CASE 7—2013
Percutaneous Closure of the Aortic Valve for Severe Aortic Insufficiency Due to a Left Ventricular Assist Device

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IN PATIENTS with a left ventricular assist device (LVAD), the presence of aortic insufficiency (AI) can be significantly problematic, leading to symptoms of congestive heart failure and end-organ dysfunction. The authors present 2 cases of percutaneous closure of the aortic valve (AV) using an artial-septal occluder device in patients with LVADs and concurrent AI.

CASE 1: UNEVENTFUL PERIOPERATIVE COURSE

A 56-year-old man (180 cm, 89 kg) with an LVAD presented to the authors' institution with symptoms of decompensated heart failure, including hypotension and pulmonary edema leading to respiratory failure. Workup revealed new-onset severe AI. Medical history included coronary artery disease and previous coronary artery bypass graft surgery. The patient subsequently developed class IV heart failure due to ischemic cardiomyopathy and underwent LVAD insertion 8 months prior to admission. His history also included episodes of ventricular tachycardia (required implantable cardiac defibrillator insertion) as well as hypothyroidism. Home medications included amiodarone, furosemide, aspirin, warfarin, and levothyroxine. Physical examination revealed a blood pressure of 69/53 mmHg and a heart rate of 90 beats/min. LVAD flow was normal (7.2 L/min). Cardiac examination was remarkable for LVAD sounds, and pulmonary examination showed coarse rales bilaterally. Complete metabolic panel demonstrated elevated levels of creatinine (2.1 mg/dL), lactic acidosis (13.5 mEq/L), and an anion gap of 28 mEq/L. The patient also had elevated levels of liver enzymes (alanine transaminase 1,286 μ/L, aspartate aminotransferase 466 μ/L), hyperbilirubinemia (5.2 mg/dL), and elevated international normalized ratio (5.0). In addition, hematologic studies found anemia (hemoglobin, 8.5 g/dL) and thrombocytopenia (platelet count, 87 K/μL). Electrocardiogram revealed an atrial and ventricular pacemaker rate of 90 beats/min. Transthoracic echocardiography revealed a severely dilated left ventricle with severely reduced function. The AV did not open during systole and demonstrated severe AI, which was continuous throughout the cardiac cycle. The right ventricle was dilated moderately, with moderately reduced function.

This patient with an LVAD presented with symptoms of cardiogenic shock and evidence of end-organ dysfunction, including acute renal failure and acute liver failure. The presence of severe AI was determined to be the cause of these symptoms. After admission to the intensive care unit, he required emergent intubation and mechanical ventilation for respiratory failure. The patient was started on dobutamine (5 μg/kg/min) and underwent diuresis with furosemide intravenously, which improved his condition. He was extubated on day 3 of admission. Due to persistent coagulopathy and renal failure, the patient was deemed a poor surgical candidate for conventional redo-surgery and AV closure. Instead, a novel procedure was devised, which involved the use of a percutaneous cardiac occluder device to anatomically close the AV, thus preventing AI.

The patient was transported to the cardiac catheterization suite for the planned procedure. A right radial arterial catheter was already in place and the standard monitors were applied. Dobutamine (5 μg/kg/min) was continued intravenously throughout induction of anesthesia. After preoxygenation, anesthesia was induced with midazolam (1 mg), fentanyl (200 μg), propofol (50 mg), and rocuronium (50 mg) intravenously. The patient was mask-ventilated and then underwent uneventful direct laryngoscopy and tracheal intubation. The patient was placed on controlled mechanical ventilation, and maintenance of anesthesia was achieved using sevoflurane and intermittent rocuronium to prevent movement during the procedure. A right internal jugular 9-French introducer, and a transesophageal echocardiography (TEE) probe were inserted. Prior to the procedure, a focused TEE examination revealed a continuous eccentric jet of severe AI (Fig 1), an AV annulus diameter of 24 mm, a sinus of Valsalva diameter of 31 mm, patent left and right coronary ostia, a severely dilated left ventricle with severely reduced function, and moderately reduced right ventricular function. The AV and sinus of Valsalva measurements were particularly important because

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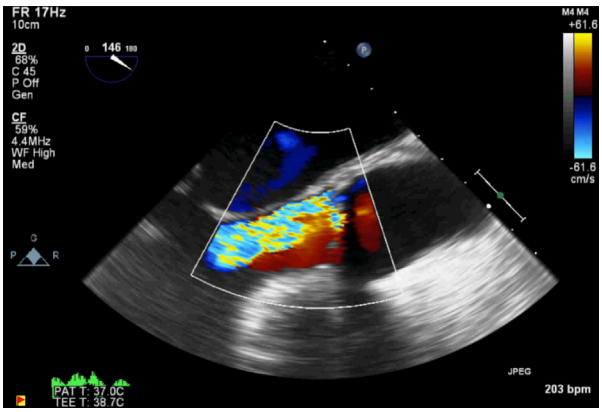

Fig. 1. Preprocedure transesophageal echocardiography revealing an eccentric jet of severe aortic insufficiency via color-flow Doppler.

they assisted with proper sizing of the double-disc closure device.

The surgeons began by accessing the right femoral artery under fluoroscopy and inserting a sheath. Next, a delivery wire containing a 25-mm Amplatzer cribriform septal occluder was advanced under TEE guidance retrograde via the descending aorta and aortic arch. Before deployment of the device, LVAD flows were reduced temporarily to approximately 1.0 L/min. The device tip was advanced through the AV, and the distal disc was deployed within the left ventricular outflow tract. Then, utilizing real-time TEE imagery, the delivery wire was withdrawn until the distal disc abutted the AV. The proximal disk was deployed within the sinus of Valsalva and the delivery wire removed. LVAD flows were increased to baseline level (7.2 L/min). A repeat TEE examination was obtained, which showed the closure device in place around the AV, with no residual AI (Fig 2). The left coronary ostium was patent, but the right coronary ostium was unable to be visualized due to ultrasound dropout from the metallic device. Left ventricular function remained poor; however, chamber size was reduced and the septum was more midline. Right ventricular function was unchanged; however, the chamber size was enlarged significantly. An epinephrine infusion (0.02 μg/kg/min) was initiated to support the severely dilated right ventricle. The patient remained hemodynamically stable and was extubated and transferred to the intensive care unit. After additional diuresis with furosemide, he was weaned from the inotropic infusions and had complete resolution of his symptoms. He was discharged home 5 days postoperatively and continues to do well more than a year after the procedure.

CASE 2: RIGHT-HEART FAILURE

A 54-year-old female (158 cm, 75 kg) with an LVAD presented to the authors' institution with symptoms of decompensated heart failure, including shortness of breath and pulmonary edema. Medical history was significant for nonischemic dilated cardiomyopathy and LVAD insertion 5 months prior to admission. Her medical history also included pulmonary hypertension, paroxysmal atrial fibrillation, pacemaker insertion, hyperlipidemia, diabetes mellitus, hypothyroidism, and gastritis. Medications included aspirin, furosemide, lisinopril, atorvastatin, famotidine, levothyroxine, glipizide, and sitagliptin. Physical examination revealed a blood pressure of 76/51 mmHg with a heart rate of 100 beats/min. LVAD flow was normal (4.2 L/min). Cardiac examination found LVAD sounds, and pulmonary examination had coarse rales bilaterally. Basic metabolic panel was remarkable for elevated levels of creatinine (1.9 mg/dL), lactic acidosis (8.8 mEq/L), and an anion gap of 16 mEq/L. The patient also had elevated levels of liver enzymes (alanine transaminase 142 μ/L, aspartate aminotransferase 72 μ/L), hyperbilirubinemia (9.9 mg/dL), and elevated international normalized ratio (2.4). In addition, hematologic studies showed anemia (hemoglobin, 8.0 g/dL) and thrombocytopenia (platelet count, 117 K/μL). Electrocardiogram revealed ventricular pacing with a rate of 100 beats/min, and chest radiography revealed diffuse bilateral pulmonary edema. Similar to the previous patient, transthoracic echocardiography revealed a severely dilated left ventricle, with severely reduced function. The AV did not open during systole and demonstrated severe AI, which was continuous throughout the cardiac cycle. The right ventricular function was severely reduced.

As a sequela of new-onset AI, this patient presented with the same constellation of clinical symptoms as the first patient. Although, in this patient, they were not quite as severe. She did not experience respiratory failure or require intubation prior to the procedure, and no inotropic medications were initiated preoperatively. Nonetheless, she too was deemed a poor surgical candidate for conventional reoperation and was taken to the cardiac catheterization suite for percutaneous AV closure. The standard monitors were applied, and a right radial arterial catheter was inserted. After preoxygenation, anesthesia was induced with midazolam (2 mg), fentanyl (300 μg), propofol (20 mg), and vecuronium (5 mg) intravenously. The patient was mask-ventilated and then underwent uneventful direct laryngoscopy and tracheal intubation. The patient was placed on controlled mechanical ventilation, and maintenance of anesthesia was achieved using sevoflurane and intermittent vecuronium. A right internal jugular 9-French introducer was inserted, and a pulmonary artery catheter was advanced into the

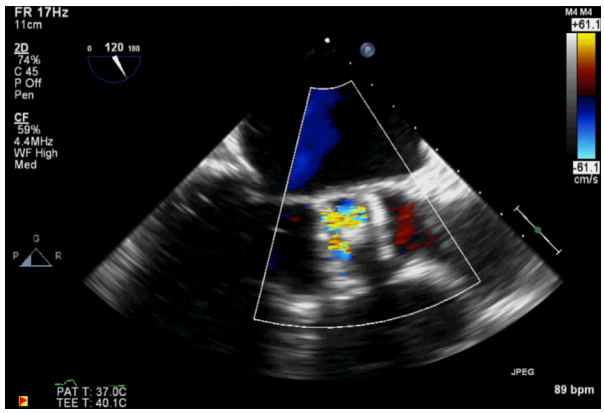

Fig. 2. Postprocedure transesophageal echocardiography revealing the occluder device in proper position over the aortic valve. Utilizing color-flow Doppler, turbulence is seen within the discs, but no residual aortic insufficiency is present.