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Balloon Aortic Valvuloplasty under Temporary Mechanical Circulatory Support as a Bridge to Aortic Valve Replacement in a Patient with Hemodynamic Failure Secondary to Critical Aortic Valve Stenosis

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ABSTRACT

Aortic valve replacement in the setting of critical aortic valve stenosis with cardiogenic shock is associated with high mortality, yet surgery is the only definitive treatment. We present the case of a patient with critical aortic valve stenosis and cardiogenic shock who received a short period of percutaneous mechanical support and balloon aortic valvuloplasty that resulted in rapid clinical improvement. The patient then underwent uneventful aortic valve replacement. We believe that temporary mechanical circulatory support coupled with balloon aortic valvuloplasty helped to restore hemodynamic stability before surgery, leading to a better outcome.

INTRODUCTION

Hemodynamic failure secondary to critical aortic valve stenosis presents a dilemma to the surgeon. Emergent surgery is high risk, yet valve replacement is the only definite solution. We present the case of a patient with hemodynamic failure secondary to critical aortic valve stenosis treated with balloon aortic valvuloplasty under temporary mechanical circulatory support that resulted in hemodynamic stabilization before urgent aortic valve replacement.

CASE PRESENTATION

A 51-year-old man presented to our institution with severe prosthetic aortic valve stenosis and in a shock state. The patient's history was significant for drug and alcohol abuse with frequent relapses despite his being in a rehabilitation program. He had undergone an aortic valve replacement with an unknown-sized biological prosthesis for infective endocarditis 5 years prior to his current presentation. The patient arrived at our institution intubated and maintained on propofol, dobutamine, and norepinephrine with a heart rate of 113/min and a systemic arterial blood pressure of 85/62 mm Hg.

We stopped the propofol and norepinephrine, but the hemodynamics did not improve. The mixed-venous oxygen saturation measured 69%, which indicated adequate oxygen delivery. We were unclear as to the cardiac function of the patient and therefore placed a pulmonary artery catheter, which demonstrated a right atrial pressure of 11 mm Hg, a pulmonary artery pressure of 40/30 mm Hg, and a cardiac index of 2.6 L/min per m^2. During this period of assessment, the hourly urine output was <30 mL/hour most of the time and responded only transiently to the administration of intravenous furosemide.

A transesophageal echocardiography evaluation showed global hypokinesia of the left ventricle with an ejection fraction of 10%. There was moderate prosthetic aortic valve regurgitation and severe aortic valve stenosis, with an estimated valve area of 0.6 cm^2 and a mean gradient of 44 mm Hg across the valve. Right ventricular function was also severely depressed. A coronary angiography examination showed no significant coronary artery disease. The patient's hemodynamics worsened with an increase in his heart rate to 125/min, a decrease in the systemic blood pressure to 79/60 mm Hg, and an increase in pulmonary artery blood pressure to 52/32 mm Hg.

We felt it unlikely that the patient would survive emergent aortic valve replacement. The calculated Society of Thoracic Surgeons (STS) risk of mortality for emergent aortic valve replacement in this patient was 43%, which prompted us to consider other modalities to treat the hemodynamic dysfunction. The patient's moderate aortic valve regurgitation precluded an attempt at intra-aortic balloon pump counterpulsation or conventional unsupported balloon aortic valvuloplasty. We elected to place the patient on percutaneous temporary mechanical circulatory support and to proceed with aortic valvuloplasty in an attempt to stabilize the patient in anticipation of urgent aortic valve replacement.

A computed tomography angiogram of the abdomen and pelvis identified no significant arterial disease that would preclude insertion of a mechanical circulatory support device. In the cardiac catheterization laboratory, the patient's hemodynamics deteriorated further to a systemic arterial pressure of 60/47 mm Hg and a pulmonary artery pressure of 52/33 mm Hg. We percutaneously inserted a TandemHeart 21F venous

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cannula (CardiacAssist, Pittsburgh, PA, USA) via the right femoral vein and positioned it across the atrial septum into the left atrium. A percutaneously inserted TandemHeart 17F arterial cannula was placed in the left femoral artery provided for arterial access. We achieved adequate anticoagulation with an activated clotting time >250 seconds, with an initial bolus and subsequent continuous infusion of intravenous heparin.

The cardiac index measured 1.76 L/min per m^2 prior to initiation of mechanical circulatory support. TandemHeart support provided a cardiac index of 2.4 L/min per m^2 and produced an immediate increase in the patient's systemic arterial blood pressure to 86/68 mm Hg with maintenance of a pulsatile arterial waveform. In addition, there was also a simultaneous reduction in the pulmonary artery pressure to 23/13 mm Hg (Figures 1 and 2) and a reduction in right ventricular cavity dilation, as observed by transesophageal echocardiography. We subsequently dilated the aortic valve twice with a 22-mm balloon. A postdilation transesophageal echocardiography evaluation showed a modest improvement in the mobility of the aortic prosthetic valve cusp without a change in the regurgitation (moderate).

Following the procedure, the patient returned to the intensive care unit off the dobutamine infusion and on an infusion of vasopressin (which we stopped 5 hours later). His systemic arterial blood pressure was 115/90 mm Hg, the pulmonary arterial pressure was 46/25 mm Hg, and the cardiac index was 2.56 L/min per m^2. The hourly urine output immediately increased to >50 mL/hour. The patient received additional intravenous fluid boluses as a means of further resuscitation but did not require any additional inotropic support.

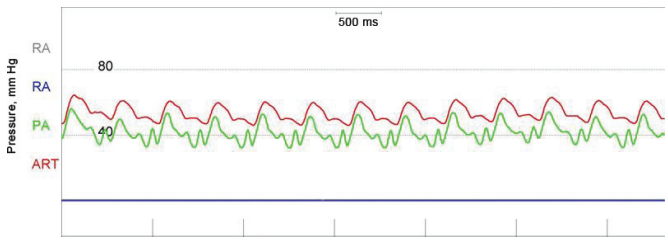

Figure 1. Hemodynamic tracing before initiation of mechanical circulatory support shows a pulmonary artery (PA) pressure of 52/32 mm Hg (green tracing) and a systemic blood pressure (ART) of 79/60 mm Hg (red tracing). RA indicates right atrium.

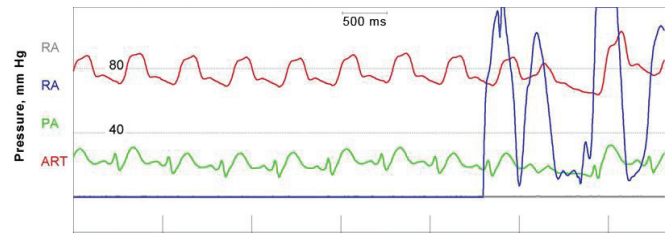

Figure 2. Hemodynamic tracing immediately after initiation of mechanical circulatory support shows a pulmonary artery pressure of 23/13 mm Hg (green tracing) and a systemic blood pressure of 88/68 mm Hg (red tracing). RA indicates right atrium.

Through the night, the TandemHeart had repeated difficulties with reduced flow, which were thought to be due to adherence of the venous cannula to the atrial wall. Nonetheless, the patient's hemodynamics remained stable. A transesophageal echocardiography examination performed the following morning demonstrated that the left ventricle ejection fraction remained at 10%. There was also moderate central mitral valve regurgitation. The period of hemodynamic and clinical improvement prompted us to move ahead with replacement of the dysfunctional aortic prosthesis. In addition, we elected to repair the mitral valve.

We placed the patient on cardiopulmonary bypass at moderate hypothermia (32C) and provided myocardial protection with combined antegrade and retrograde cold blood cardioplegia. We explanted what appeared to be a frozen prosthesis (Figure 3) and replaced it with a 21-mm Perimount pericardial valve (Edwards Lifesciences, Irvine, CA, USA). We repaired the mitral valve with a 30-mm Carbomedics complete-ring (Sorin Group, Arvada, CO, USA) annuloplasty. The patient was weaned from cardiopulmonary bypass on a moderate amount of inotropic support. We removed the circulatory-support cannulae and repaired the atrial septum and femoral vessels. The patient had an uneventful postoperative course and went home on the sixth postoperative day. A transthoracic echocardiography examination at the time of discharge demonstrated a left ventricular ejection fraction of 42%.

DISCUSSION

The mortality associated with aortic valve replacement has been reduced significantly over the last 10 years, but cardiogenic shock and emergent operation remain significant predictors of an increased mortality, with a noted odds ratio of 3.77 (95% confidence interval, 2.75-5.16) in the most recent STS report [O'Brien 2009]. There have been isolated reports of success with emergent aortic valve replacement in the setting of cardiogenic shock; however, postoperative complications are frequent and include low cardiac output, prolonged convalescence, and renal and respiratory failure [Christ 1997].

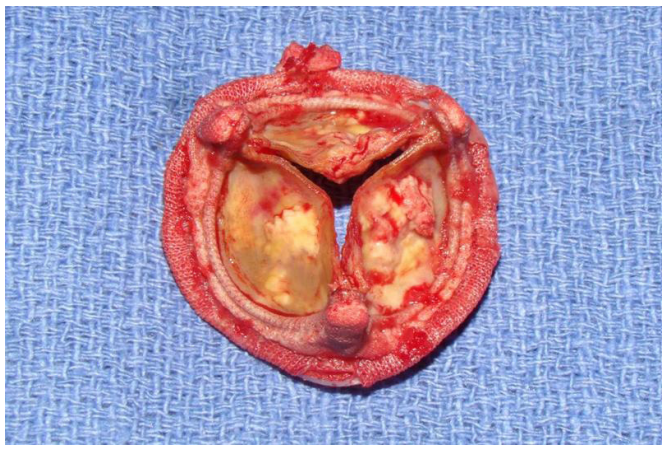

Figure 3. Explanted prosthesis shows diffuse calcification involving the cusps with a frozen orifice.

Resuscitation and stabilization of the patient with pharmacologic or invasive strategies prior to aortic valve replacement may protect against some of these complications.

Pharmacologic intervention, however, may not lead to a significant increase in stroke volume in the presence of critical aortic valve stenosis and a severely dysfunctional left ventricle. Invasive intervention strategies include intra-aortic balloon pump counterpulsation, percutaneous balloon aortic valvuloplasty, or mechanical circulatory support. The use of balloon valvuloplasty in adults with critical aortic valve stenosis has been reported [Doguet 2010]. However, significant aortic valve regurgitation is a contraindication to the use of either intra-aortic balloon pump counterpulsation or conventional unsupported balloon valvuloplasty, because their use can increase valvular regurgitation and left ventricular dysfunction.

Mechanical circulatory support represents an additional option that functions to offload the left ventricle by reducing left atrial and left ventricular diastolic volume and pressure, which improves systemic perfusion [Frank 2006]. Several reports describe the effective use of temporary mechanical circulatory support in patients with cardiogenic shock refractory to vasopressor therapy and intra-aortic balloon pump counterpulsation, or in those with cardiogenic shock undergoing high-risk percutaneous coronary artery interventions [Lemos 2003; Kar 2010]. The TandemHeart is such a device and consists of an inflow cannula (placed through the femoral vein across the atrial septum into the left atrium), an arterial outflow cannula, and a centrifugal pump that provides up to 5 L/min of cardiac output. Complications associated with percutaneous mechanical circulatory support include bleeding around cannulae (29%), groin hematoma (5%), limb ischemia (3%), and femoral artery dissection (1%). Wire perforation of the left atrium occurred in 1% of patients [Kar 2010].

TandemHeart support provided hemodynamic support and stability during completion of balloon aortic valvuloplasty in our critically ill patient. Following the procedure, there was immediate and persistent clinical improvement despite subsequent apparent intermittent function of the TandemHeart device and only modest improvement in aortic valve function as seen in the echocardiography evaluation. It is unclear to us whether the clinical improvement was from the TandemHeart, the balloon aortic valvuloplasty, a combination of the two, or, possibly, to changes in medical management. Regardless, the combination of an increase in systemic arterial pressure, a reduction in pulmonary artery pressures, and an increase in urine output all suggested a better prognosis for our patient. For that reason, we proceeded to the urgent operation and obtained a good result. We were able to remove the TandemHeart device from our patient immediately following weaning from cardiopulmonary bypass, which may have improved our chances of avoiding cannulation-related complications.

An additional treatment option in this patient would be transcutaneous aortic valve-in-valve insertion, which is a relatively new technique in the management of biological prosthetic aortic valve degeneration. Described in 2007 by Wenaweser et al, the valve-in-valve procedure is gaining momentum as an alternative to open surgery in high-risk patients with biological valve degeneration [Wenaweser 2007]. Presently, valve options include the Sapien valve (Edwards Lifesciences) and the CoreValve Revalving System (Medtronic).

Pasic and colleagues published their experience with the aortic valve-in-valve technique (Sapien valve) in 14 patients [Pasic 2010]. They reported 100% procedural success with no associated mortality. The authors state that absolute contraindications to the procedure include severe paravalvular leak, endocarditis, and valve thrombosis. Of importance is that the mean postoperative aortic valve area of the prosthesis was 1.35 cm^2, which is consistent with residual moderate aortic stenosis; 4 patients (29%) had a postoperative aortic valve area of <1.0 cm^2, which is consistent with severe aortic stenosis. The relatively new technology may add to the treatment of prosthetic aortic valve degeneration. At present, however, more experience, a clarification of indications, and a longer follow-up are required to clarify this new technology's role in management of this difficult clinical condition.

CONCLUSION

The present case report provides the hypothesis that invasive modalities such as temporary mechanical circulatory support and balloon aortic valvuloplasty can reverse hemodynamic failure secondary to critical aortic valve stenosis. Restoration of hemodynamic stability prior to aortic valve operation may improve outcome, as in this instance. Transcutaneous aortic valve-in-valve insertion represents an alternative technique in the treatment of such patients, but it requires further investigation prior to its widespread adoption.

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