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Placement of a Continuous-Flow Ventricular Assist Device in the Failing Ventricle of an Adult Patient with Complex Cyanotic Congenital Heart Disease

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ABSTRACT

For patients with end-stage heart failure and contraindications to transplantation, insertion of a continuous-flow left ventricular assist device (LVAD) is an effective treatment strategy. We present a case of LVAD insertion in a 46-year-old man with cyanotic complex congenital heart disease and an extensive surgical history who presented with failure of his systemic ventricle. The insertion of an LVAD in our patient restored cardiac output and improved cyanosis and native ventricular function. As the number of patients with congenital heart defects surviving to adulthood increases, destination LVAD therapy may be increasingly considered as an alternative.

INTRODUCTION

Insertion of left ventricular assist devices (LVADs) is an effective treatment strategy for patients with end-stage heart failure who are poor candidates for transplantation. The new generation of axial-flow pumps can achieve flows of up to 8 L/min with minimal hemolysis and mechanical wear. In the adult patient with complex congenital heart disease, anatomic and physiologic abnormalities present unique challenges in the use of LVADs. We present a case of HeartMate II (Thoratec Corporation, Pleasanton, CA, USA) LVAD insertion in a 46-year-old man with dextrocardia, congenitally corrected transposition of the great arteries (TGA), pulmonary hypertension, and an extensive surgical history, who developed progressive cyanosis and failure of his systemic ventricle.

CASE REPORT

A 46-year-old man with known congenital heart disease was referred to our service for evaluation of refractory fatigue and lethargy. His medical history was notable for congenitally corrected TGA, dextrocardia, pulmonic stenosis, ventricular septal defect, a partially anomalous pulmonary venous return, and absence of the suprarenal inferior vena cava. He had received a shunt of the left pulmonary artery to the descending thoracic aorta at age 7 and a shunt from the aorta to the main pulmonary artery at age 21, both of which were for progressive cyanosis.

The patient did reasonably well until 4 years before our involvement, when he presented once again with congestive failure and progressive cyanosis. He was found to have failure of his pulmonary ventricle, so both previously constructed shunts were taken down. His left pulmonary artery was reconstructed, an atrial septectomy was performed, and an anastomosis of the superior cava to the right pulmonary artery was constructed to create Fontan physiology. Although the patient initially reported feeling subjectively better, the improvement was short-lived, and he presented to our institution with a 3-year history of progressive decline. On admission, his blood pressure was 103/51 mm Hg, and the central venous pressure was 29 mm Hg. Systemic arterial oxygen saturation was 29%, and his hemoglobin concentration was 17 mg/dL with a serum sodium level of 121 μmol/L. The patient's elevated homocysteine concentration was consistent with his known congenital heart disease and surgical history. His systemic ventricle had an ejection fraction of 30%. His pulmonary ventricle was dilated and severely hypokinetic.

A physical examination suggested no other obvious deformities except for severe kyphoscoliosis. The patient had well-healed sternotomy and left thoracotomy scars. He was awake but lethargic and was otherwise neurologically intact. There was pitting edema to the knees, and the patient was visibly cyanotic. Heart-lung transplantation was considered, but we felt it to be contraindicated by his chronic renal insufficiency, severe scoliosis, and extensive surgical history. Therefore, we felt that improving his cardiac output and decreasing congestion with an LVAD would provide the greatest potential benefit and the least risk.

Surgical Technique

The patient's massively dilated heart was accessed through a redo sternotomy incision. ε-Aminocaproic acid was administered

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preoperatively to facilitate postoperative hemostasis. Cardiopulmonary bypass (CPB) was established with cannulation of the left common femoral artery and vein. A second venous cannula was placed into the common atrium. During CPB, normothermia was maintained, and the heart was allowed to beat. The inflow cannula of the HeartMate II axial-flow LVAD was placed at the apex of his systemic ventricle. The LVAD pump housing was placed below the diaphragm in the peritoneal cavity, with the pump housing resting on the liver. The LVAD outflow graft was brought up to the left of the heart and anastomosed to the ascending aorta with a partial-occlusion clamp. The leftward course of the outflow graft produced an acceptable lie, given the position of the patient's transposed great vessels. After standard deairing techniques, the patient was weaned from CPB, and the pump was activated. Surgical hemostasis was reasonable, but because of marked coagulopathy, the patient required multiple transfusions of platelets, fresh frozen plasma, and cryoprecipitate. The patient's chest was initially left open, but the sternum was successfully closed the following day.

Postoperatively, the pump generated 5 L/min at a pump speed of 10,000 rpm. The patient's systemic arterial saturation rose to 75% (PAO_2, 41 mm Hg). His PCO_2 was 40 mm Hg, and the blood pH was 7.38. One week after surgery, the patient was awake, alert, following commands, and tolerating progressive physical rehabilitation. He developed transient renal insufficiency, and he required a tracheostomy for prolonged ventilatory support. His kidney function ultimately improved, and he was successfully weaned from ventilatory support. After extensive physical rehabilitation, the patient was discharged home.

COMMENT

The use of LVADs in cyanotic heart disease has been limited until recently to postcardiotomy recovery in infants. The current report represents the first documented use of an axial-flow LVAD as destination therapy in an adult patient with cyanotic congenital heart disease and is the third reported case of LVAD implantation in a patient with dextrocardia [Musci 2002; Southard 2005]. Our institution previously demonstrated effective bridge-to-transplant therapy with an LVAD in a 53-year-old woman with congenitally corrected TGA and severe congestive heart failure [Gregoric 2005].

Implantation of an axial-flow LVAD in our cyanotic patient reduced cyanosis and improved cardiac output and ventricular function. The outcome of this case suggests a possible expanded role for LVADs as destination therapy in patients with congenital heart disease. Our findings and those of others support the implantation of assist devices as destination therapy in patients with end-stage heart failure and contraindications to transplantation [Rose 2001; Frazier 2003; Park 2005; Frazier 2007].

Once cardiopulmonary transplantation was deemed unlikely for our patient, discussions centered around 2 questions: (1) Would increasing cardiac output with a device improve his symptoms and physiology, and (2) would he also require correction of the remaining right-to-left shunt (the hepatic venous return to the right atrium)? The answers were unclear, and the patient's anatomy and surgical history presented formidable challenges for shunt correction. After weighing the options, we decided that device implantation without shunt correction would be the safest strategy.

The augmented cardiac output to the patient's lungs and ventricular unloading improved the patient's postoperative arterial oxygen saturation and relieved symptoms of congestive heart failure. He had lived with severe cyanosis his entire life. His postoperative arterial saturation of 75%, with increased forward blood output from the pump, was therefore sufficient to achieve adequate oxygen delivery to his tissues.

In conclusion, a VAD can be an excellent treatment option for patients with end-stage heart failure and contraindications to transplantation. Although a complex congenital anatomy can be intimidating, the insertion of a device can be straightforward. Even patients with cyanotic disease may benefit from the physiologic effects of a VAD, as shown with the patient we have described. As the number of adult patients surviving with congenital heart defects increases, VAD therapy should be increasingly considered as an alternative in the treatment algorithm of ventricular failure in these patients.

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