

Percutaneous Ventricular Assist Device and Extracorporeal Membrane Oxygenation Support in a Patient with Postinfarction Ventricular Septal Defect and Free Wall Rupture

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ABSTRACT

We describe the case of a 54-year-old woman with a postinfarction ventricular septal defect (VSD) and ventricular free wall rupture who was stabilized with a percutaneous ventricular assist device (pVAD) to allow for myocardial infarct stabilization. Following the rupture of the right ventricular free wall and cardiopulmonary arrest on hospital day 10, pVAD support was promptly converted to extracorporeal membrane oxygenation (ECMO) support for stabilization. After surgical repair was completed, pVAD support was continued for 4 days to allow recovery. The patient was discharged on postoperative day 11 and is alive and well 4 years later. Postinfarction VSD with free wall rupture may be salvaged with pVAD and ECMO support.

INTRODUCTION

Ventricular septal defect (VSD) is an infrequent but potentially devastating complication of acute myocardial infarction. The mortality rate for patients with acute myocardial infarction, VSD, and severe refractory cardiogenic shock is approximately 80% to 90% [Birnbaum 2002; Sibai 2010]. The optimal treatment of a post-myocardial infarction VSD remains controversial. We describe the case of a 54-year-old woman with a postinfarction VSD and rupture of the ventricular free wall who was stabilized with a percutaneous ventricular assist device (pVAD) and extracorporeal membrane oxygenation (ECMO) to permit stabilization of the myocardial infarct.

CASE REPORT

A 54-year-old woman was admitted to a community hospital after experiencing an episode of chest pain, dyspnea, and decreased exercise tolerance. An electrocardiogram demonstrated ST-segment elevation with inferior myocardial

infarction. A cardiac catheterization evaluation revealed an occluded right coronary artery, a severely stenosed diagonal artery, and a large VSD (1.2 cm). An intra-aortic balloon pump was inserted, and inotropic therapy was started. The patient was transferred to our hospital for a higher level of care. The patient presented with hypotension, pulmonary edema, and poor systemic perfusion. A repeat cardiac catheterization evaluation and an echocardiogram revealed that the VSD had enlarged to 1.7 cm and that the patient had pulmonary hypertension with systemic hypotension. The TandemHeart pVAD (CardiacAssist, Pittsburgh, PA, USA) was placed to stabilize the patient's hemodynamics and to allow scarring for the development of thicker septal tissue to aid closure of the VSD. Percutaneous closure of the VSD was attempted on hospital day 6. The catheterization examination showed that the VSD had enlarged to 2 cm and that the septal dissection extended into the right ventricular wall, which was very thin. Because VSD closure was technically not possible at this time, the patient was taken back to the intensive care unit, and surgical repair was planned for a later date. After 4 days in the intensive care unit, the patient suddenly became severely hypotensive (arterial blood pressure <40 mm Hg) with electromechanical dissociation. Cardiopulmonary resuscitation (CPR) was initiated because the pVAD flow became <1 L/min. Following the initiation of CPR, the blood in the inflow cannula became dark red. A quick examination with a portable transthoracic echocardiography device showed that the transeptal pVAD inflow cannula had dislodged from the left atrium into the right atrium and that a large pericardial effusion was present. These observations suggested myocardial free wall rupture with cardiac tamponade. The immediate infusion of 2 units of blood and 250 mL of normal saline increased the pVAD flow to 2 L/min. An oxygenator was promptly coupled into the pVAD outflow tubing for conversion to ECMO support. Immediately after pVAD/ECMO was started, the patient's oxygenation improved, as evidenced by a change in the color of the blood in the tubing from dark to bright red. Blood flow in the device increased to approximately 3.8 L/min. The patient was emergently taken to the operating room for VSD closure, repair of the myocardial rupture, and aortocoronary bypass. At the completion of the operation, the dislodged transeptal cannula was repositioned into the left atrium, and pVAD support was resumed. The

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patient was weaned from pVAD support 4 days later and was discharged home on the 11th postoperative day. The patient continues to do well 4 years later.

DISCUSSION

Our case demonstrates that hemodynamic stabilization with a pVAD and immediate conversion to ECMO support upon free wall rupture can be lifesaving in this potentially catastrophic complication. The mortality rate for patients in severe refractory cardiogenic shock after acute myocardial infarction and VSD, or with double cardiac rupture (VSD and ventricular free wall) as in the present case, is extremely high [Birnbaum 2002; Sibal 2010]. The risk factors for post-myocardial infarction double cardiac rupture (VSD and ventricular free wall) include female sex, advanced patient age, anterolateral infarction, hypertension, and current smoking [Birnbaum 2002; Sibal 2010]. The optimal treatment of post-myocardial infarction VSD remains controversial. Percutaneous closure of the VSD may have a theoretical advantage of potentially avoiding the morbidity and mortality associated with surgical procedures; however, there is no optimal percutaneous VSD closure device. In addition, the lack of a suitable anatomy for landing the device, the large size of the VSD, and the location of the rupture at the septal margin may limit the application of this technique. Given that the myocardial VSD edges are very friable and the VSD enlarges during the first 10 days after acute myocardial infarction, the timing for the VSD closure can pose a major problem, and waiting for tissue maturation may not be possible.

During the last decade there have been noticeable attempts to use left ventricular assist devices (LVADs) for hemodynamics stabilization in patients with VSD and refractory cardiogenic shock [Samuels 2001; Faber 2002; Thiele 2003; Conradi 2009]. Waiting for the VSD size to stabilize and for scar development over the first week carries the potential risk of increasing left-to-right shunt and symptoms of congestive heart failure. We previously reported the use of a pVAD in treating a patient with a postinfarction VSD [Gregoric 2008], whereas others have used the Impella LVAD (Abiomed, Danvers, MA, USA) [Patane 2009], or ECMO to stabilize patients with cardiogenic shock and VSD [Formica 2005; Rohn 2009]. Total artificial heart is another mechanical support device that can be used for an extremely large, irreparable VSD; however, the goal is to save the native heart whenever possible.

Converting the pVAD support to ECMO immediately after confirmation of the dislodged transeptal cannula into the right atrium and post-myocardial infarction rupture of the free wall with cardiac tamponade was lifesaving for our patient. Unloading the right side of the heart from the right

atrium with ECMO decreased right ventricular filling and potentially decreased blood loss in the pericardium. Furthermore, this rapid conversion to ECMO support reversed severe hypoxemia and allowed safe transport to the operating room.

CONCLUSION

In the complicated case of a postinfarction VSD with severe refractory cardiogenic shock, pVAD support can stabilize hemodynamics to allow time for more definitive treatment. If free wall rupture and cardiopulmonary arrest ensues, immediate conversion to ECMO can be lifesaving.

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