure, allowing it to recover. To our knowledge, ours is the 1st reported use of the TandemHeart for support before and after repair of a postinfarction VSR. Our patient received pVAD support for 18 days. Further study is needed to determine whether this therapy can improve survival in patients who have undergone surgical or percutaneous repair of a VSR.

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