Insufficiency of the native aortic valve and left ventricular assist system inflow valve after support with an implantable left ventricular assist system: Signs, symptoms, and concerns

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A 57-year-old man with a history of idiopathic dilated cardiomyopathy was admitted to Hahnemann University Hospital, Philadelphia, with progressive symptoms of heart failure and was listed for heart transplantation. His condition deteriorated despite institution of inotropic drug support. An echocardiogram showed global biventricular dysfunction with no aortic stenosis or insufficiency. On June 20, 2000, a TCI VE HeartMate left ventricular assist system (LVAS) (Thoratec Corporation, Pleasanton, Calif) was implanted. The patient had an uneventful postoperative course and was discharged to his home on July 23, 2000. He was doing well with LVAS rates between 70 and 80 beats/min and flows ranging between 6 and 8 L/min. Two and one-half months after implantation, he began to have mild dyspnea with his usual activities. The LVAS rates and flows were inappropriately elevated at rest (80-100 beats/min, flows 8-9 L/min). An echocardiogram showed moderate LVAS inflow valve regurgitation and mild native aortic valve insufficiency. On October 3, 2000, an orthotopic heart transplantation was performed. The LVAS inflow valve had two small linear perforations in the leaflets and partial dehiscence of two commissures. The LVAS outflow valve was intact. The aortic valve was fused along the anterior aspect of the left noncoronary commissure (Figure 1). The posterior aspect of the commissure was open. A small thrombus was present along the right lunula of the left coronary cusp of the aortic valve. Examination of the ostium of the outflow graft insertion revealed it to be situated above the right coronary cusp with the angle of the graft directed toward the left coronary cusp. The ostia of the coronary arteries were patent. No

mineralization was noted. Microscopic examination of the valve across the area of fusion revealed the fusion tissue to be composed of myxomatous granulation tissue adherent to the inflow aspect of the left and noncoronary cusps (Figure 1). No evidence of remote endocarditis or of organizing thrombus at the point of fusion was detected. The remainder of the valve cusps showed no evidence of chronic rheumatic valve disease.

The patient recovered well and was discharged to his home on October 13, 2000, with no shortness of breath.

Discussion

As the duration of support with implantable LVAS units is extended, the issues regarding device durability and cardiac remodeling have begun to surface. Issues of drive-line infection, controller malfunction, battery life span, and inflow valve integrity have raised concern regarding current LVAS units as destination devices. A new problem that has been recently recognized is acquired native aortic valve disease induced by long-term LVAS support.¹

To our knowledge, the only report of acquired native aortic valve disease associated with an implantable LVAS was by Rose and colleagues¹ from the University of Minnesota. In this report, 4 of 6 patients with the TCI Heartmate LVAS showed evidence of commissural fusion at the time of explantation for transplantation. The mechanism was believed to be thrombus formation, organization, and subsequent acquired aortic stenosis. The mean duration of LVAS support was 180 days, but there was no correlation between the duration of LVAS implantation and the generation of aortic valve thrombosis and commissural fusion. Furthermore, there were no clinical signs suggestive of this process.

In our case, the aortic valve disease was suggestive of "systemic" pressure-related changes on the valve leaflets, particularly those of the native aortic valve and the LVAS inflow valve (25 mm porcine valve, Medtronic, Inc, Minneapolis, Minn). The findings on the native valve were not present before the LVAS by echocardiography, so that the emergence of this abnormality can be considered an acquired phenomenon from the LVAS. The mechanism of the inappropriately elevated rates and flows is related to the recycling of blood into the left ventricular cavity through the insufficient valves combined with a lower resistance to LVAS flow.

As a result of this finding, we began to routinely use echocardiography to monitor patients with long-term (>6 months) LVAS support, whether or not they had symptoms or demonstrated inappropriately elevated LVAS rates and flows. Interestingly, 2 of 4 patients having long-term outpatient LVAS support had

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Figure 1. Native aortic valve demonstrating fusion of the anterior commissure between the left and noncoronary cusps. A small thrombus is present on the right lunula of the left cusp.

inappropriately elevated rates and flows, 1 of whom admitted to increased shortness of breath. The other 2 patients were symptom-free with appropriate LVAS rates and flows. Echocardiography in the patients with inappropriate rates and flows showed mild to moderate degrees of LVAS inflow valve and native aortic valve regurgitation. No native aortic or LVAS inflow valve insufficiency was detected in the patients having appropriate rates and flows.

From the clinicopathologic finding in the case report and the observations of the remaining patients, we recommend a surveillance strategy for patients requiring long-term support with implantable LVAS units. Routine echocardiography should be performed at 6-month intervals in the absence of symptoms to document native aortic and LVAS inflow valve integrity. In the presence of new symptoms (dyspnea) or inappropriately elevated LVAS rates and flows, echocardiography should be performed to search for valve abnormalities.

Figure 2. Microscopic view of area of fusion between the left and noncoronary cusps. Granulation tissue spans the space between the two valve cusps causing adherence at the inflow surface.

Addendum

Since the preparation of this report, 3 additional patients have demonstrated inappropriately elevated LVAS rates and flows with inflow valve and native aortic valve insufficiency.

Reference

1. Rose AG, Park SJ, Bank AJ, Miller LW. Partial aortic valve fusion induced by left ventricular assist device. *Ann Thorac Surg.* 2000;70:1270-4.