A 76-year-old man with severe degenerative aortic stenosis, coronary artery disease, and progressive congestive heart failure with severe systolic dysfunction underwent implantation of a Heartmate II left ventricular assist device (LVAD; Thoratec Corporation, Pleasanton, CA) after developing refractory cardiogenic shock. Because there was moderate aortic insufficiency (AI), the aortic valve was closed with a pledgeted central coaptation stitch that approximated the Noduli of Arantius with minimal residual regurgitation.

After initial clinical improvement, by 5 months after LVAD implantation, the patient was hospitalized with recurrent heart failure symptoms. Echocardiography revealed new onset moderate to severe AI with 2 dominant jets that were present during systole and diastole (Video I in the online-only Data Supplement, Figure). Over the next several months he developed worsening heart failure symptoms (class IIIb) with no evidence of LVAD malfunction. Concomitantly, his left ventricular end diastolic dimension increased from 4.3 cm to 5.8 cm. Right heart catheterization revealed a pulmonary capillary wedge pressure of 20 mm Hg with a cardiac index of 2.6 L/min. Surgical correction, including aortic valve replacement and closure of the left ventricular outflow tract were considered; however, because of the patient’s advanced age and 2 prior sternotomies, percutaneous closure of the aortic valve was pursued.

A 2-dimensional and 3-dimensional transesophageal echocardiography was used to deploy a 6 mm Amplatzer septal occluder device (St. Jude Medical, St. Paul, MN) between the left- and noncoronary cusps of the oversewn aortic valve. A second 5 mm Amplatzer device was deployed between the left- and right-coronary cusps of the aortic valve (Video II in the online-only Data Supplement). There was immediate improvement in the severity of aortic insufficiency after the procedure (Video III in the online-only Data Supplement).

Five months after the procedure the patient has only trace AI with class II heart failure symptoms and has been free of further heart failure hospitalizations. He has moderate hemolysis (lactate dehydrogenase preprocedure 195 IU/L and post-procedure 457 IU/L; normal reference range 98–192 IU/L) but his hemoglobin remains >10 g/dL and stable.

A growing number of patients with AI have undergone concomitant aortic valve surgery at the time of LVAD implantation to eliminate valvular insufficiency. These concomitant procedures include patch closure of the left ventricular outflow tract, replacement of the aortic valve with a bioprosthesis, or closure of the aortic valve with a central coaptation stitch. Little is known about the durability of these strategies, and in the case we report, the initial repair strategy was not durable. Regardless of the preoperative degree of AI, late onset of regurgitation is a well-recognized complication of continuous-flow LVAD placement, and it is associated with increased morbidity and mortality.

The use of an Amplatzer device after LVAD placement to eliminate aortic insufficiency has been described previously. However, this is the first reported case to use multiple small Amplatzer devices to treat aortic insufficiency in a previously oversewn valve. The postprocedural concerns include hemolysis from peridevice regurgitant flow, obstruction of the coronary ostia, peridevice thrombus formation, erosion into the aortomitral curtain, and device embolization. Proper device selection (diameter and length) may prevent these complications. It is also important to note that this strategy for aortic valve closure results in complete LVAD dependency, and accidental power loss will likely be fatal.

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Disclosures

None.

References


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