Catheterization and Cardiovascular Interventions 73:267–271 (2009)

Case Reports

Percutaneous Closure of an Iatrogenic Atrial Septal Defect

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The use of the Tandem Heart™ percutaneous ventricular assist device (Tandem Heart pVAD, Cardiac Assist technologies, Pittsburgh, PA) as a bridge to recovery or to other cardiopulmonary support systems has been rising. One requirement for placement of this device is an intraatrial septostomy which is usually closed during the surgical insertion of more permanent ventricular assist devices. We present a case of a 62-year-old man with a residual acquired atrial septal defect (ASD) from Tandem Heart™ placement, which could not be closed surgically during insertion of left and right ventricular assist devices. The patient remained intubated and hypoxemic after removal of his right ventricular assist device due to the presence of persistent right to left shunting. With closure of the ASD using an 8-mm Amplatzer® septal occluder (ASO; AGA Medical Corp., Golden Valley, MN) the patient stabilized and was successfully extubated. © 2009 Wiley-Liss, Inc.

Key words: ASD; Tandem Heart™; peripheral interventions; LVAD; RVAD; Amplatzer®

INTRODUCTION

Treatment options for patients with cardiogenic shock and systolic heart failure have been expanding with the use of extracorporeal life support such as the Tandem Heart™ percutaneous left ventricular assist device (pVAD). These systems can be used as a bridge to recovery or as a bridge to more permanent surgically implanted ventricular assist devices (VAD). First described in 2001 by Thiele et al., the Tandem Heart™ pVAD is a low-speed centrifugal continuous flow pump that can be used as an effective bridge for short-term circulatory support [1].

The insertion of the Tandem Heart™ pVAD requires an atrial septostomy in order to allow placement of the left atrial cannula. We describe a case where a patient who required a Tandem Heart™ device as a bridge to right and left ventricular assist device (RVAD/LVAD) support had a residual acquired atrial septal defect (ASD) upon removal of the Tandem Heart™ device. Routine surgical closure of the intraatrial septostomy could not be performed in this case due to technical difficulty. The degree of shunting anticipated through the small acquired ASD was felt to be minimal, given the TEE results, and the intraoperative decision was made to leave the ASD and to consider closing using a percutaneous method if clinically significant. Significant right to left shunting through the residual acquired ASD causing refractory hypoxemia was unmasked following RVAD removal during weaning of mechanical ventilatory support. Successful percutaneous closure of the ASD was then performed using an Amplatzer® septal occluder (ASO) device with rapid correction of the patient's hypoxemia and ventilatory support needs.

CASE REPORT

A 62-year-old man with a history of severe ischemic dilated cardiomyopathy was admitted to our hospital

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Conflict of interest: Nothing to report.

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Received 16 May 2008; Revision accepted 4 August 2008

DOI 10.1002/ccd.21768
Published online 26 January 2009 in Wiley InterScience (www.interscience.wiley.com).

with multisystem organ failure and New York Heart Association functional class IV decompensated congestive heart failure. Physical examination revealed mild distress, a blood pressure of 74/49, biventricularly paced at a HR of 89 beats per minute, breathing 22 times a minute saturating 96% on room air. His cardiac examination was significant for a holosystolic murmur over the mitral window radiating to the axilla as well as an S3. His extremities were cool with significant pitting edema to the waist. His chest X-ray revealed severe cardiomegaly, mild pulmonary edema, and pleural effusions. Transthoracic echocardiogram confirmed biventricular dilatation and dysfunction with severe mitral regurgitation and moderate tricuspid regurgitation. His admission labs were significant for blood urea nitrogen (BUN) of 76 mg/dL, serum creatinine (SCr) of 1.9 mg/dL, and glomerular filtration (GFR) rate of 38 mL/min worsened from his baseline BUN of 42 mg/dL, SCr of 1.1 mg/dL, and GFR of 60 mL/min. His coagulation studies revealed a prothrombin time of 37.9 sec and an international normalized ratio (INR) of 4.1 while on Coumadin therapy.

The patient was admitted to the coronary care unit where IV diuretics and multiple IV inotropic agents were trialed with no significant success. Eventually, the patient's status gradually deteriorated to cardiogenic shock and worsening organ function evidenced by a rise in his BUN to 99 mg/dL, SCr to 2.9 mg/dL, and persistent elevation of his INR at 1.8 off all anticoagulation. On day 18 of his admission, while transplant workup was being performed, he was taken to the cardiac catheterization lab for hemodynamic assessment and possible placement of intraaortic balloon pump (IABP) and/or Tandem Heart^{TM} device. An IABP was placed and with that his hemodynamics were as follows: a right atrial pressure of 28 mmHg, pulmonary artery pressure of 59/30 mm Hg (44), pulmonary capillary wedge pressure of 24 mm Hg, and a systemic arterial pressure of 77/50 mm Hg (57). Thermodilution cardiac output and cardiac index were 3.9 L/min and 1.89 L/min/m^2, respectively. Fick cardiac output and cardiac index were 4.04 L/min and 1.95 L/min/m^2, respectively. Pulmonary artery blood saturation was 43.1%, while femoral arterial blood saturation was 92%. The calculated systemic vascular resistance at the time, with the balloon pump in place, was 574.26 dyne sec cm^{-5}. The decision at that time was made to insert a Tandem Heart^{TM} pVAD for better hemodynamic support. Given the concern for a new onset of fevers with possible infection and his multisystem organ dysfunction, surgical VAD placement was deferred.

The Tandem Heart^{TM} was inserted in the cardiac catheterization lab via the right femoral vein and femoral artery in the standard approach [1]. A transseptal puncture was performed using a BRK Brockenbrough needle, and a Mullins sheath was then inserted into the left atrium. An Inoue guidewire (Toray International, Houston, TX) was advanced into the left atrium through the Mullins sheath and the sheath was then removed. Over an Inoue guidewire, a 21 French (Fr) left atrial catheter, after using a dilator, was positioned in the LA across the intraatrial septum. A 15 Fr arterial catheter was positioned in the right common iliac artery via a right femoral arteriotomy. Initial settings of the Tandem Heart^{TM} device were 4000 RPM with flow rates of 3 L/min providing the intended partial assistance while preserving aortic valve opening and the prevention of stagnation of flow in the aortic root. All cannulae positions were confirmed with fluoroscopy.

With Tandem Heart^{TM} support, the patient had improvement in his hemodynamics and organ function. His urinary output increased from less than 2 L/day to a urinary output of over 5 L/day with net output improving from a daily net positive balance to a net negative balance of over 1.5 L a day. His BUN improved to 79 mg/dL and SCr to 1.7 mg/dL after 3 days of Tandem Heart^{TM} support. He had stable oxygenation and was at most supported with 3 L of oxygen by nasal cannulation. After the exclusion of active infection and stable noncardiac organ function, he was taken on hospital day 21 for definitive placement of both right and left ventricular assist devices. Successful placement of a Heartmate XVE® LVAD (Thoratec Corp., Pleasanton, CA) and an Abiomed BVS 5000® RVAD (Abiomed, Danvers, MA) was performed. At the time of surgery, due to severe continuous calcification of the diaphragm that extended onto the inferior vena cava (IVC) and inferior and lateral aspect of the right atrium, a caval snare could not be placed to selectively drain the IVC. The right atrium ultimately was never opened and therefore the iatrogenic ASD was not directly visualized. Intraoperative TEE, however, revealed no significant shunting across the ASD when both the RVAD and LVAD systems were functioning. Biventricular support was maintained for 17 days with resolution of his organ dysfunction demonstrated by a drop in his BUN to 40 mg/dL and SCr to 0.9 mg/dL. At this point, he continued to have a low oxygen requirement of 1–2 L by nasal cannula, with pulse oximeter saturations of 95–99%. In view of significant improvement of right ventricular function he returned to the operating room on hospital day 38, postoperative day 17, for RVAD removal. Following the removal of the RVAD, the patient developed significant difficulty with oxygenation during weaning of mechanical ventilatory support and nitric oxide and he was unable to be extubated following the procedure.

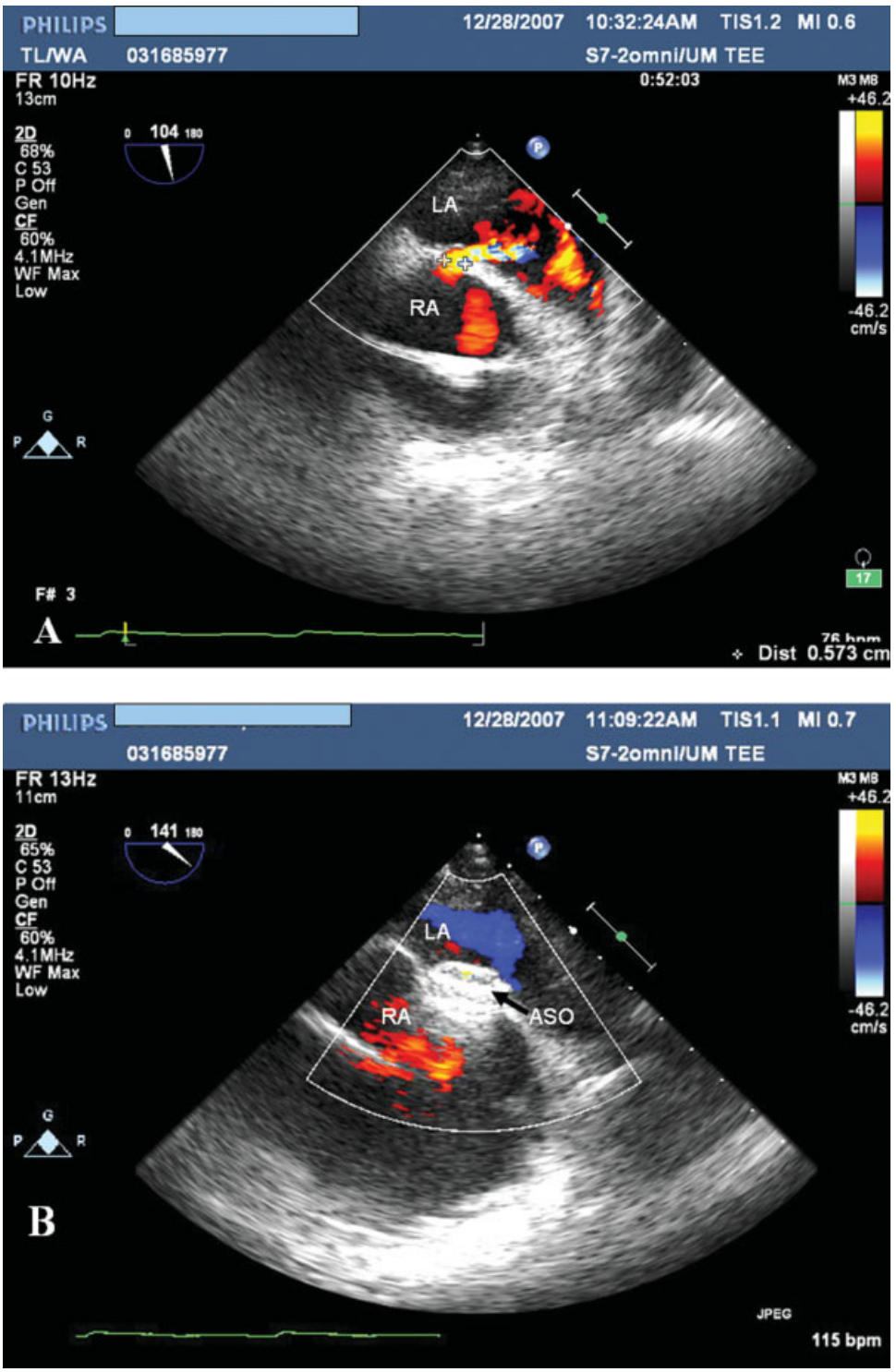

Fig. 1. A: ASD with color flow Doppler demonstrating right to left shunting. B: Following deployment of the Amplatzer septal occluder device, no residual shunt is seen by TEE. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

He required assisted ventilation with fractions of inspired oxygen (FiO2) of 100% with adjunctive inhaled nitric oxide at 20 ppm to maintain oxygen saturations in the 90% and higher range by arterial blood gas measurement. His LVAD flows were maintained at a level of 5–6 L/min. With only trivial increase in his central venous pressure, concern for RV failure after RVAD removal was not a likely explanation for his re-

TABLE I. Arterial Blood Gas Measurements Preprocedure (gray) and in the 48 hr Following Closure of the ASD with the Amplatzer® Septal Occluder Device

Date/time	pH	PaCO2	PaO2	HCO3	%Oxygen saturation	%FiO2	PPM of NO
4 hr Preprocedure	7.47	30	73	22	93	65	20
10 hr Post	7.4	35	144	22	97	50	9
24 hr Post	7.47	31	83	22	95	30	–
48 hr Post	7.46	34	83	24	95	6 L NC	–

fractory hypoxemia. Additionally, his organ function was maintained following RVAD removal. Transthoracic echo performed at bedside with an agitated saline bubble study revealed marked right to left shunting. Therefore, it was decided to proceed with percutaneous closure of the ASD.

The patient was brought to the cath lab 4 days after RVAD removal on hospital day 42, postoperative day 21 for percutaneous closure of the ASD. With transesophageal echocardiography (TEE) and fluoroscopic guidance using a sizing balloon, the ASD was sized at 7 mm in diameter. Initial TEE demonstrated exclusive right to left shunting through the ASD (Fig. 1A). Bivalirudin (Angiomax™ The Medicines Company, Parsippany, NJ) was used for anticoagulation due to a mildly positive HIT ELISA assay. Using standard techniques, an 8-mm ASO device was successfully deployed across the ASD [2,3]. Once deployed, correct positioning of the 8-mm ASO was confirmed on cinefluoroscopy and on TEE. Following deployment of the ASO device, there was no continued shunting seen across the intraatrial septum (Fig. 1B).

After returning to the ICU, the patient had nearly immediate improvement in his arterial saturations (Table I). He was able to be weaned off inhaled nitric oxide within 24 hr. His FiO2 requirements also dramatically improved shortly after he returned from the procedure. Within 48 hr, on hospital day 44, postoperative day 23 the patient was successfully extubated and successfully weaned off much of his inotropic support. His hemodynamic improvement continued and he was eventually discharged to home after rehabilitation to await cardiac transplantation.

DISCUSSION

Several authors have reported the discovery of intraatrial communications with significant right to left shunting when patients with congestive heart failure with severely elevated left atrial pressure undergo abrupt reduction in pressure following cannulation with a LVAD. Infrequent case reports have described the successful closure of those defects discovered after LVAD placement with atrial septal occluder devices [4–6]. Others have identified clinically significant iatrogenic ASDs after percutaneous transseptal balloon mitral valvuloplasty, which were closed using a septal occluder device as well [7–10]. One group has also reported the identification and successful percutaneous Amplatzer® closure of a previously undiscovered patent foramen ovale (PFO) in two patients while being supported by a Tandem Heart™ pVAD device [11]. We describe a case where an acquired persistent ASD that occurred during the transseptal placement of a Tandem Heart™ device required closure with a percutaneous approach, because it could not be closed surgically. Furthermore, it was only after the removal of RVAD support that the clinical significance of this residual ASD was unmasked.

RVAD and LVAD support provide a unique hemodynamic situation in patients with biventricular failure and an ASD as they reduce both right and left atrial and ventricular diastolic filling pressures. Given simultaneous biventricular unloading when used concomitantly, any shunting that might occur could go underappreciated. Usually, the intraatrial septostomy caused by the Tandem Heart™ device, if not closed surgically at the time of more invasive support implantation, has been shown in animals to heal completely at the end of a 4-to 6-week period [12,13]. If persistent, it usually results in insignificant shunting in those eventually weaned off the Tandem Heart™ with no eventual LVAD placement. In our patient, who had inadequate time for the septostomy to close on its own, a clinically relevant ASD with significant right to left shunting was identified at the time of RVAD removal.

The use of anticoagulation during the placement of such occlusion devices is imperative as thrombosis and ultimate embolization of thrombus is a risk factor of this procedure [3]. Great care is taken to exclude patients who are hypercoagulable when considering the use of a septal occluder device. Because of reduced platelets in this patient, evaluation for heparin-induced thrombocytopenia (HIT) was performed identifying a mildly positive HIT ELISA assay. Shammas et al. have previously reported the safe use of bivalirudin in ASD closure [14]. With this information, we elected to refrain from using heparin and chose to use bivalirudin during the placement of the ASO. The patient was also loaded with clopidogrel (Plavix®, Bristol-Myers

Squibb, New York, NY) 600 mg in the cath lab and continued on aspirin 325 mg and clopidogrel 75 mg po qd for a planned 6 months. To date, he has had no evidence of thrombotic complication due to the placement or existence of the ASO device.

In conclusion, the acquired ASD from placement of a Tandem Heart™ pVAD device rarely causes significant problems for patients in whom the device is used. In the situation where patients have a pVAD device used as a bridge to LVAD support, one needs to consider the possible complications created by the residual ASD once the Tandem Heart™ device is removed. This case highlights the possible clinical relevance of these iatrogenic acquired ASDs and that percutaneous closure of the residual ASD is an effective treatment option when surgical closure cannot be performed.

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